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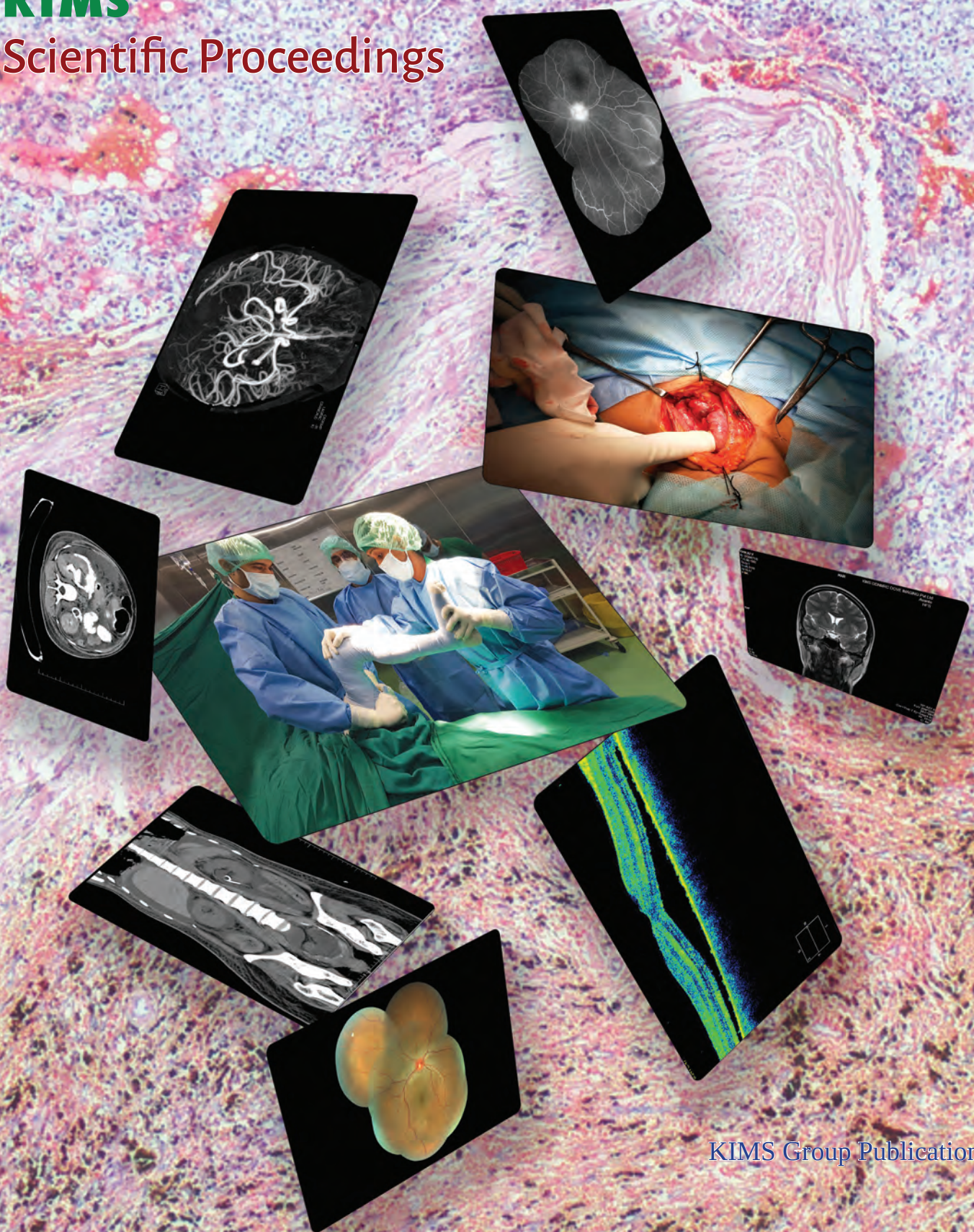
Scientific Proceedings



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- ACHSI (Australian Council on Healthcare Standards International)
  KIMS got ACHSI accreditation in the year 2006 for demonstrating continuous improvements in patient safety and delivery of quality healthcare that is at par with international standards.
- NABH (National Accreditation Board for Hospitals & Healthcare Providers - India)
  KIMS received NABH in the year 2006 as a recognition of its commitment to ensure safe healthcare practices and infection control measures.
- NABL (National Accreditation Board for Testing & Calibration Laboratories)
  The Laboratory at KIMS is accredited by NABL in the year 2008, for ensuring precise diagnosis and following safe practices.
- NABH (National Accreditation Board for Hospitals & Healthcare Providers - India)
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Issue Eleven

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Editorial

The impact of the corona virus is hitting nations worldwide, but with widely different levels of severity. As of now, countries are moving towards a relaxation of the most severe levels of lockdown. The focus now shifts to the shape of the economic recovery. Healthcare professionals across the globe face one of the greatest challenges of the era, since there are no proven therapies as yet to treat COVID-19. Efforts are on in various countries including India, to develop an effective vaccine to conquer this pandemic.

It is a matter of relief and pride that Kerala implemented one of the best containment strategies and it is now referred to as the 'Kerala Model' as far as testing and containment strategies are concerned. Kerala achieved a flattening of the deadly curve by measures that included early detection, aggressive testing and contact tracing, and a 28-day quarantine period; double that prescribed by the WHO. What is making it possible is a robust public health system.

KIMS has offered its hand in support to the government contributing to the relief fund. We are active members of a medical team with expertise to combat any emergency situation that might arise.

This edition of KIMS Scientific Proceedings, which is the 11th release, has been prepared as a concise edition amidst this Pandemic. We have included contributions from as many specialities as possible like Chromoblastomycosis, a review report on the management of carcinoma prostate, a case of Pancreatic cancer, an unusual case of mild hemoptysis which are unique and are discussed in detail followed by a structured report on our Academic activities like paper presentations by KIMS doctors and their accolades.

We thank the contributors and their coordinators. We also request your continued support. Having completed 10 editions and going further we would welcome your feedback and suggestions.

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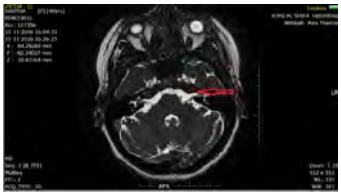

Pancreatitis in periampullary Ca and its consequences—A case report and literature review

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Abstract

Pancreatic cancer has been known to be an uncommon etiology of Acute pancreatitis which can result in diagnostic dilemma and delay in appropriate surgical intervention. However when present, pancreatitis is usually an early symptom of pancreatic cancer and carries a good prognosis provided accurate diagnosis and timely intervention is carried out.Here we describe case report of a patient with pancreatic cancer who presented elsewhere with pancreatitis and was misdiagnosed resulting in multiple abandoned surgical procedures and delay in appropriate surgery

Keywords: NMDA, Acute pancreatitis(AP), Pancreatic cancer(PC), Periampullarycancer, Left sided portal hypertension, Sinistral portal hypertension(SPH), Neoadjuvantchemotherapy, Walled off necrosis(WON), Acute necrotic collection (ANC)

Introduction

Pancreatic cancer is one of the most lethal human cancers and continues to be a major unsolved health problem. Symptoms of pancreatic cancer are generally precipitated by compression or invasion of surrounding structures: the bile duct, the pancreatic duct, the duodenum and mesenteric and celiac nerves. Major symptoms of pancreatic cancer are obstructive jaundice, and abdominal or backpain. Acute pancreatitis is diagnosed in the appropriate clinical setting with the aid of laboratory values and imaging studies. Laboratory analysis usually reveals elevated pancreatic enzymes. Abdominal computed tomography(CT) supports the diagnosis and excludes othercauses. Alcohol and gallstones are the two most commoncauses of acute pancreatitis. Although pancreatic cancer produces obstructive pancreatitis upstream from a constricted portion of the pancreatic duct, acute pancreatitis is a less common manifestation of pancreatic cancer. Knowing that acute pancreatitis as an initial presentation of pancreatic cancer may provide information that can eventually help to identify this subgroup of patients with pancreatic cancer early and timely and appropriate treatment can be offered.

Case presentation

48 yr old gentleman from Kanyakumari who is not an alcoholic or smoker initially noticed weight loss and melena in April 2019.He was evaluated at local hospital and found to be jaundiced. CT showed soft tissue lesion involving terminal CBD extending into periampullary area with venous collateral in upper abdomen. Robotic resection of periampullary Tumorwas attempted elsewhere on May 2019 but abandoned due to intraop finding of left sided Portal hypertension and acute pancreatitis.Intraop biopsy from head of pancreas showed features suggestive of pancreatitis only. He underwent CBD stenting and repeat imaging after 2 weeks which showed metabolically active tissue at ampulla which seemed resectable and some collections near body and tail of pancreas. Hence laparotomy was attempted by end of May 2019 but had to be abandoned again due to extensive collaterals and adhesions. Following, this patient sought treatment at KIMS.

* Professor Emeritus

At KIMS repeat imaging revealed ongoing pancreatitis with ill defined cystic collections in head on tail suggestive of ANC(fig.1 & fig.3) and features of left sided portal hypertension were also noted (fig.5). Side viewing endoscopy on July 2019 which revealed ulceroproliferative lesion at ampulla. Biopsy from this lesion detected moderately differentiated adenoCA. In view of the unique clinical presentation and multiple failed surgical attempts his case was discussed in a multi disciplinary tumor board and it was decided that the best course of treatment would be to allow some time for resolution of the inflammation related to acute pancreatits and to start him on chemotherapy to buy time and to prevent tumor progression in the waiting period.

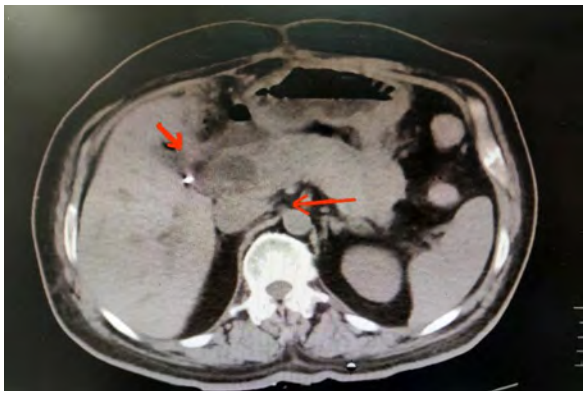

Fig.1:Initial CT showing ill defined cystic areas in head of pancreas-

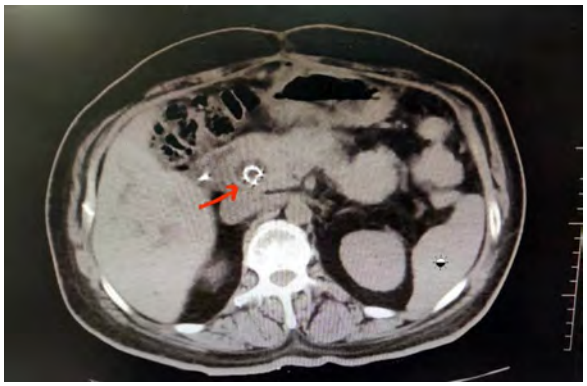

Fig.2:CBD stent visualised

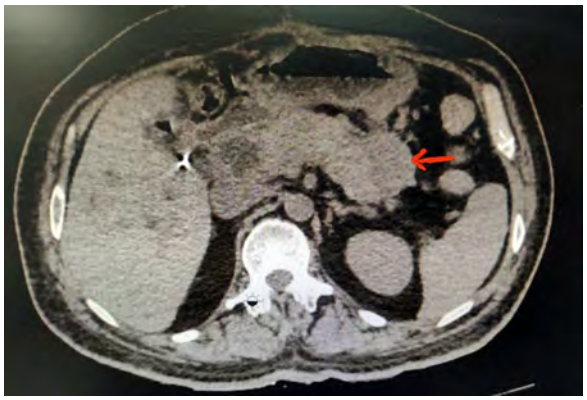

Fig.3:Ill defined cystic areas - ANC visualized at tail of pancreas

Hence he was started on Neoadjuvant chemotherapy with FOLFOX. Repeat PETCT in Sep 2019 after 3 cycles of FOLFOX showed the periampullary lesion and the cystic areas in previous CT were now well defined(Fig.4). After rediscussion in tumor board it was decided that now would be an apt time for surgical intervention and we proceeded with preoperative evaluation and surgery. Difficulty in dissection was anticipated and appropriate planning was done by the surgical team.

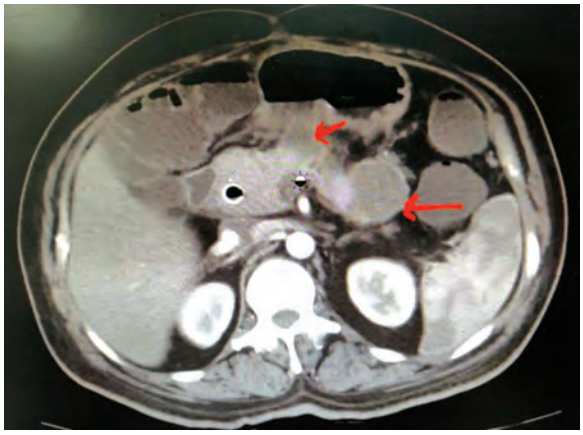

Fig.4: Well defined cystic collections have evolved into WON

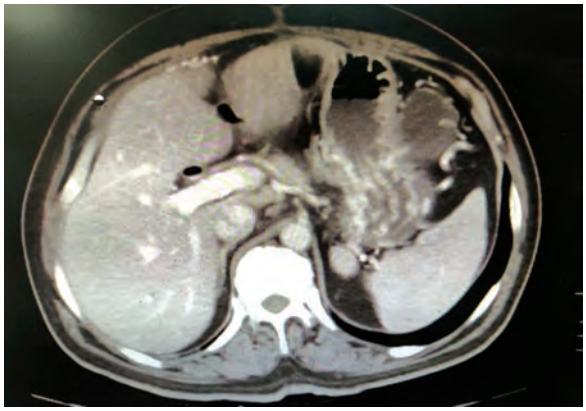

Fig.5: Venous collaterals due to Sinistral Portal hypertension

He underwent Whipples procedure at KIMS on October 2019.There were dense omental adhesions to midline wound and port sites and loss of planes between structures. Head of pancreas was bulky with Walled off necrosis in

body of pancreas and periportal inflammatory nodes.The entire surgery lasted about 16 hours but was successful and and R0 resection was ensured. Postop recovery was smooth—he had Grade A postoperative pancreatic fistula which settled with conservative management but otherwise had no other complications. Histopathology of resected specimen showed a 0.7cm tumor in the ampulla with perineural and lymphovascularinvasion. Resection margins were free of tumorand lymph nodes harvested showed only reactive changes. Adjacent tissues showed features suggestive of chronic pancreatitis. He is now doing well and receiving Adjuvant chemo--FOLFOX.

Discussion

Pancreatic Cancer has been described to be the cause of Acute pancreatits in 6-13%[1].This can cause diagnostic dilemma and delay in surgical intervention.

Presentation

AP is associated with pain in almost 100% cases and jaundice /weight loss alone is rare[2]. AP secondary to PC is usually mild and relapsing3.Average of 1 month (but upto 1 year) delay in diagnosis has been described[3]—mostly because PC is a rare ethology and usually not considered. Pancreatic inflammation may also mask the mass offence of PC. Elevated CA 19-9 and early EUS FNA in suspected cases may aid in diagnosis.

Sinistral/left sided PHT

SPH is a syndrome in which thrombosis of splenic vein manifests with gastric varies in a patient with patent PV and normal hepatic function.Upper gastrointestinal bleed is the commonest presentation. PC and Chronic pancreatitis are among the commonest causes but tumor location is usually body/tail when PC is the cause. PC must be suspected and ruled out in any patient with SPH. First line of management is endoscopic management of bleeding gastric varices. Definitive management is removal of the primary cause which may be combined with splenectomy.Gastricvariceal bleed from Splenic vein thrombosis occurs only in 4 % of patients,hence routine splenectomy is not recommended[4]

Why PC causes AP?

Even though exact cause is unclear, many theories have been considered[5]. Obstruction of pancreatic duct,blood vessel obstruction by mass, inflammatory mediators / enzyme activation by the PC have all been thought to be contributing factors. AP can occur without dilation of Main pancreatic duct because of slow growth of the CA which may not narrow the duct. AP has been shown to accelerate development of PC in animal models but this has not been proven conclusively in humans.

When to operate?

No clear guidelines exist as the problem is rarely encountered.Here we review data from existing limited case series related to this issue. AP itself has high morbidity and mortality—hence it is best avoid interventions during acute phase. Studies have shown that patient may be benefited by waiting at least 4 weeks if necrosis develops i.e allow walled off necrosis to form. Necrosectomy during the time of Whipples may be needed if only WON is infected. Only existing study which analysed PC in patients with mild AP suggested a cut off of 24.5 days before surgery[3].

Effect on survival

As long as diagnosis of PC is not missed due to overlying AP and appropriate intervention is provided ,survival outcomes have been shown to be similar. Early intervention (within 1 month of AP diagnosis) results in increased local complications and delayed post op recovery. Mean survival after resection is 20-24 months in PC with and without AP—suggesting that etiology as such does not affect the prognosis as long as appropriate and timely surgical intervention is carried out[1].

Conclusion

Even though PC is an uncommon cause for AP, there is a significant proportion in whom this is the case and missing this diagnosis can have disastrous consequences as we have shown here. Anatomic evaluation of pancreas

must be done even in mild cases of AP—especially when etiology is unclear. EUS-FNA and CA 19-9 may aid in timely diagnosis of PC in suspicious cases. Accurate clinical and imaging assessment must be utilised to decide on timing of surgical intervention in these patients. With timely diagnosis and surgery survival is comparable to other patients with PC.

References

1. Minato Y, Kamisawa T, Tabata T, Hara S, Kuruma S, Chiba K, et al. Pancreatic cancer causing acute pancreatitis: a comparative study with cancer patients without pancreatitis and pancreatitis patients without cancer. Journal of Hepato-Biliary-Pancreatic Sciences. 2013 Aug;20(6):628–33.
2. Mujica VR, Barkin JS, Go VLW. Acute Pancreatitis Secondary to Pancreatic Carcinoma: Pancreas. 2000 Nov;21(4):329–32.
3. Li S, Tian B. Acute pancreatitis in patients with pancreatic cancer: timing of surgery and survival duration. Medicine. 2017 Jan;96(3):e5908.
4. Saif MW, Kaley K, Lamb L. Pancreatic Adenocarcinoma Complicated by Sinistral Portal Hypertension. Cureus [Internet]. 2016 Jul 14 [cited 2020 Mar 7]; Available from: http://www.cureus.com/articles/4692-pancreatic-adenocarcinoma-complicated-by-sinistral-portal-hypertension.
5. Minato Y, Kamisawa T, Tabata T, Hara S, Kuruma S, Chiba K, et al. Pancreatic cancer causing acute pancreatitis: a comparative study with cancer patients without pancreatitis and pancreatitis patients without cancer. Journal of Hepato-Biliary-Pancreatic Sciences. 2013 Aug;20(6):628–33.
6. Gislason H, Horn A, Hoem D, Andrén-Sandberg Å, Imsland AK, Søreide O, et al. Acute Pancreatitis in Bergen, Norway: A Study on Incidence, Etiology and Severity. Scand J Surg. 2004 Mar;93(1):29–33.

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Small bowel bleed- Case series

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Abstract

Small bowel bleeding is an uncommon etiology of GI bleeding. Evaluation of Small bowel bleed needs exhausting evaluation that includes Upper GI Endoscopy, colonoscopy and CT abdomen. Some patients may need evaluation with Capsule endoscopy or Enteroscopy. Here we are presenting two cases of Small bowel bleed evaluated and managed by Capsule endoscopy and novel Power Spiral Enteroscopy.

Keywords: OAC – Oral anti-coagulation, Small bowel bleed, Capsule endoscopy, Power spiral Enteroscopy, Angiodysplasia

Introduction

Gastro-intestinal (GI) bleeding is one of the most common Emergencies seen in a hospital. The small intestine is the least common site of GI bleeding but it is the most commonest cause of obscure GI bleed. It is estimated that upper gastro-intestinal bleeding (UGIB) (from the esophagus to duodenum), lower gastro-intestinal bleeding (LGIB) (from the colon and anorectum) and obscure bleeding account, respectively, for 50%, 40% and 10% of total GI bleeding[1].

The small bowel is called 'the dark continent of the GI tract' because of its inaccessibility to endoscopists, due to its intra-peritoneal location, excess mobility and long length. Approximately 5% of GI bleeding occurs from the small bowel, defined as the region between the ligament of Treitz and the ileocecal valve[2].

Because of an inability to visualize the small bowel properly, patients with a small bowel GI bleed usually end up undergoing multiple diagnostic investigations, requiring multiple hospitalizations and transfusions; therefore, it is necessary to identify the cause and site of hemorrhage accurately, so as to institute appropriate, effective therapy. Here we are presenting two cases of Obscure GI bleeding evaluated in our institute.

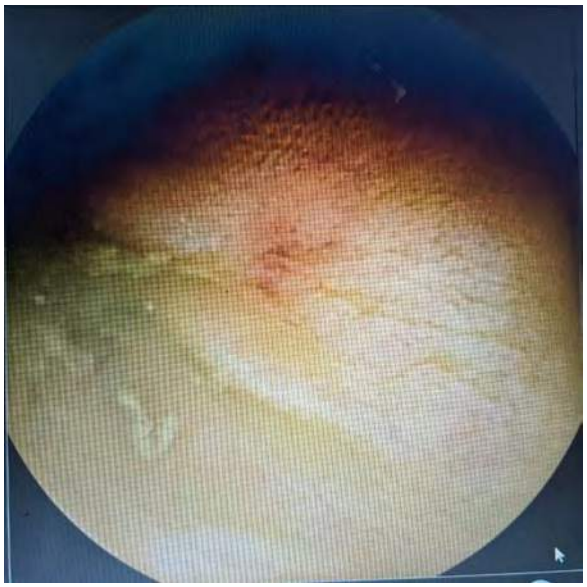
Case 1

46 yr old male from Kollam came with c/o transient loss of consciousness and associated with h/o black color stools for past 2 days. No h/o vomiting/hemetemesis, abdomen pain or distension. There is no h/o Diabetes or Hypertension or Coronary artery disease. He had h/o occasional ethanol intake. No h/o any surgery in past. Pt on examination had Pallor and his vitals showed tachycardia and his blood pressure was within normal limits. His systemic examination was normal and per rectal examination showed black color stools. On receiving in Emergency department – Ryles Tube was inserted which is clear no evidence of any active bleeding and his hemogram showed Low hemoglobin of(9.6 g/dl) His Liver function test, Renal function and INR is normal. Ultrasound abdomen done which is suggestive of fatty liver and no evidence of CLD.

After stabilisation Upper GI endoscopy done which showed Small hiatus hernia and Antral gastritis with erosions. There was no evidence of any bleeding and Colonoscopy showed black stools seen throughout colon and ileum. In view of suspected Small bowel bleed CT angiogram was done which showed no evidence of any contrast extravasations. No evidence of intestinal

obstruction. Capsule Endoscopy done which showed Angiodysplasia in Jejunum

Referred to higher centre for enteroscopy.



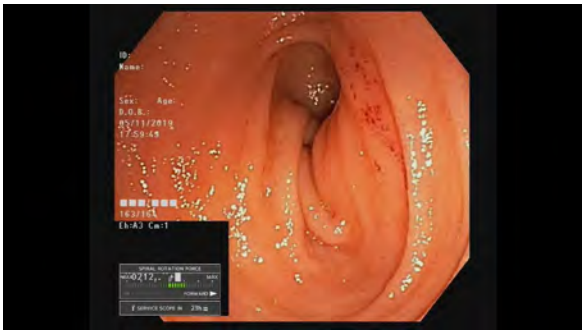
Angiodysplasia in Jejunum – Capsule Endoscopy

Case 2

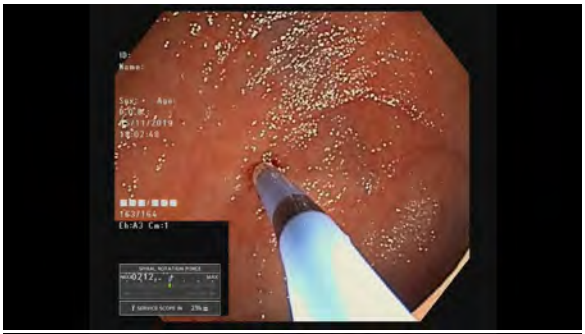
72 yr old female from Trivandrum came with c/o loose stools for 1 week with black colour stools for 2 days. There was no evidence of vomiting, hemetemesis, and abdomen pain or abdomen distension. No h/o fever or loss of weight. Her past h/o not suggestive of Diabetes, Hypertension, CAD. She had h/o severe necrotising fascitis for which fasciotomy was done. On examination she had severe Pallor and no icterus or pedal edema. Vitals showed tachycardia of 102/min and her Blood pressure was-110/70 mm hg. Systemic examination is normal. Basic investigations showed low hemoglobin of 3.6 g/dl and her renal functions showed high creatinine of 2.2 g/dl.

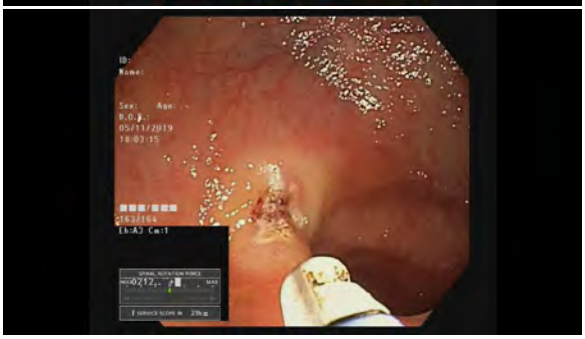
USG abdomen showed no evidence of CLD. OGD done which showed features of Hiatus hernia/Grade B esophagitis no varices and Colonoscopy showed altered blood seen throughout colon and ileum. CT abdomen angiogram was considered to rule out small bowel bleed but In view of altered renal function patient relatives are not willing to proceed.Hence after ruling out other causes planned for Spiral enteroscopy.

Spiral enteroscopy done showed Angiodysplasia in jejunum and ileum. Through power spiral enteroscope Argon Plasma photocoagulation of the lesion done and pt kept for observation. Her haemoglobin was stable.

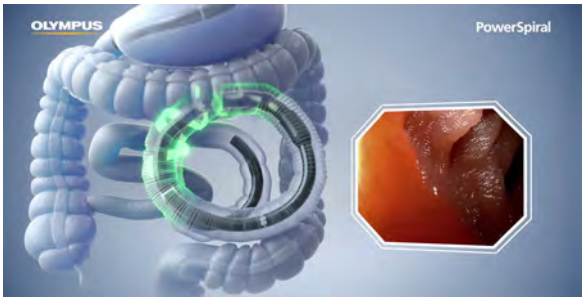


Angiodysplasia in Ileum – PowerSpiral Enteroscopy





Angiodysplasia in Ileum – PowerSpiral Enteroscopy



Olympus- PowerSpiral - First time in South Kerala

Discussion

Small bowel bleeding being commonest reason for obscure GI bleeding possess different etiology based on age group (Table 1). The commonest cause for small bowel GI bleeding are vascular, with other causes being tumors, inflammatory lesions, and medications, as well as some rare causes like haemobilia, haemosuccus pancreaticus and aorto-enteric fistula.

Vascular lesions and small bowel lesions induced by non-steroidal anti inflammatory drugs (NSAID) are the common causes of small bowel GI bleeding in the elderly, whereas tumors, Meckel's diverticulum, Dieulafoy's lesion and Crohn's disease are the common causes in patients under 40 years of age[3, 4].

Zhang et al. studied 385 OGIB patients and found that, in elderly patients (>65 years), vascular anomalies

Table 1. Etiology of obscure gastro-intestinal bleeding according to age[5,6]

Elderly (>65 years)	Middle-Aged (41-65 years)	Young Adult (17-40 years)
• Vascular anomalies • Small intestinal ulcer • NSAID enteropathy • Small intestinal tumours • Non-specific enteritis • Celiac disease	• Vascular anomalies • Small intestinal tumours • Non-specific enteritis • Small intestinal ulcer	• Crohn's disease • Small intestinal tumours • Meckel's diverticulum • Non-specific enteritis • Dieulafoy's lesion • Vascular anomalies • Celiac disease

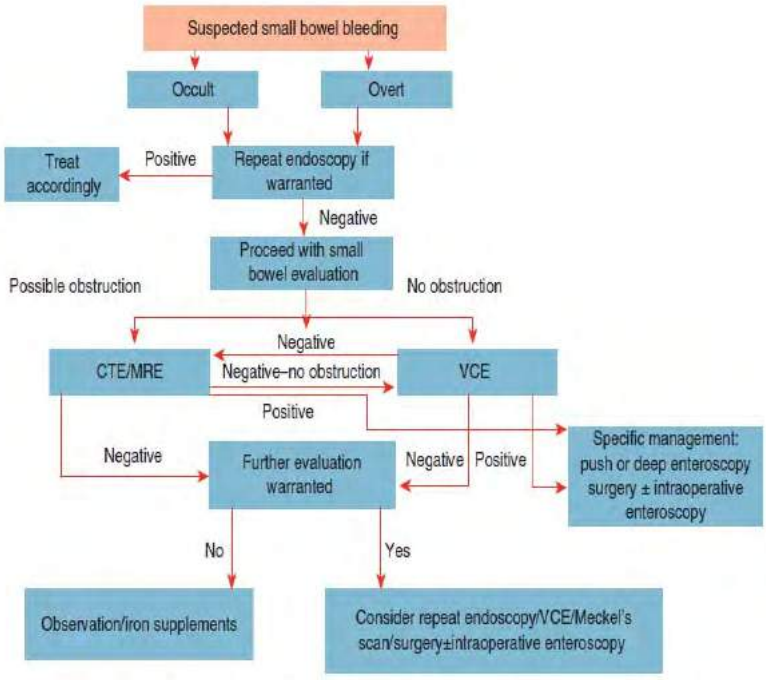


Fig. 1: Algorithm for suspected small bowel bleeding. CTE, computed tomographic enterography; MRE, magnetic resonance enterography; VCE, video capsule endoscopy

(54.35%), small intestinal ulcer (13.04%), small intestinal tumours (11.96%) were the common cause of small intestinal bleeding; in middle age (41–64 years) vascular anomalies (34.82%), small intestinal tumours (31.25%), non-specific enteritis (9.82%) were the major causes and in young adults.

Angiodysplasia (angioectasia or vascular ectasia) is abnormally dilated, tortuous, thin-walled vessels, involving small capillaries, veins and arteries[5,6]. They are visualized within the mucosal and submucosal layers of the gut, are lined by endothelium with little or no smooth muscle, and lack inflammatory or fibrotic changes as well as fibrosis[6, 7]. They are the most common cause of small bowel bleeds.

In a systematic review by Liao et al. that included 227 studies and 22 840 small bowel capsule endoscopies, OGIB, at 66%, was the most common indication and angiodysplasia was the most common underlying lesion (50%) [8]. Meyer et al. reviewed 218 cases of arterio-venous malformations (AVM) and found that the cecum or right colon was the most common location (78%), whereas the jejunum (10.5%), ileum (8.5%) and duodenum (2.3%) are other sites for AVM[9].

Bleeding from angiodysplasia in patients with aortic stenosis (AS)—termed Heyde's syndrome—is a well-known clinical syndrome[10]. It has been shown that high stress in aortic stenosis causes shear-dependent cleavage of high molecular weight multimers of von Willebrand's factor (vWF), leading to acquired vWF deficiency[11]: vWF is essential for the adhesion and aggregation of platelets to the sub-endothelium of damaged blood vessels. Aortic valve replacement had ameliorated the acquired vWF abnormality, suggesting an association between them[11].

Deep enteroscopy is being used for the complete examination of the small bowel by using double balloon enteroscopy (DBE), single balloon enteroscopy (SBE) and spiral enteroscopy (SE).

All these endoscopes have both diagnostic and therapeutic potential and require an over tube for advancement of the scope. The complication rate has been described more commonly in therapeutic DBE (4.3%) as compared with diagnostic DBE (0.8%)[12].

SBE has a single balloon at tip of over tube and has similar diagnostic and therapeutic yield[13]. The complete visualization is possible in 11% of the patients, in contrast to 18% in DBE[14]. When compared with DBE, SE reduces the examination time[15.16]. When SE was compared with SBE, SE was found to be having greater depth of insertion but similar diagnostic yield and procedural time[17].

Olympus Power spiral enteroscope is a novel motorized spiral enteroscope with safety features and easy learning curve. This is the first patient done in our hospital.

Intra-operative enteroscopy is a last resort and gold standard for evaluation of occult GI bleeding. Due to the availability of Capsule Endoscopy and deep enteroscopy, it is infrequently used in current practice. IOE can be accessed either by open laparotomy or by laparoscopic assisted technique[18].

Conclusion

Evaluation of Small bowel bleed is clinically challenging. With advent of Capsule endoscopy and Power Spiral enteroscopy the armamentarium for evaluation and therapeutic option for Small bowel bleed is expanding and more patients are getting benefits.

References

1. Savides TJ and Jensen DM. Gastrointestinal Bleeding. In: Feldman M, Frience S and Brandt LJ (eds), Sleisenger and Fordtran's Gastrointestinal and Liver Disease. 9th Edition. Elsevier Medicine, 2010.
2. Katz LB. The role of surgery in occult gastrointestinal bleeding. Semin Gastrointest Dis 1999;10:78–81.
3. Raju GS, Gerson L, Das A et al. American Gastroenterological Association (AGA) institute technical review on obscure gastrointestinal bleeding. Gastroenterology 2007;133:1697–717.

4. Zhang BL, Chen CX and Li YM. Capsule endoscopy examination identifies different leading causes of obscure gastrointestinal bleeding in patients of different ages. Turk J Gastroenterol 2012; 23:220–5.

5. Lewis BS. Small intestinal bleeding. Gastroenterol Clin North Am 2000;29:67–95.

6. Poralla T. Angiodysplasia in the renal patient: how to diagnose and how to treat? Nephrol Dial Transplant 1998;13:2188–91.

7. Regula J, Wronska E and Pachlewski J. Vascular lesions of the gastrointestinal tract. Best Pract Res Clin Gastroenterol 2008;22: 313–28.

8. Liao Z, Gao R, Xu C et al. Indications and detection, completion, and retention rates of small bowel capsule endoscopy: a systematic review. Gastrointest Endosc 2010;71:280–86.

9. Meyer CT, Troncale FJ, Galloway S et al. Arteriovenous malformations of the bowel: an analysis of 22 cases and a review of the literature. Medicine (Baltimore) 1981;60:36–48.

10. Shoenfeld Y, Eldar M, Bedazovsky B et al. Aortic stenosis associated with gastrointestinal bleeding. A survey of 612 patients. Am Heart J 1980;100:179–82.

11. Vincentelli A, Susen S, Tourneau TL et al. Acquired von Willebrand Syndrome in Aortic Stenosis. N Engl J Med 2003;349:343–349.

12. Mensink PB, Haringsma J, Kucharzik T et al. Complications of double balloon enteroscopy: a multicenter survey. Endoscopy 2007;39:613–5.

13. Efthymiou M, Desmond PV, Brown G et al. SINGLE-01: a randomized, controlled trial comparing the efficacy and depth of insertion of single- and double-balloon enteroscopy by using a novel method to determine insertion depth. Gastrointest Endosc 2012; 76:972–80.

14. Domagk D, Mensink P , Aktas H et al. Single- vs. double-balloon enteroscopy in small bowel diagnostics: a randomized multicenter trial. Endoscopy 2011;43:472–6.

15. May A, Manner H, Aschmoneit I et al. Prospective, cross-over, single-center trial comparing oral double-balloon enteroscopy and oral spiral enteroscopy in patients with suspected small bowel vascular malformations. Endoscopy 2011;43:477–83.

16. Rahmi G, Samaha E, Vahedi K et al. Multicenter comparison of double-balloon enteroscopy and spiral enteroscopy. J Gastroenterol Hepatol 2013;28: 992–8.

17. Khashab MA, Lennon AM, Dunbar KB et al. A comparative evaluation of single-balloon enteroscopy and spiral enteroscopy for patients with mid-gut disorders. Gastrointest Endosc 2010;72:766–72.

18. Poulose BK. Intra-operative endoscopy to identify lesions. Tech Gastrointest Endosc 2013;15:180–3.

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Malignant catatonia with orofaciolingual dyskinesia in a young female - an interesting case of Anti-NMDA receptor encephalitis

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Abstract

Any young female presenting with malignant catatonia and orofaciolingual dystonias associated with neurobehavioural symptoms should be considered in the differential diagnosis for NMDAR encephalitis as it is a potentially treatable condition.

Keywords: NMDA, Encephalitis, Orofacial dyskinesia, catatonia, Ovarian teratoma

Introduction

Anti NMDA receptor (NMDAR) encephalitis is a potentially fatal autoimmune disease characterised by a complex neuropsychiatric syndrome associated with presence of serum and CSF antibodies against the GluN1 subunit of NMDAR. The disease is frequently associated with ovarian teratomas and young women are disproportionately affected. Neurobehavioural symptoms, seizures, movement disorders, insomnia, psychosis and central hypoventilation are some of the clinical features. Approximately 80% of the patients improve after timely immunotherapy and tumour resection.

Case presentation

A 19 year old female with no prior comorbids presented with acute onset behavioural changes and memory disturbances associated with low grade fever. Examination revealed normal vitals. Neurological examination revealed hypomobile mute state with waxy flexibility and catalepsy suggestive of catatonia. She was having intermittent abnormal movements suggestive of orofaciolingual dystonias. MRI Brain plain and contrast was normal. CPK was elevated. EEG revealed extreme delta brushes. Serum and CSF were positive for Anti NMDA receptor antibody. CT Abdomen plain and contrast showed no evidence of ovarian teratoma. All these features were suggesitive of anti NMDAR encephalitis presenting as catatonia with orofaciolingual dystonia. She was initiated on pulse IV methylprednisolone followed by IvIg and rituximab. She improved drastically and was discharged home.

Discussion

An increasing number of case reports of anti NMDAR encephalitis since its description in 2007 suggest that it is not a rare disorder. It is a syndrome characterized by neuropsychiatric symptoms associated with seizures and movement disorder. Overlap with psychiatic symptoms often causes misdiagnosis or late diagnosis. Neurobehavioural symptoms associated with movement disorder in a young female should always raise the suspicion of NMDAR encephalitis as in our case .

References

1. Movement disorders with neuronal antibodies: syndromic approach, genetic parallels and pathophysiology.

Bettina Balint, Angela Vincent, Hans-Michael Meinck, Sarosh R Irani, and Kailash P Bhatia.

2. An update on anti-NMDA receptor encephalitis for neurologists and psychiatrists: mechanisms and models.

Prof Josep Dalmau, Thais Armangué, Jesús Planagumà, Marija Radosevic, Francesco Mannara, Frank Leypoldt, Prof Christian Geis, Eric Lancaster.

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Minimal invasive approach for iatrogenic bilateral ureteric injury

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Abstract

The index case is a 46/F diagnosed to have Didelphys uterus with B/L HUN. She was operated at outside hospital - pre-op B/L DJ stenting followed by hysterectomy. DJS removal was done on the next day following surgery. Right stent was removed with difficulty and on the left side, stent could not be removed. Over 4-5 days, she developed abdominal distension with decreased urine output and elevation of serum creatinine. She was misdiagnosed to have renal failure and managed accordingly. She was referred to our center for further management. CECT abdomen showed bilateral mild HDUN with b/l renal pelvic leak with left DJ stent in place followed by dialysis. She underwent right side antegrade stenting with B/L PCN. Subsequently she underwent left URS during which it was found that a vicryl suture was encircling and kinking the stent at midureteric level at the level of pelvic brim. The visualized suture was cut with laser at 15 W, following which the stent could be removed without difficulty. Subsequently left side re DJ stenting done. Thereafter she had an uneventful recovery. Both nephrostomies were removed after 2 days and both stents were removed after 3 months. This case is being presented to highlight the utility of laser in dealing with sutures that have gone through the ureters and to emphasize correlation of clinical details to arrive at a correct diagnosis. With minimally invasive techniques, we could avoid open exploration or a probable surgery for ureteric stricture later.

Keywords: Latrogenic, Ureteric, Injury, Ureteroscopy, Ho:Yag laser, Endourology

Introduction

The accidental iatrogenic ureteral injuries are still one of the most serious postoperative complications in intra-abdominal, retroperitoneal, or pelvic operations with significant morbidity due to the possible impairment of renal function and susceptibility to microbial pathogens despite the ongoing advances in modern surgical techniques[1,2]. The surgical interventions responsible for iatrogenic ureteral injuries include the gynaecological surgeries such as total abdominal hysterectomy, bilateral salpingo-oophorectomy followed by radical hysterectomy[3] and also consist of the colorectal and urological operations; in such procedures the ureters run close to the field of dissection[4,5]. The ureteral injuries most commonly reported with gynecological procedures. The injuries are associated with accidental ligation, laceration, crushing, stretching, and devascularization[6]. The incidence documented is 0.3% to 1.5%[7]. Generally, iatrogenic lower ureteral injuries are caused by the aberrant sutures responsible for the strictures or obstructions. These complications generally have been repaired utilizing end-to-end ureteral anastomosis or a Boari flap with ureteral reimplantation or some combinations, as well as a psoas hitch[8,9]. However, the above mentioned traditional open surgical techniques are somehow no longer widely accepted as options for the management of iatrogenic lower ureteral injury, as some reports have associated ureteroureterostomy with a relatively high rate of severe complications, although the issue is still controversial[10,11]. Besides, another reason for ureteroureterostomy rarely

being introduced to repair lower ureteral injury was that distal ureters had a relatively poor vascular and blood supply while the pelvic ureter appears to have a high preponderance of plexiform vessels, which has been considered susceptible to ischemia after transection[12]. Thus, it is highly desirable to seek the alternative treatment approach.

Fortunately, laparoscopic reconstructions of ureteral injuries have been introduced in the clinical practice, and the experience is continuously extending[12], among which, endoscopic minimally invasive approaches increasingly emerge to be applicable options for the management of ureteral lesions. Several cases of iatrogenic ureteral injuries successfully managed by percutaneous nephrostomy were reported[13], whereas various modalities applied to reconstructions of ureteral injuries still have many disadvantages to overcome, such as hemorrhage and the risk of damage to surrounding tissues.

With the development of minimally invasive techniques, including endoscopic ureteral incision, several reports have focused on the use of the holmium laser[14-18]. The holmium: yttrium–aluminum-garnet (Ho:YAG) laser emits light at a frequency of 2100 nm. As the laser energy is well absorbed by water, effecting a penetration depth of 0.5 mm, the vaporization effects are quite concentrated. The predominant vaporization and minimal coagulation effects associated with this instrument tend to result in minimal rescarring, particularly when compared with other energy sources, which are associated principally with coagulation effects. At high energy levels, the Ho:YAG laser is capable of incising strictures and suture materials with good hemostasis and minimal thermal damage to surrounding tissues[19].

Here, we report an ideal, novel, available, and also minimally invasive technique with combined utilization of the percutanous nephrostomy with balloon dilatation with antegrade Double J stenting with the use of ureteroscope and Ho:YAG laser to manage the ureteral injuries and related obstructive symptoms sustained during surgeries, where injuries and strictures generally are caused by the inappropriate suture passing through or surrounding the ureter.

The index case is 46 year old female patient who presented with back pain with heavy menstrual bleeding since last 2-3 months. She underwent evaluation under the Gynecologist in outside hospital. No abnormality detected on general physical examination. Initially pre operatively her creatinine level was 0.8 mg/dl. Ultrasound showed Didelphys uterus with uterine fibroid with bilateral (B/L) hydroureteronephrosis (HUN). So she underwent contrast enhanced CT scan which showed the Didelphys uterus with fibroid which was compressing both ureter resultant B/L HUN. After normal pre operative work up she underwent B/L Double J stenting followed by total abdominal hysterectomy (TAH) with bilateral salpingo-oopherectomy (BSO) through pfannenstiel incision with intra operative involvement of urologist for peri-ureteric dissection.

Immediate post operative blood investigations were within normal range. She was clinically stable. On 1st post operative day, her creatinine level was 1.2 mg/dl (increased as compared to pre op). She was vitally stable with complains of operative site pain. Catheter was removed on same day. After removal of catheter she developed stent related symptoms. She underwent DJ stents removal under local anesthesia on same day. Right side DJ stent was removed with difficulty and strain and left DJ stent could not be removed. From 2nd post operative day onwards she developed abdominal distention with subsequent rise in creatinine levels with decrease urine output. Within 4-5 days her creatinine level reached to 9.7 mg/dl and urine output nil with features of paralytic ileus. She was deteriorating vitally and developed tachycardia, hypotension and tachypnea. She was diagnosed as acute renal injury and given lasix challenge several time but was not responding to it. Finally she underwent 2 subsequent hemodialysis. Still she was not improving and developed signs of sepsis. She underwent ultrasound which showed

gross free fluid in abdominal cavity with B/L HUN. Then she referred to our center for further management.

On presentation she was vitally stable. Her pulse rate was 110/min and blood pressure was 100/70 mmHg. On admission to our center her Creatinine level was 4.4 mg/dl and total count was 22000 cells/cumm. She underwent CT urography (CT IVP) under antibiotic cover followed by hemodialysis. Conclusion of CT urography was features of renal pelvic injury both side at PUJ with contrast extravasation with urinoma with left DJ stent in situ. In Fig. 1, few CT cuts are presented

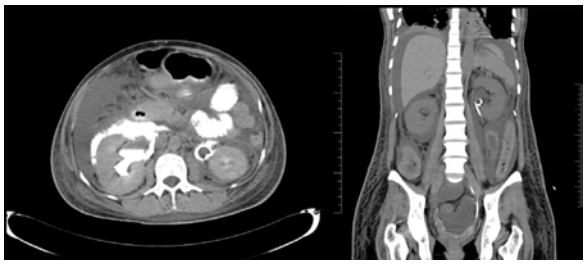

Fig. 1

As she was vitally stable she was shifted to cath lab. Right side mid pole calyx was accessed percutaneously using 22G Chiba needle. Calyx was confirmed by ultrasound and contrast injected. A guide wire was passed into the ureter. Neff sheath passed over the wire. Urogram performed confirmed the intraureteric position with obstruction at the lower end. Guide wire was passed into the bladder and the lesion was predilated with 4 mm Percutaneous transluminal angioplasty (PTA) balloon. 8 F DJ stent was deployed in right ureter over the guidewire. 8F multipurpose drainage catheter maintained in the right renal pelvis. Left mid pole calyx was accessed percutaneously using 22G Chiba needle. Calyx was confirmed by ultrasound and contrast injection. A guide wire was passed into the ureter. After serial dilatation of the tract, 8 F nephrostomy drainage catheter was positioned into the left renal pelvic region. So after this procedure she had B/L DJ stent in situ with B/L nephrostomies in situ. On 1st post op day her creatinine drastically reduced to 0.4 mg/dl. Urinoma was also drained ultrasound guided. She was improved. On 3rd post op day she underwent nephrostomogram which suggestive of both side DJ stent in situ and right side peri

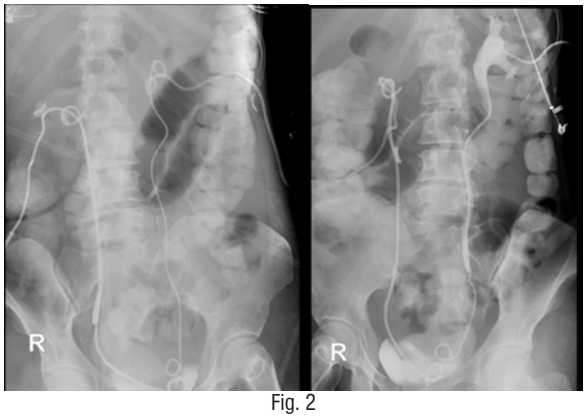

Fig. 2

stent drainage present but left side peri stent drainage was absent. In Fig. 2 nephrostomogram is presented.

On 5th day she underwent cystoscopy under general anesthesia. Both side DJ stent were in stu. Left side DJ stent was tried to pull out but could not be removed. Right side 6/7.5 semirigid ureteroscopy (URS) done alongside DJ stent. No area of narrowing noted and scope was passed upto Right renal pelvis and coil of right PCN tube seen. Left URS done alongside the DJ stent. An area of kinking was seen at about 6-7 cm from vesicoureteric junction. Guidewire was not negotiable beyond this point. A short segment of vicryl suture material was seen in the lumen of ureter which was constricting and kinking the stent. This suture loop was divided using Ho:Yag

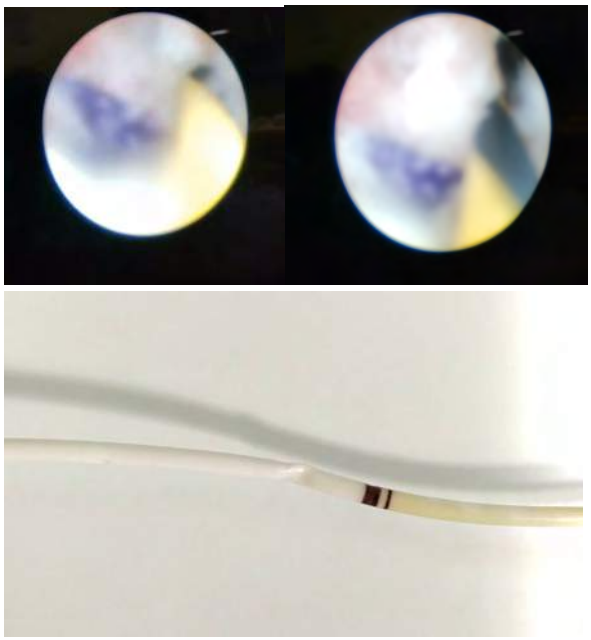

Fig. 3

laser energy and left DJ stent removed. Left URS done after passing guidewire into left kidney under fluroscopy guidance. Left side 8 Fr DJ stent passed over guide wire under fluoroscopy guidance. Coil confirmed in renal pelvis. Intraoperative picture showed in Fig. 3.

Both nephrostoies were removed 2 days after URS and patient was discharged. On the day of discharge patient's creatinine was 0.7 mg/dl. Patient was asymptomatic. Patient was followed up 2 weeks, 4 weeks and 8 weeks after discharge. On each follow up creatinine level was normal and urine culture was sterile. After 3 month of discharge ultrasound was done which suggestive bilateral DJ stents in situ and no dilatation in both pelvicalyceal systems. Creatinine was 0.6 mg/dl. Urine culture was sterile. On same follow up both DJ stents were removed. After 6 months of discharge, she was again followed up. Creatinine was 0.8 mg/dl and urine culture was sterile. She underwent intravenous pyelography which suggestive of normal functioning both kidneys with no evidence of obstruction or leakage. Eventually, this novel and innovative minimal invasive technique has worked for the patient without adding any significant morbidity to patient.

Discussion

The ureter is a vulnerable anatomic structure which can be easily compromised in the retroperitoneal space during general, gynecologic, and urologic surgeries. The traditional open surgical techniques such as end-to-end ureteroureterostomy with or without psoas hitch or Boari flap for the iatrogenic ureteral injuries have shown the obvious drawbacks of relatively high rate of severe complications and poor vascular blood supply in the distal ureters[20]. The increasing applications of endourological approaches to treating lower ureteral strictures have become routinely techniques over the last decade. Currently, various modified techniques have been introduced to incise ureteral strictures based on the utilization of hydrophilic apparatus that can facilitate the traversal of strictures, whereas classic balloon dilation management cannot solve hemostasis problem, as well as the electrocautery techniques which might lead to adjacent tissues damage[21]. Surprisingly, in our clinical practice, the Ho:YAG laser has been revealed to be an outstanding instrument for incising the sutures during the ureteroscopy, as the penetration depth of the energy is under well-control; besides minimal scarring and precise cutting are also valid. A few studies have reported series of approaches such as using antegrade and retrograde to incise or eliminate the ureteral strictures[21,22]. However, to the best of our knowledge, the combined application of ureteroscope, Ho:YAG laser, and balloon dilation to incise and simultaneously remove the sutures responsible for the strictures or obstructions has not been previously elucidated. This communication illustrates that lower iatrogenic ureteral injury with strictures or obstructions caused by the aberrant sutures can be effectively managed by minimally invasive endourological approaches.

Conclusion

This case is being presented to highlight the utility of laser in dealing with sutures that have gone through the ureters and to emphasize correlation of clinical details to arrive at a correct diagnosis. With minimally invasive techniques, we could avoid open exploration or a probable surgery for ureteric stricture later.

References

1. J.Li,Z.Chen,Q.Zhu,Y.Zhao,H.Wang,andW.Liu,"Early repair of pelvic and abdominal nonurological surgery-induced iatrogenic ureteral injuries in three distinct waiting-for-repair time periods," The American Surgeon,vol.78,no.11,pp.1270– 1275, 2012.
2. H.A.Mahendran,P.Singam,C.Ho,G.E.Hong,T.G.Hee, and Z. Md Zainuddin, "Iatrogenic ureter injuries: eleven years experience in a tertiary hospital," Medical Journal of Malaysia, vol.67,no.2,pp.169–172,2012.
3. K. A. Mteta, J. Mbwambo, and M. Mvungi, "Iatrogenic ureteric and bladder injuries in obstetric and gynaecologic surgeries," East African Medical

Journal,vol.83,no.2,pp.79–85,2006.

4. Halabi,M.D.Jafari,V.Q.Nguyenetal.,"Ureteralinjuriesin colorectal surgery: an analysis of trends, outcomes, and risk factors over a 10-year period in the United States," Diseases of the Colon and Rectum,vol.57,no.2,pp.179–186,2014.
5. M. Rafique and M. H. Arif, "Management of iatrogenic ureteric injuries associated with gynecological surgery," International Urology and Nephrology,vol.34,no.1,pp.31–35,2002.
6. H. Tezval, M. Tezval, C. von Klot et al., "Urinary tract injuries in patients with multiple trauma," World Journal of Urology,vol. 25,no.2,pp.177–184,2007.
7. N. C. Palaniappa, D. A. Telem, N. E. Ranasinghe, and C. M. Divino, "Incidence of iatrogenic ureteral injury after laparoscopic colectomy," Archives of Surgery,vol.147,no.3,pp.267– 271, 2012.
8. T.H.Lynch,L.Martinez-Pi˜neiro, E. Plas et al., "EAU guidelines on urological trauma," European Urology,vol.47,no.1,pp.1–15,2005.
9. P. Sakellariou, A. G. Protopapas, Z. Voulgaris et al., "Management of ureteric injuries during gynecological operations: 10 years experience," European Journal of Obstetrics Gynecology and Reproductive Biology,vol.101,no.2,pp.179–184,2002.
10. J.-S.Paick,S.K.Hong,M.-S.Park,andS.W.Kim,"Management of postoperatively detected iatrogenic lower ureteral injury:should ureteroureterostomy really be abandoned?" Urology,vol. 67, no. 2, pp. 237–241, 2006.
11. S. Witters, M. Cornelissen, and R. Vereecken, "Iatrogenic ureteral injury: aggressive or conservative treatment," The American Journal of Obstetrics and Gynecology,vol.155,no.3,pp.582– 584, 1986.
12. C. de Cicco, R. Schonman, M. Craessaerts, B. van Cleynenbreugel, A. Ussia, and P. R. Koninckx, "Laparoscopic management of ureteral lesions in gynecology," Fertility and Sterility, vol. 92, no. 4, pp. 1424–1427, 2009.
13. E. N. Liatsikos, D. Karnabatidis, K. Katsanos et al., "Ureteral injuries during gynecologic surgery: treatment with a minimally invasive approach," Journal of Endourology,vol.20,no. 12, pp. 1062–1067, 2006.
14. Erhard MJ, Bagley DH. Urologic applications of the holmium laser: Preliminary experience. J Endourol 1995;9:383.
15. Singal RK, Denstedt JD, Razvi HA, et al. Holmium:YAG laser endoureterotomy for treatment of ureteral stricture. Urology 1997; 50:875.
16. Biyani CS, Cornford PA, Powell CS. Ureteroscopic endopyelotomy with the holmium:YAG laser: Mid-term results. Eur Urol 2000;38: 139.
17. Giddens JL, Grasso M. Retrograde ureteroscopic endopyelotomy using the holmium:YAG laser. J Urol 2000;164:1509.
18. Hibi H, Kato K, Mitsui K, et al. Endoscopic ureteral incision using the holmium:YAG laser. Int J Urol 2001;8:657.
19. Zongyao Hao et al. Minimally Invasive Management of Iatrogenic Ureteral Injuries with Ureteroscope Facilitated by Holmium Yttrium-Aluminum-Garnet Laser - International Scholarly Research Notices Volume 2014, Article ID 307963, 4 pages
20. D.Koukouras,T.Petsas,E.Liatsikosetal.,"Percutaneous minimally invasive management of iatrogenic ureteral injuries," Journal of Endourology,vol.24,no.12,pp.1921–1927,2010.
21. P. Simon, M. A. Roder, P. Aslan, M. Brown, and W. Lynch, "A case of iatrogenic ureteric injury presenting with headache," Nature Clinical Practice Urology,vol.5,no.2,pp.113–116,2008.
22. J. H. Hong, S. S. Jeon, and K.-S. Lee, "Result of endoscopic ureteroureterostomy with holmium: YAG laser for complete ureteral obstruction," Journal of Endourology,vol.19,no.8,pp.979–983, 2005.

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An unusual case of recurrent hemoptysis with normal chest x-ray

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Abstract

A 39 year old lady presented with complaints of two episodes of streaky hemoptysis nine months apart. Her clinical examination was unremarkable and chest x-ray was normal. CT chest subsequently showed an endobronchial lesion in her left lower lobe bronchus and bronchoscopic biopsy was suggestive of a low grade neuroendocrine tumour. She underwent a left lower lobe sleeve lobectomy surgery and histopathology with immunohistochemistry confirmed the diagnosis of a Pulmonary Atypical Carcinoid tumour. She resumed her work 3 weeks later and is having a good quality of life. She is under regular follow-up from our department.

In the span of last two months, we had two other similar cases of Pulmonary Atypical Carcinoid tumour with different outcomes.

Keywords: Hemoptysis with normal chest x-ray, Carcinoid tumour.

Introduction

Lung neuroendocrine tumours are malignant neoplasms in adults that are characterised by neuroendocrine differentiation and indolent clinical behaviour. The term “Carcinoid” is still used to describe these tumours; however, preferred terms include neuroendocrine tumours (NET) or neuroendocrine neoplasm (NEN).

As per the latest 2015 WHO classification of lung tumours, pulmonary neuroendocrine tumours are classified under epithelial cell tumours of lung. Like NETS at other body sites, lung NETS are thought to be derived from peptide- and amine-producing neuroendocrine cells. NETS can arise at a number of sites throughout the body, including the thymus, lung, gastrointestinal tract, and ovaries. The gastrointestinal tract is the most frequently involved site, while lung is the second most common site[1].

Case presentation

39 year old lady, working as an office staff, eight months lactating mother, with h/o multinodular goitre s/p total thyroidectomy in 2016, on thyroxin 75mcg daily, initially presented to department of ENT with complaints of a single episode of streaky hemoptysis.

She had a similar single episode of streaky blood stained expectoration nine months earlier during late part of third trimester of pregnancy (which was not evaluated further according to the patient because it was only a single episode of blood streaking of sputum and it did not recur. According to her, it had been attributed to the low dose aspirin which she was taking daily from her first trimester as advised by her obstetrician at local hospital).

As there was no upper airway cause of her complaints, she was referred to department of Respiratory Medicine for further evaluation.

Her general and systemic clinical examination was unremarkable. Routine blood investigations including coagulation profile were normal. A chest x-ray was taken which was normal (fig. 1).

Although her complaints were only two episodes of streaky hemoptysis nine months apart with no further recurrence and her chest x-ray was normal, she was evaluated further with a CT chest, which showed an

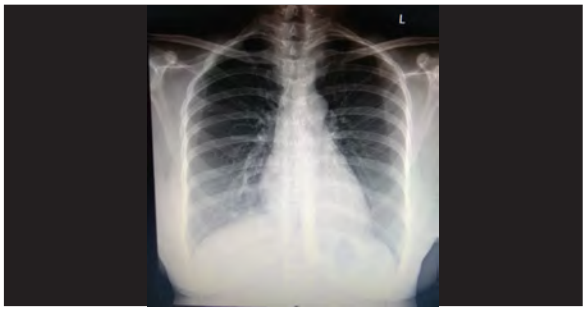

Fig. 1

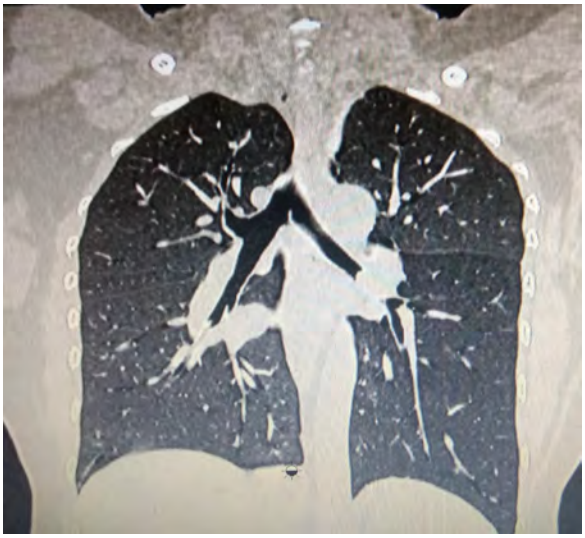

Fig. 2(a)

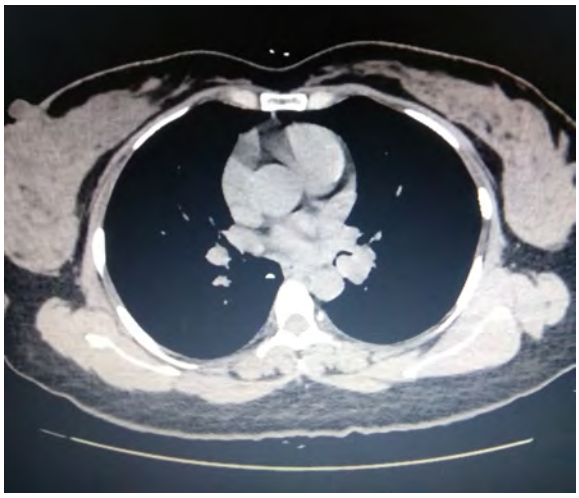

Fig. 2(b)

endobronchial lesion in her left lower lobe bronchus with heterogenous enhancement post contrast (fig. 2(a), 2(b).

She underwent fibre optic bronchoscopy which showed a fleshy intraluminal growth in her left lower lobe bronchus, bleeding to touch (fig. 3). Biopsy was taken from the same and hemostasis was achieved using Argon Plasma coagulation. Histopathological examination with Immunohistochemistry of biopsy specimen was suggestive of a low grade Neuroendocrine tumour (fig. 4). She was then subjected to a whole body 68 Gallium Dotatate Pet/CT scan which revealed uptake from the lesion (fig. 5a, 5b) and no other lesions elsewhere in the body.

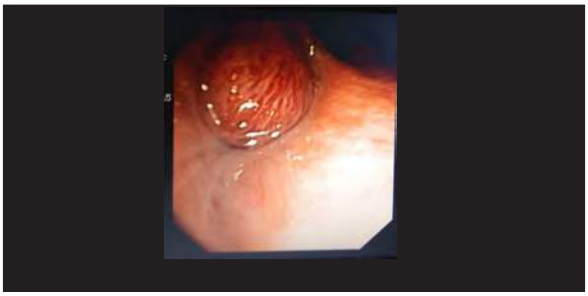

Fig. 3

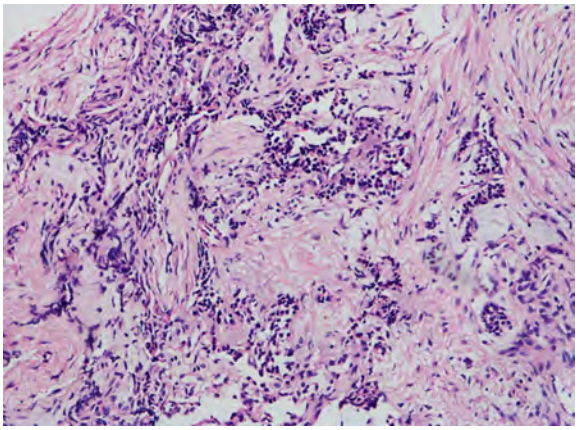

Fig. 4
(Image courtesy: Dept. of Pathology, KIMS Trivandrum)

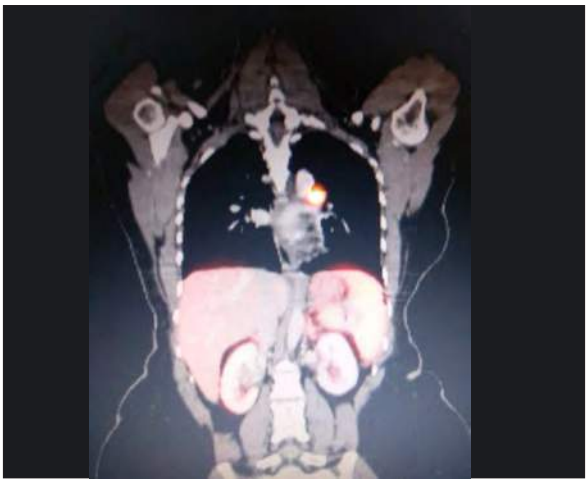

Fig. 5a

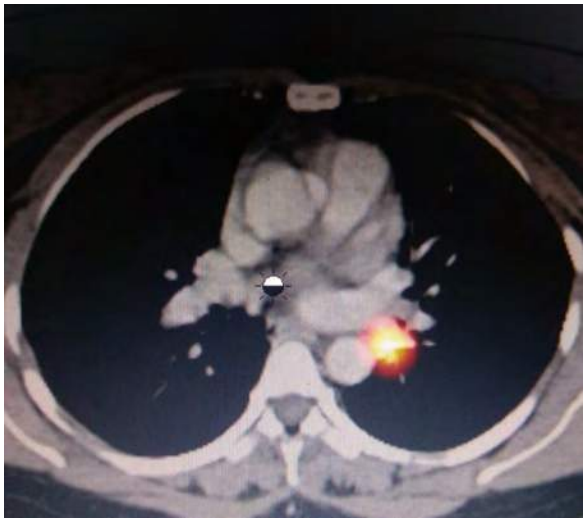

Fig. 5b

She was referred to CTVS department and underwent a left lower lobe sleeve lobectomy. Histopathology with immunohistochemistry confirmed the diagnosis of a Pulmonary Atypical Carcinoid tumour (fig. 6, 7); and resected margins and a hilar lymph node were free of neoplasm.

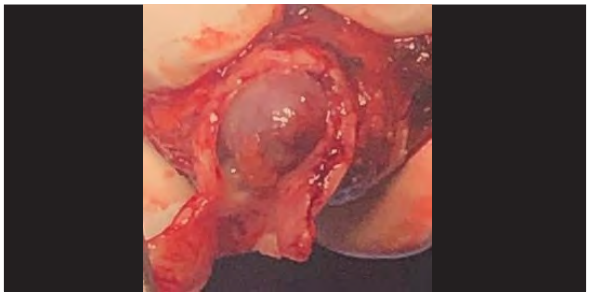

Fig. 6

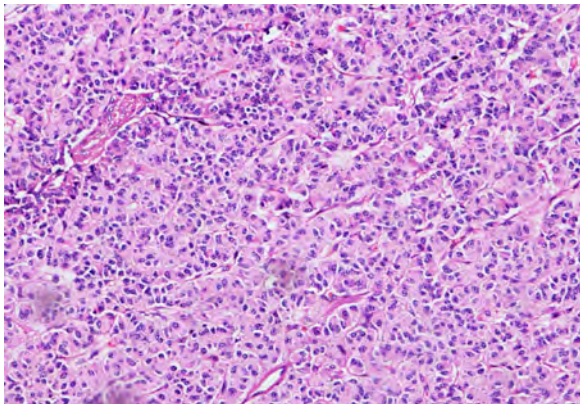

Fig. 7
(Image courtesy: Dept. of Pathology, KIMS Trivandrum)

Post-operative period was uneventful and she resumed her work three weeks later and is having a good quality of life. She is under regular follow-up from our department Follow-up chest x-ray (fig. 8) shown below.

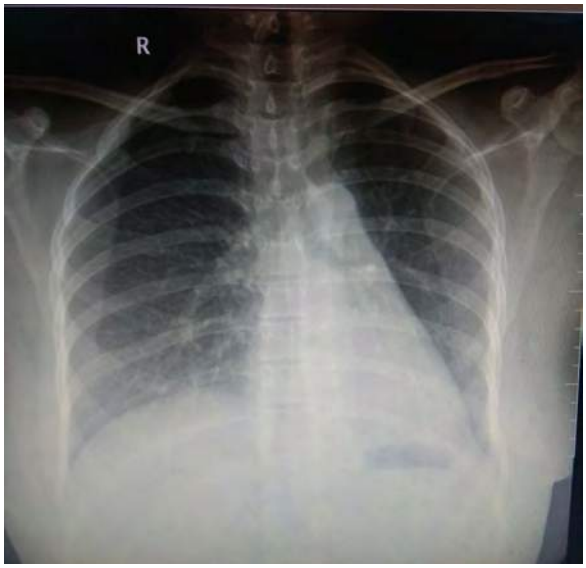

Fig. 8

In the span of last two months, we had two other similar cases of Pulmonary Atypical Carcinoid tumour.

The first amongst these two patients had being evaluated at multiple local hospitals for recurrent left sided pneumonia for the past one year; and on evaluation with CT chest and bronchoscopic biopsy from our department, a diagnosis of Pulmonary Atypical Carcinoid left lower lobe bronchus was made. She had to undergo a left pneumonectomy for adequate tumour clearance (fig. 9) is the post operative chest x-ray.

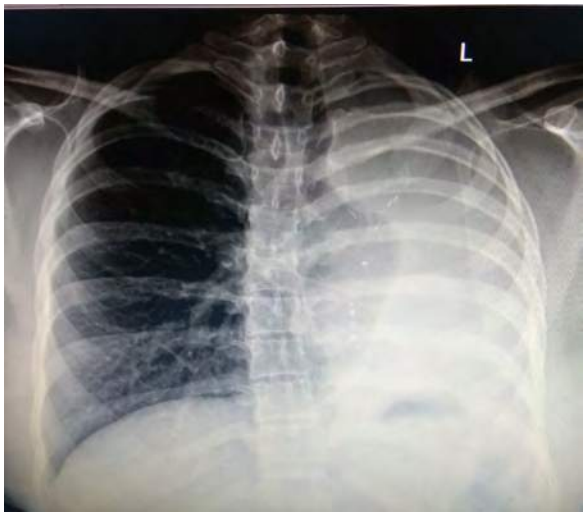

Fig. 9

The second patient, also a case of streaky hemoptysis with normal chest x-ray, was evaluated similarly from our department and a diagnosis of Pulmonary Atypical Carcinoid tumour was made from bronchoscopic biopsy. She was advised surgical resection, but was unwilling for the same. She did not come for further follow-up. Upon telephonic enquiry in March 2020, she did not undergo surgery yet and is remaining asymptomatic.

Discussion

Lung Neuroendocrine tumours (NETs) account for approximately 1 to 2 percent of all lung malignancies in adults and roughly 20 to 30 percent of all NETS[2]. Association between lung NETS and smoking is unclear. No other known carcinogens or exposure to environmental agents has been implicated in carcinogenesis. Patient symptoms depend on location and size of tumour, and patents may either be asymptomatic or have complaints of cough, breathlessness, wheeze, hemoptysis, and obstructive pneumonia. Carcinoid syndrome is relatively uncommon, but it can occur both in patients with locoregional or disseminated disease[3].

CT of the chest is the most useful imaging procedure, and the diagnosis is generally confirmed either by bronchoscopic biopsy (for central lesions) or by transthoracic needle biopsy (for peripheral lesions). These tumours show over expression of somatostatin receptors (Sstrs) on their cell membrane and Sstr-based 68 Gallium-Tetraazacyclododecane tetraacetic acid (DOTA)–peptide PET/CT is an exciting imaging modality that has shown significant advantages over conventional imaging in diagnosis and management of NETS[4].

Histologically, lung NETS are composed of cytologically bland cells containing regular round to oval nuclei with finely dispersed chromatin and inconspicuous small nucleoli. The cells are usually polygonal in shape and are arranged in distinct organoid, trabecular, or insular growth patterns with a delicate vascular stroma. The terms 'typical' and 'atypical' are used to describe low-versus intermediate grade tumours arising in the lung. The mitotic rate (rather than Ki-67 index) and presence of necrosis are the primary determinants of grade. Diffuse idiopathic pulmonary neuroendocrine cell hyperplasia (DIPNECH) is a generalized proliferation of pulmonary neuroendocrine cells that may be confined to the mucosa of the airways, invade locally to form "tumorlets," or develop into invasive NETS (Carcinoid tumours). Immunohistochemical identification of secreted and cytoplasmic products such as synaptophysin, neuron-specific enolase, and chromogranin can confirm neuroendocrine differentiation. Approximately 50 percent of lung NETS stain positive for thyroid transcription factor 1 (TTF1), although the staining is often weak and focal[5].

Lung NETS are staged using the same TNM classification from the combined American Joint Committee on Cancer (AJCC)/Union for International Cancer Control (UICC) that is used for bronchogenic lung carcinomas. Low-grade (typical) lung NETS seldom metastasize and have an excellent prognosis; intermediate-grade (atypical) lung NETS have a higher likelihood of metastases and a worse prognosis[6].

Surgery is the treatment of choice for lung NETS offering the best chance of cure. In general, anatomic resections and lung-sparing techniques (eg, sleeve resection) are preferred. However, lobectomy, bilobectomy, and pneumonectomy are justified especially in those located proximally. After surgical resection of the tumour, follow-up for cancer is conducted in a manner similar to that for other lung cancers[7]. Other treatment options depending on staging of the tumour include somatostatin analotues therapy, radiotherapy, chemo/radioembolization, radiofrequency ablation etc.

The 10-year survival rate for typical and atypical pulmonary Carcinoid tumours are 82-87%.and 35-60% respectively[8].

Conclusion

Hence, patients with mild hemoptysis should always be evaluated in detail even if their clinical and radiological examination are normal.

References

1. Kulke MH, Mayer RJ. Carcinoid tumors. N Engl J Med 1999; 340:858.
2. Modlin IM, Lye KD et al. A 5-decade analysis of 13,715 carcinoid tumors. Cancer. 2003; 97(4):934.
3. Leoncini E, CarioliG et al. Risk factors for neuroendocrine neoplasms: a systematic review and meta-analysis. Ann Oncol 2016; 27:68.
4. Krenning EP, Bakker WH, Breeman WA, et al. Localisation of endocrine-related tumours with radioiodinated analogue of somatostatin. Lancet 1989; 1:242–244
5. Skuladottir H, Hirsch FR et al. Pulmonary neuroendocrine tumors: incidence and prognosis of histological subtypes. A population-based study in Denmark. Lung Cancer 2002; 37:127.
6. Gustafsson BI, Kidd M, Chan A, et al. Bronchopulmonary neuroendocrine tumors. Cancer 2008; 113:5.
7. Terzi A, Lonardoni A, Feil B, et al. Bronchoplastic procedures for central carcinoid tumors: clinical experience. Eur J CardiothoracSurg 2004; 26:1196.
8. Thomas CF Jr, Tazelaar HD, Jett JR. Typical and atypical pulmonary carcinoids : outcome in patients presenting with regional lymph node involvement. Chest 2001; 119:1143.

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An itchy rash on the buttock

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Abstract

Chromoblastomycosis, most common endemic implantation (subcutaneous) mycoses, is a chronic, indolent, granulomatous fungal disease caused by the transcutaneous inoculation of spores from several species of melanized fungi. The diverse clinical findings of chromoblastomycosis mimic other infectious or noninfectious diseases. No randomized trials have evaluated treatments for chromoblastomycosis. Treatment is recommended but can be challenging.

Here we present the case of a 47 year old gentleman who presented with recurrent itchy rash on the buttock who threw us a diagnostic as well as therapeutic dilemma.

Keywords: Chromoblastomycosis, Itraconazole, Treatment duration

Introduction

Chromoblastomycosis is a chronic fungal infection affecting the cutaneous and subcutaneous tissue in tropical and subtropical climates and is one of the most common endemic implantation (subcutaneous) mycoses[1]. The most common causative organisms are Fonsecaeapedrosoi and Cladophialophora carrionii.[2-4]. These fungi can be found in soil and plant fragments, and infection usually results from cutaneous trauma.[5-9]. Patients may not recall the inciting trauma[1,5]

A variety of fungal virulence factors likely contribute to infection, including the ability of causative fungi to acquire a muriform cell architecture in tissue.[1]Muriform cells (also known as sclerotic bodies, Medlar bodies, fumagoid bodies, copper pennies, chestnut cells, and meristematic cells) are resistant to host defenses and facilitate persistence of infection[10]

The clinical manifestations are polymorphic.[1,11] .First appear at the site of inoculation weeks to months after trauma. Most often, the infection occurs in trauma-prone sites, such as exposed areas of the lower and upper limbs. Involvement of shoulders, trunk, buttocks, eyelids, nose, or ears also has been rarely reported[1,12-14] Early disease often presents as erythematous macules or papules that develop verrucous or hyperkeratotic features . Pruritus is common and may be accompanied by local pain.

Without treatment, the infection slowly progresses to larger areas of skin involvement with nodular, verrucous, tumoral, plaque, or scar-like morphologies[1]Tissue fibrosis, lymphedema, secondary infection, and malignancy are potential complications.[15,16] Internal organ involvement is rare.[17]

Diagnosis- confirmed through the detection of muriform cells in a potassium hydroxide preparation or skin biopsy[18,19]. Fungal cultures are used to identify the causative organism[13].

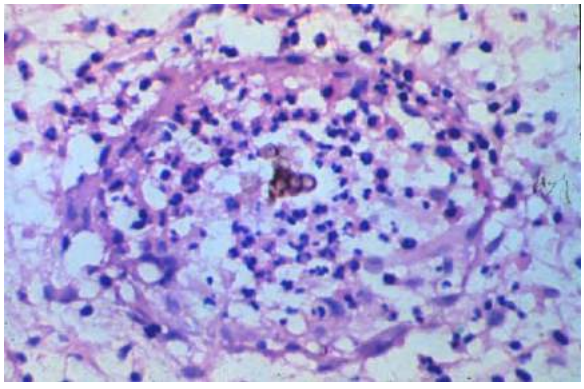

Fig 1

Treatment can be challenging. For small, solitary, well-defined lesions, surgical excision is the initial treatment (Grade 2C). For more extensive disease, oral antifungal therapy rather than surgical excision as initial treatment (Grade 2C). Itraconazole and terbinafine are the most common oral antifungal therapies. Other interventions, such as cryotherapy, heat therapy, and light-based therapies, may be useful as adjunctive treatments[1,20-28]

Long-term follow-up is necessary to determine whether patients have achieved cure. Periodic clinical, mycologic, and histologic assessments are needed.[1,5,29,30]

Case presentation

47 year old gentleman, with no known comorbidities presented to Dermatology OP with complaints of raised wart like plaque on the right buttock since 2 years associated with mild pruritus. He gave no history of precedent trauma. He was initially evaluated in a nearby hospital where a biopsy was done and was diagnosed as Chromoblastomycosis and he was treated with oral antifungal (Itraconazole) for 2 weeks with some relief. Later the lesion recurred and gradually increased in size. This time he sought medical help elsewhere (Mumbai), where a repeat biopsy was in favour of Lupus vulgaris and he was referred to KIMS for treatment.

A semilunar plaque of 4 cm diameter with irregular margins and warty surface.

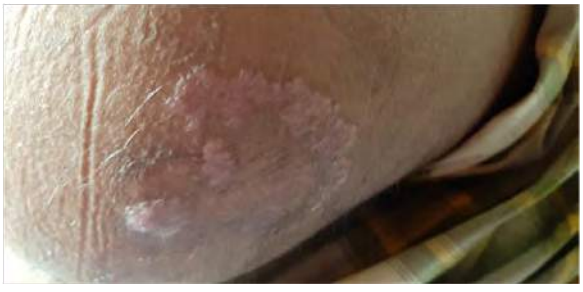

Fig 2

Considering differentials of Chromoblastomycosis and Lupus vulgaris we went forward with the investigations. Mantoux was done and reported as negative. Discussed with Dermatopathologist who had a relook of the slides and reported chromo bodies and affirmed the diagnosis as chromoblastomycosis.

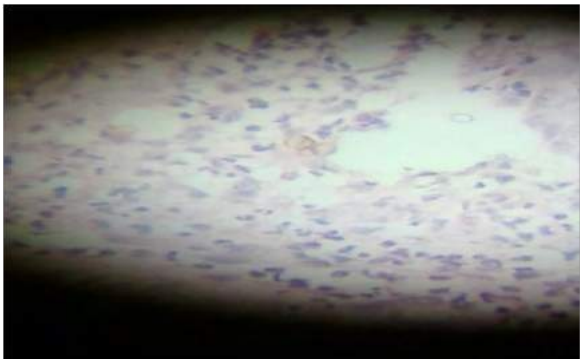

Fig. 3

Discussed with patient regarding the treatment and the need for prolonged treatment with oral antifungals (Itraconazole – atleast 6 months) and also the need for frequent checking of his blood parameters- Complete blood counts (CBC), Renal function tests (RFT) and Liver function tests (LFT) and initiated him on Tab. Itraconazole 200mg twice daily and kept him on fortnightly follow up.

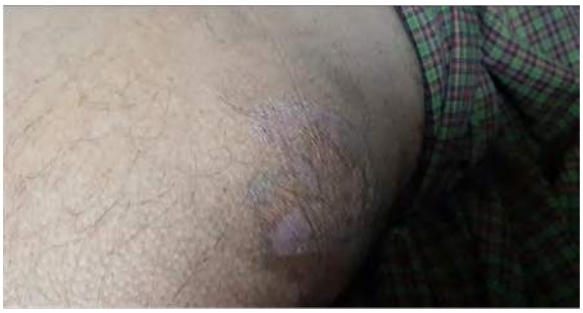

Fig. 4

On completing 5 months of twice daily oral antifungal therapy there was complete resolution of the lesion with only scar tissue visible. His blood parameters were within normal limits and Itraconazole was discontinued.

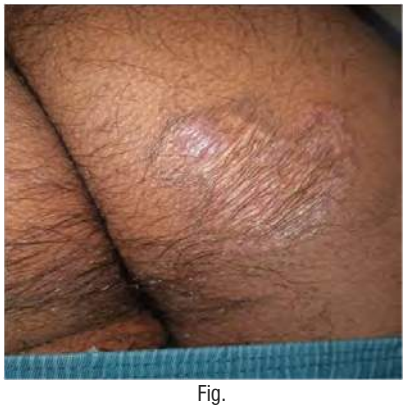

Fig.

Discussion

Our case on presentation posed a diagnostic challenge- Chromoblastomycosis vs Lupus Vulgaris, owing to the rarity of the condition and unusual presentation on an unusual site (buttocks). A high index of clinical suspicion and reexamining the biopsy slide for thechromo bodies helped in confirming the diagnosis.

The next step in management was educating the patient regarding the condition and treatment schedule.

It was a therapeutic dilemma as the duration of treatment is not mentioned anywhere in theliterature and also our patient was already treated with oral antifungals for a short durationelsewhere and prescribing Itraconazole for longer periods posed a threat.

In the end evidence based diagnosis and a proper treatment with proper follow up and monitoring gave the result.

References

1. Queiroz-Telles F, de Hoog S, Santos DW, et al. Chromoblastomycosis. ClinMicrobiol Rev 2017; 30:233.
2. De Hoog GS, Attili-Angelis D, Vicente VA, et al. Molecular ecology and pathogenic potential of Fonsecaea species. Med Mycol 2004; 42:405.
3. Yaguchi T, Tanaka R, Nishimura K, Udagawa S. Molecular phylogenetics of strains morphologically identified as Fonsecaeapedrosoi from clinical specimens. Mycoses 2007; 50:255.
4. Badali H, Gueidan C, Najafzadeh MJ, et al. Biodiversity of the genus Cladophialophora. Stud Mycol 2008; 61:175.
5. Queiroz-Telles F, Nucci M, Colombo AL, et al. Mycoses of implantation in Latin America: an overview of epidemiology, clinical manifestations, diagnosis and treatment. Med Mycol 2011; 49:225.
6. Esterre P, Andriantsimahavandy A, Ramarcel ER, Pecarrere JL. Forty years of chromoblastomycosis in Madagascar: a review. Am J Trop Med Hyg 1996; 55:45.
7. Al-Doory Y. Chromomycosis. In: Occupational Mycoses, Di Salvo AF (Ed), Lea &Febiger, Philadelphia 1983. p.95.
8. Mehregan AH, Rudner EJ. Implantation dermatosis. Wood splinter with fungus contamination. J CutanPathol 1980; 7:330.
9. Tschen JA, Knox JM, McGavran MH, Duncan WC. Chromomycosis. The association of fungal elements and wood splinters. Arch Dermatol 1984; 120:107.
10. Rosen T, Gyorkey F, Joseph LM, Batres E. Ultrastructural features of chromoblastomycosis. Int J Dermatol 1980; 19:461.
11. Mugleston BJ, Usatine RP, Rosen T. Wide Morphologic Variability of Chromoblastomycosis in the Western Hemisphere. Skinmed 2016; 14:423.
12. Bonifaz A, Carrasco-Gerard E, Saúl A. Chromoblastomycosis: clinical and mycologic experience of 51 cases. Mycoses 2001; 44:1.
13. Queiroz-Telles F, Esterre P, Perez-Blanco M, et al. Chromoblastomycosis: an overview of clinical manifestations, diagnosis and treatment. Med Mycol 2009; 47:3.
14. Queiroz-Telles F, Santos DW, Pedroso C. Fungal infections of implantation (chromoblastomycosis, mycetoma, entomophthoramycosis, and lacaziosis). In: Diagnosis and Treatment of Fungal Infections, 2nd, Hospenthal DR, Rinaldi MG (Eds), Springer International Publishing, 2015. p.271.
15. Esterre P, Risteli L, Ricard-Blum S. Immunohistochemical study of type I collagen turn-over and of matrix metalloproteinases in chromoblastomycosis before and after treatment by terbinafine. Pathol Res Pract 1998; 194:847.

16. Ricard-Blum S, Hartmann DJ, Esterre P. Monitoring of extracellular matrix metabolism and cross-linking in tissue, serum and urine of patients with chromoblastomycosis, a chronic skin fibrosis. Eur J Clin Invest 1998; 28:748.

17. de Azevedo CM, Gomes RR, Vicente VA, et al. Fonsecaeapugnacius, a Novel Agent of Disseminated Chromoblastomycosis. J ClinMicrobiol 2015; 53:2674.

18. Banerjee U, Mohapatra AK, Sarkar C, Chaudhery R. Cladosporiosis (cerebral phaeohyphomycosis) of brain--a case report. Mycopathologia 1989; 105:163.

19. Khan I, Khan AR, Khan MS. Clinicopathological study of cutaneous chromoblastomycosis in Pakistan. Journal of Pakistan Association of Dermatologists 2012; 22:122.

20. Kumarasinghe SP, Kumarasinghe MP. Itraconazole pulse therapy in chromoblastomycosis. Eur J Dermatol 2000; 10:220.

21. Borelli D. A clinical trial of itraconazole in the treatment of deep mycoses and leishmaniasis. Rev Infect Dis 1987; 9 Suppl 1:S57.

22. Esterre P, Inzan CK, Ramarcel ER, et al. Treatment of chromomycosis with terbinafine: preliminary results of an open pilot study. Br J Dermatol 1996; 134 Suppl 46:33.

23. Tanuma H, Hiramatsu M, Mukai H, et al. Case report. A case of chromoblastomycosis effectively treated with terbinafine. Characteristics of chromoblastomycosis in the Kitasato region, Japan. Mycoses 2000; 43:79.

24. Bonifaz A, Saúl A, Paredes-Solis V, et al. Treatment of chromoblastomycosis with terbinafine: experience with four cases. J Dermatolog Treat 2005; 16:47.

25. Silva-Rocha WP, Cardoso FJ, Colalto W, et al. Clinical improvement of chromoblastomycosis refractory to itraconazole successfully treated with high dose of terbinafine. J Dermatol 2013; 40:775.

26. Xibao Z, Changxing L, Quan L, Yuqing H. Treatment of chromoblastomycosis with terbinafine: a report of four cases. J Dermatolog Treat 2005; 16:121.

27. Tsianakas A, Pappai D, Basoglu Y, et al. Chromomycosis--successful CO2 laser vaporization. J EurAcadDermatolVenereol 2008; 22:1385.

28. Hira K, Yamada H, Takahashi Y, Ogawa H. Successful treatment of chromomycosis using carbon dioxide laser associated with topical heat applications. J EurAcadDermatolVenereol 2002; 16:273.

29. Queiroz-Telles F, Santos DW. Challenges in the therapy of chromoblastomycosis. Mycopathologia 2013; 175:477.

30. Bayles MA. Chromomycosis. In: Bailliere's Clinical Tropical Medicine and Communicable Diseases: Tropical Fungal Infections, Hay RJ (Ed), WB Saunders, London 1986. p.45.

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Active surveillance for carcinoma prostate

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Carcinoma of prostate is the second most common cancer in males worldwide and sixth most common cause of cancer related death[1]. Management of carcinoma of prostate has always been a controversial topic amongst Urologists. There are several guidelines and multiple studies available for the same topic. This topic has always been a debatable one, especially for low and intermediate risk groups of patient with life expectancy less than 10 years because carcinoma of prostate is slow growing tumor.

There are two types of observational strategies described till now for Ca prostate: 1) Watchful waiting and 2) Active Surveillance. Watchful waiting was used for Ca prostate in pre PSA era when patients mostly presented in an incurable stage and available options were associated with high mortality rate. Nowadays watchful waiting would be considered preferable for all men without high-risk disease who have less than a 5-year life expectancy, and an option for those without high-risk disease and a life expectancy below 10 years. Most urologists today would favor active surveillance (not watchful waiting) as an observational strategy for men without high-risk disease and a life expectancy of 5 to 10 years[2].

PSA based screening for Ca prostate led to early detection of prostate cancer known as stage migration which led to an alteration of the natural history of disease leading to a decrease in the burden of cancer related mortality[3]. The outcomes of moderately and poorly differentiated carcinoma managed without treatment in the pre PSA and in the PSA era were compared by Yu-Yao et al in 2009[4]. The results were suggestive low mortality rates in PSA based screening era. The 15-year prostate cancer mortality in the PSA era was estimated to be 0% to 2% for men aged 55 to 74 years with Gleason score 6 or less managed conservatively[5]. These low-grade prostate cancers make up two of three cancers found with initial PSA-based screening, and three of four or more with follow-up screening using 1- to 4-year screening intervals[6,7]. Thus, the pool of disease carrying a low risk of cancer-related death without treatment over a 10 to 15 year period is large with PSA-based screening.

Etzioni and associates[8] estimated that early detection due to PSA based screening accounted for 45% to 70% of the prostate mortality decline in the United States. If half of the 40% mortality decline in the United States were due to PSA-based screening, the mortality reduction would approximate the relative mortality reduction of 20% that was observed in the European Randomized Study of Screening for Prostate Cancer (ERSPC)[9].

Few studies including the Prostate Cancer Outcomes Study[10], have evaluated the outcomes of men undergoing radiotherapy and surgery for localized prostate cancer after a diagnosis. There was significant decline in urinary, sexual, and bowel function noted after 15 years of primary treatment. These quality-of-life declines, in urinary, bowel, and sexual function occur to a significantly greater extent among those who undergo treatment for prostate cancer as compared to a normal aging

population without a diagnosis of prostate cancer, and symptom distress is more common among men with prostate cancer who are treated compared to those not treated[11,12].

The term overdiagnosis and overtreatment we have to dissect more in view of understanding importance of active surveillance for Ca prostate. The detection of a cancer that would otherwise not have been diagnosed in the lifetime of the host is defined as overdiagnsis. Treatment of men who would otherwise not have known about their cancer in the absence of PSA testing is overtreatment. Incidents of overdiagnosis and overtreatment is increasing due to PSA based screening, patient awareness and availability of minimal invasive techniques for localized Ca prostate. Definitive procedures for localized Ca prostate can significantly reduce quality of life of patient as compared to no treatment. Overtreatment exacts a cost to the health care system and potential harm to a patient, with no benefit. For a previously asymptomatic patient, the cost can be substantial in terms of quality of life, and thus the bar should be high for determining the need for curative intervention. Several studies noted in support of this statement[13,14].

The Scandinavian Prostate Cancer Group Study Number 4 (SPCG-4) randomized 695 men (mean age 65 years) to observation versus radical prostatectomy; 5% were diagnosed through PSA-based screening, 3 of 4 had palpable disease, and the mean PSA level was 13 ng/mL at diagnosis[15]. After 15 years of follow-up, men who underwent surgical treatment had significantly lower rates of distant metastatic disease and death from prostate cancer—an absolute between-group difference of 11.7% and 6.1%, respectively. Among men below age 65 years, there was a significant absolute between-group difference in rates of distant metastatic disease and prostate cancer death of 18.3% and 9.4%. However, for men aged 65 years and above, surgery did not provide a benefit in terms of freedom from metastatic disease or prostate cancer death over 15 years of follow-up. Out of seven men with low-risk disease who underwent surgery and died of prostate cancer, tumors in six patients were upgraded to Gleason score 7 or 8 at radical prostatectomy, further evidence that death from prostate cancer among men with low-risk disease occurs more likely from unrecognized high-grade disease rather than progression of low-grade to high-grade disease[15].

The Prostate Cancer Intervention versus Observation Trial (PIVOT) randomized 731 men diagnosed with localized prostate cancer to radical prostatectomy or observation (mean age 67 years; median PSA 7.8 ng/mL)[16]. While follow-up through 12 years revealed no all-cause or cancer-specific mortality reduction with surgery, a subset analysis suggested an all-cause mortality reduction with surgery for men with a PSA above 10 ng/mL and those with intermediate to high-risk disease[16].

The findings from the SPCG-4 study and PIVOT signify that for older men with low-risk disease, especially those with associated comorbidities unlikely to benefit from curative intervention. For these men, no treatment may be the most rational initial management considering that harm (quality-of-life decrement) is likely to outweigh any benefit (prostate cancer mortality reduction). The ProtecT Study is one of the largest study going on in this field. It is in 3rd phase. They released primary results which were in favour of active surveillance. Final result of this study would change further perception of active surveillance and we can expect clear cut guidelines for AS[17].

Following the PRISMA statement guidelines (http://www.prisma-statement.org/), we carried out a systematic review of AS for PCa in the literature using the Kerala Institute of Medical Sciences' electronic database PubMed (www.pubmed.gov). We carried out a search in English using the terms: active surveillance, prostate cancer,

watchful waiting and conservative management.

Selection criteria for Active Surveillance

The National Comprehensive Cancer Network (NCCN) guidelines for carcinoma prostate risk stratification provided by Mohler et al,2012[2] are depicted below in Table - 1. This risk stratification is based on patient's T stage of cancer, PSA, PSA density, Gleason score and biopsy.

Active surveillance can be offered to very low-, low- and intermediate-risk groups of patients or localized carcinoma of prostate to whom aggressive local therapy could be offered with intent to intervene if the disease progresses[18]. Active surveillance is not advisable for high risk patients or those with primary Gleason score 4 or 5, who have substantial risk of harbouring systemic disease[19]. The NCCN now recommends active surveillance as primary treatment for men with "very" low-risk prostate cancer defined by PSA level 10 ng/mL or less, clinical stage T2a or less, Gleason grade 3+3 or less, PSA density 0.15 ng/mL per gram or less, two or fewer cores positive, and any single-core positivity 50% or less. The NCCN guidelines recommend offering the option of active surveillance for men presenting with low-risk disease defined as PSA level 10 ng/mL or less, clinical stage T2a or less, and Gleason grade 3+3 or less[2]. The American and European Urologic Associations have similarly published guidelines for offering active surveillance for men with prostate cancer.

RISK PROFILE	CRITERIA
Favourable Very low risk	• T1c • Gleason score ≤6 • PSA <10 ng/mL • Fewer than 3 biopsy cores positive, ≤50% cancer in any core • PSA density <0.15 ng/mL per gram
Low risk	• T1 or T2a • Gleason score 2-6 • PSA <10 ng/mL
Intermediate risk	• T2b-T2c or • Gleason score 7 or • PSA 10-20 ng/mL
High risk	• T3a or • Gleason score 8-10 or • PSA >20 ng/mL

The Epstein criteria were selected to identify potentially low-risk tumors and are among the most popular used for patient selection for active surveillance. By these criteria, "insignificant" tumors are predicted by clinical Gleason pattern 3 or less, clinical stage T1c and either (1) PSA density 0.1 ng/mL per gram or less, two or fewer positive biopsy cores, and no cores with greater than 50% involvement or (2) PSA density of 0.15 ng/mL per gram or less and cancer smaller than 3 mm on only one biopsy core[20]. Patient factors such as age, comorbid illness, and willingness to adhere to surveillance strategies must also be considered during patient selection.

Eligibility characteristics described from the experiences of several large published cohorts are presented in following Table – 2[21-26].

	JOHNS HOPKINS (Tosolan et al, 2011)	UCSF (Porten et al, 2011)	PRIAS (Bul et al, 2013)	UNIVERSITY OF TORONTO (Klotz, 2012)	UNIVERSITY OF MIAMI (Eggener et al, 2013)	ROYAL MARSDEN (Selvadurai et al, 2013)	MEMORIAL SLOAN KETTERING (Eggener et al, 2013)
Entry criteria	PSA ≤10 ng/ML PSA density ≤0.15 ng/mL per gram Stage ≤T2a Grade ≤3+3 No. cores positive ≤2 Single-core positivity ≤50%	PSA ≤10 ng/mL Stage ≤T2a Grade ≤3+3 %Cores positive ≤1/3 Single-core positively ≤50%	PSA ≤10 ng/mL PSA density ≤0.2 ng/mL per gram Stage ≤T2a Grade ≤3+3 No. cores positive ≤2	PSA ≤10 ng/mL Grade ≤3+3	PSA ≤10 ng/mL Stage ≤T2a Grade ≤3+3 No. cores positive ≤2 Single-core positively ≤20%	PSA ≤15 ng/mL Stage ≤T2a Grade ≤3+4 % Cores positive ≤50	PSA ≤10 ng/mL Stage ≤T2a Grade ≤3+3 No. cores positive ≤3 Single-core positivity ≤50%

Prostatic Biopsy

Initial Biopsy - European urology guidelines for prostate biopsy recommend at least eight cores for glandular volumes of 30–40 mL[27]. A review of 87 studies, with a total of 20,698 patients, found that prostate biopsies that took between 10 to 12 cores led to significantly increased cancer detection rates compared with sextant biopsies. However, obtaining more than 12 cores did not appear to significantly improve the detection rate[28]. Other studies have shown that the number of biopsy cores is an independent predictor of the presence of insignificant tumors in the RP specimen[29], Ploussard et al. reported that

a 21-core protocol increased the rate of men that would be eligible for AS compared with a 12core approach without significantly increasing the rate of detected insignificant tumors[30]. According to the National Comprehensive Cancer Network guidelines, AS is suitable for patients at very low risk of tumor progression who have undergone a minimum of a 10-core baseline biopsy with PSA density of ≤0.15 ng/mL/g. PSA density appears to be associated with adverse histological findings when the biopsy is repeated, and also predicts the existence of insignificant PCa in the RP sample[2].

Identification of patients with purely low-grade cancer is problematic at present because of disease misclassification[31]. Thus the use of the term **progression** while on surveillance should be replaced with disease **reclassification**, since most patients meeting surveillance criteria who are found to have high-grade or more extensive disease on surveillance biopsies are thought to have been misclassified initially, rather than experiencing true disease progression[32].

One the largest study evaluating upgrading at radical prostatectomy, found that 36% of men with Gleason score 5-6 on needle biopsy were found to have higher-grade disease at radical prostatectomy[33]. The 10-year actuarial rate of upgrading on annual surveillance biopsies in a large active surveillance experience was approximately 30%[21]. The similarity in the rate of upgrading at radical prostatectomy, and reclassification to high-grade disease on annual biopsies over a decade for men with low-grade cancer, strongly suggest that initial misclassification is the more common reason for reclassification on surveillance, and not "true" disease progression from low to high grade. For this reason, some have recommended "confirmatory" biopsies and/or extensive biopsy strategies to reduce the risk of biopsy misclassification prior to considering active surveillance[34].

Confirmatory Biopsy - The risk of Ca prostate must be accurately assessed before a patient can be considered for inclusion into an active surveillance program. Most published protocols include a confirmation biopsy, especially when the baseline biopsy was not carried out using extended techniques. The time between baseline and confirmation biopsies varies between 3 and 12 months. Although some protocols postpone the confirmatory biopsy up to 1 year (Table 3), it should still be carried out early, especially if there is a possibility of high-risk features and/or insufficient prior sampling. Repeat biopsies carried out 3– 6 months after the initial procedure more clearly identify patients who would be ineligible for AS as a result of disease staging or volume[35]. The percentage of patients with tumors previously classified as low risk, but who are reclassified as being unsuitable for AS on confirmation biopsy varies between 16% at 6 months[36] and 27% at 3 months[37]. Motamedinia et al.[38] reported that the higher incidence of progression observed in repeat biopsies would likely be related to the higher number of cores taken in comparison with other studies that used 12–14 cores. As a result, the majority of AS protocols recommend that the number of samples taken in a repeat biopsy should be in proportion to prostate size.

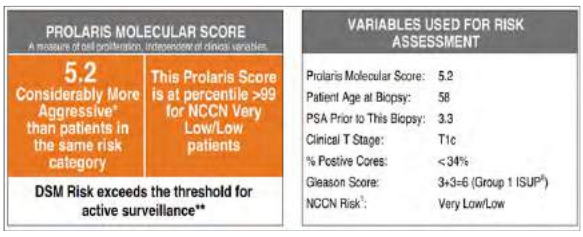
Role OF MRI

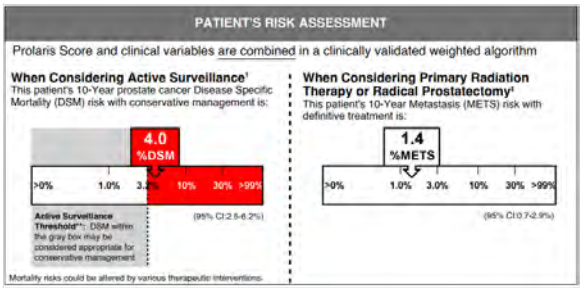
MRI of the prostate is increasingly used because of its potential as a non-invasive technique. As a diagnostic tool, 3 Tesla multiparametric MRI can identify abnormal lesions in the prostate that might be missed by transrectal ultrasound, especially in the anterior lobe[39]. Also, a positive preoperative MRI can predict higher upgrading rate compared with negative MRI (43% vs 27%). However, upstaging will remain even (10% vs 8%)[40]. Emerging techniques combining MRI and ultrasound to generate higher reclassification percentages than those obtained with standard biopsy confirmation (22–27%) are also becoming more common[41]. A negative MRI will have a reclassification

rate of 17% compared with a positive MRI with 39%[40]. To date, although MRI can be used to detect clinically significant disease in men undergoing AS, there is no evidence to support the replacement of repeated biopsies to detect progression. However, MRI-TRUS fusion biopsies might be important to AS protocols, as they target high-grade tumors and avoid detecting low-grade tumors. MRI-TRUS biopsies have proven to have twice the detection rate of random TRUS biopsies, and are able to detect 67% more Gleason ≥4 + 3 tumors while missing 36% of Gleason ≤3 + 4 tumors, thus reducing the detection of low-risk tumors[41].

Proteomic and genomic biomarkers

To improve the detection of potentially aggressive tumors, several biomarkers are being studied. Recently, two laboratory developed tests have been described to analyze prostate biopsy samples. Oncotype DX, from Genomic Health, measures the expression of 12 cancer-related genes representing four different biological pathways (androgen pathway, cellular organization, proliferation pathway and stromal response) and five reference genes[42]. They are combined to calculate a Genomic Prostate Score that is used to improve National Comprehensive Cancer Network risk criteria discrimination of PCa into very low, low and modified intermediate risk. Prolaris, from Myriad genetics, is a molecular test that measures cell growth characteristics to stratify disease progression by testing 46 different gene expressions. High expression of these genes is associated with higher risk of disease progression, thus closer monitoring or active treatment should be required for those patients[43].





Follow up for patients under AS

Patients who elect AS as a management strategy for their PCa need to be properly informed of the difference between Active surveillance (AS) and Watchful waiting (WW), and the benefits and pitfalls of AS. Additionally, patients need to understand the importance of compliance to a rigorous follow-up schedule. Follow up in most AS protocols is based on clinical data, including digital rectal examination, PSA results and repeat biopsies. Table-3[18,21,23,2437,44,45], lists some of the most commonly used follow-up schedules.

Protocol	DRE	PSA	Biopsy	Imaging techniques
Tosalan et at. (Johns Hopkins)[15]	6 months	6 months	Annual	MRI optional
Klotz et al. (University of Toronto)[16]	3 months (2 years) 6 months if PSA stable	3 months (2 years) 6 months if stable	Confirmation: 6-12 months Repetition: 2 years (to age 80 years) 1,4 and 7 years	
Bul et al. (PRIAS)[17]	3 months (2 years) 6 months (after)		If PSADT=3=10, repeat biopsy	
Dall Era et al. (UCSF)[18]	3 months	3 months	1-2 years (since 2003)	TRUS 6-12 months
Berglund et al. (MSKCC)[19]			Confirmation: 3 months Repetition: Annual	MRI prior to confirmation biopsy
Soloway et al. (Miami)[21]	3 months (2 yeas) 6 months if PSA stable	3 months (2 years) 6 months if stable	Confirmation: 9/12 months Repetition: annual	
Carter et al. (Johns Hopkins)[55]	6 months	6 months	Annual	

PSA

Although not very specific, PSA is a valid marker for both the diagnosis and monitoring of patients with PCa, including patients in AS. It was found, for example, that patients with PSA doubling time (PSADT) <3 years tended to have more aggressive Gleason scores at repeat biopsy, and generally had higher mean PSA levels[23]. However, post-diagnostic PSA kinetics do not reliably predict adverse pathology, and should not be used to replace annual surveillance biopsy for monitoring men on AS[46] (Table-4). PSA density (PSAD) proved to be a more specific marker for the diagnosis of PCa in patients with PSA <10, as well as a good predictor of adverse pathological

features and biochemical recurrence after RP. Its usefulness as a marker depends on finding a cut-off point that can be used to determine when to take action, but its sensitivity and specificity are too low to allow its use as a single marker. 57 Data from several AS protocols suggest that a PSAD <0.15 ng/mL/g indicates smaller and less aggressive tumors (Table-4)[18,21,24,44,48].

Protocol	Gleason	Positive cores	Percentage of core affected	PSADT
Tosolan et al (Johns Hopkins)[15]	>6	>2	>50	–
Klotz et al. (University of Totonto)[16]	4+3	--	--	<3
Dall'Era et al. (UCSF)[18]	Increase	--	--	--
Soloway et al. (Miami)[21]	>3 + 3	>2	--	--
Thomsen et al. (University of Copenhagen[63])	<4 + 3	>3	--	<3/5

Repeat biopsies

Repeat biopsies form the basis of follow up for patients in an AS program. However, there is no consensus on whether a repeat biopsy is necessary or when it should be carried out. Similarly, there is little uniformity in the intervals between repeat biopsies, though protocols using more restrictive criteria tend to suggest that they should be carried out annually, whereas others use wider intervals of up to 3 years (Table-3).

Other follow-up methods

In 2012, the European Society for Urogenital Radiology published a guide for MRI for PCa[49]. Some groups already include MRI in their protocols, either as standard procedure or when dealing with specific situations (Table-3). The literature shows mixed results regarding the utility of Prostate Cancer Antigen 3 (PCA3) molecule in AS protocols, although the majority of publications suggest that it can be useful in predicting tumor size and/or aggressiveness[50,51]. For example, a recent study showed that PCA3 <20 could identify indolent tumors that would be good candidates for AS[52].

NICE guideline follow up protocol and progression criteria[53]

Timing	Tests[a]
Year 1 of active surveilance	Every 3 to 4 months: measure prostate- specific antigen (PSA)[b] Throughout active surveillance: monitor PSA kinetics[c] At 12 months: digital rectal examination (DRE)[d] At 12 to 18 months: multiparametric MRI
Year 2 and every year thereafter until active surveillance end	Every 6 months: measure PSA[b] Throughout active surveillance: monitor PSA kinetics[c] Every 12 months:DRE[d]

[a] If there is concern about clinical or PSA changes at any time during active surveillance, reassess with multiparametric MRI and/or re-biopsy.

[b] Could be carried out in primary care if there are agreed shared-care protocols and recall systems.

[c] Could include PSA density and velocity.

[d] Should be performed by a healthcare professional with expertise and confidence in performing DRE. In a large UK trial that informed this protocol, DREs were carried out by a urologist or a nurse specialist.

If a person wishes to move from active surveillance to radical treatment at any stage in their care, make a shared decision to do so based on the person's preferences, comorbidities and life expectancy. Offer radical treatment to people with localised prostate cancer who had chosen an active surveillance regimen and who now have evidence of disease progression.

Conclusions

Active Surveillance appears to reduce overtreatment in patients with low-risk carcinoma of prostate without compromising cancer-specific survival at 10 years. Therefore, AS is an option for very low-, low- and selected cases of intermediate-risk of patients who want to avoid the side-effects including quality of life inherent to the different types of immediate treatment. However, inclusion criteria for AS and the most appropriate method of monitoring patients on AS have not yet been standardized. The heterogeneity in criteria of selection, deselection and follow-up protocols stems from the evolving knowledge about PCa, and the change in risk assessment tools.

References

1. Center MM, Jemal A, Lortet-Tieulent J et al. International variation in prostate cancer incidence and mortality rates. Eur. Urol. 2012; 61: 1079–92.
2. Mohler JL, Armstrong AJ, Bahnson RR, et al. Prostate cancer, Version 3.2012: featured updates to the NCCN guidelines. J Natl Compr Canc Netw 2012;10:1081–7.
3. Etzioni R, Gulati R, Falcon S, et al. Impact of PSA screening on the incidence of advanced stage prostate cancer in the United States: a surveillance modeling approach. Med Decis Making 2008a;28:323–31.
4. Lu-Yao G, Albertsen PC, Stanford JL, et al. Screening, treatment, and prostate cancer mortality in the Seattle area and Connecticut: fifteen-year follow-up. J Gen Intern Med 2008;23:1809–14.
5. Parker C. Active surveillance: towards a new paradigm in the management of early prostate cancer. Lancet Oncol 2004;5:101–6.
6. Andriole GL, Crawford ED, Grubb RL 3rd, et al. PLCO Project Team. Mortality results from a randomized prostate-cancer screening trial. N Engl J Med 2009;360:1310–9.
7. Schröder FH, Hugosson J, Roobol MJ, et al. ERSPC Investigators. Screening and prostate-cancer mortality in a randomized European study. N Engl J Med 2009;360:1320–8.
8. Etzioni R, Gulati R, Tsodikov A, et al. The prostate cancer conundrum revisited: treatment changes and prostate cancer mortality declines. Cancer 2012;118:5955–63.
9. Schröder FH, Hugosson J, Roobol MJ, et al. ERSPC Investigators. Prostatecancer mortality at 11 years of follow-up. N Engl J Med 2012b;366: 981–90.
10. Resnick MJ, Koyama T, Fan KH, et al. Long-term functional outcomes after treatment for localized prostate cancer. N Engl J Med 2013b;368: 436–45.
11. Mols F, Korfage IJ, Vingerhoets AJ, et al. Bowel, urinary, and sexual problems among long-term prostate cancer survivors: a population-based study. Int J Radiat Oncol Biol Phys 2009;73:30–8.
12. Johansson E, Steineck G, Holmberg L, et al; SPCG-4 Investigators. Longterm quality-of-life outcomes after radical prostatectomy or watchful waiting: the Scandinavian Prostate Cancer Group-4 randomised trial. Lancet Oncol 2011;12:891–9.
13. Cooperberg MR, Broering JM, Carroll PR. Time trends and local variation in primary treatment of localized prostate cancer. J Clin Oncol 2010a; 28:1117–23.
14. Heijnsdijk EA, der Kinderen A, Wever EM, et al. Overdetection, overtreatment and costs in prostate-specific antigen screening for prostate cancer. Br J Cancer 2009;101:1833–8.
15. Bill-Axelson A, Holmberg L, Ruutu M, et al; SPCG-4 Investigators. Radical prostatectomy versus watchful waiting in early prostate cancer. N Engl J Med 2011;364:1708–17.
16. Wilt TJ, Brawer MK, Jones KM, et al; Prostate Cancer Intervention versus Observation Trial (PIVOT) Study Group. Radical prostatectomy versus observation for localized prostate cancer. N Engl J Med 2012;367: 203–13.
17. Prostate Testing for Cancer and Treatment (ProtecT) Trial, https://www.baus.org.uk/_userfiles/pages/files/13-30%20BAUS%20ProtecT%20Nov%202016.pdf
18. Dall'Era MA, Albertsen PC, Bangma C, et al. Active surveillance for prostate cancer: a systematic review of the literature. Eur Urol 2012;62:976–83.
19. Eggener SE, Scardino PT, Walsh PC, et al. Predicting 15-year prostate cancer specific mortality after radical prostatectomy. J Urol 2011;185:869–75.

20. Epstein JI, Walsh PC, Carmichael M, Brendler CB. Pathologic and clinical findings to predict tumor extent of nonpalpable (stage T1c) prostate cancer. JAMA 1994; 271: 368–74.

21. Tosoian JJ, Trock BJ, Landis P, et al. Active surveillance program for prostate cancer: an update of the Johns Hopkins experience. J Clin Oncol 2011; 29:2185–90.

22. Porten SP, Whitson JM, Cowan JE, et al. Changes in prostate cancer grade on serial biopsy in men undergoing active surveillance. J Clin Oncol 2011;29:2795–800.

23. Bul M, Zhu X, Valdagni R, et al. Active surveillance for low-risk prostate cancer worldwide: the PRIAS study. Eur Urol 2013;63:597–603.

24. Klotz L. Active surveillance: the Canadian experience with an "inclusive approach". J Natl Cancer Inst Monogr 2012;2012(45):234–41.

25. Eggener SE, Scardino PT, Walsh PC, et al. Predicting 15-year prostate cancer specific mortality after radical prostatectomy. J Urol 2011;185:869–75.

26. Selvadurai ED, Singhera M, Thomas K, et al. Medium-term outcomes of active surveillance for localised prostate cancer. Eur Urol 2013;64:981–7.

27. Mottet N, Bastian PJ, Bellmunt J et al. Guidelines on prostate cancer. 2014. [Cited 18 Nov 2015.] Available from URL: http://uroweb.org/wp-content/uploads/1607-Prostate-Cancer_LRV3.pdf

28. Eichler K, Hempel S, Wilby J, Myers L, Bachmann LM, Kleijnen J. Diagnostic value of systematic biopsy methods in the investigation of prostate cancer: a systematic review. J. Urol. 2006; 175: 1605–12.

29. Villa L, Capitanio U, Briganti A et al. The number of cores taken in patients diagnosed with a single microfocus at initial biopsy is a major predictor of insignificant prostate cancer. J. Urol. 2013; 189: 854–9.

30. Ploussard G, Nicolaiew N, Marchand C et al. Prospective evaluation of an extended 21-core biopsy scheme as initial prostate cancer diagnostic strategy. Eur. Urol. 2014; 65: 154–61.

31. Tosoian JJ, JohnBull E, Trock BJ, et al. Pathological outcomes in men with low risk and very low risk prostate cancer: implications on the practice of active surveillance. J Urol 2013;190:1218–22.

32. Inoue LY, Trock BJ, Partin AW, et al. Modeling grade progression in an active surveillance study. Stat Med 2014;33:930–9.

33. Epstein JI, Feng Z, Trock BJ, et al. Upgrading and downgrading of prostate cancer from biopsy to radical prostatectomy: ncidence and predictive factors using the modified Gleason grading system and factoring in tertiary grades. Eur Urol 2012;61:1019–24.

34. Barzell WE, Melamed MR, Cathcart P, et al. Identifying candidates for active surveillance: an evaluation of the repeat biopsy strategy for men with favorable risk prostate cancer. J Urol 2012;188:762–7.

35. Miocinovic R, Jones JS, Pujara AC, Klein EA, Stephenson AJ. Acceptance and durability of surveillance as a management choice in men with screen detected, low-risk prostate cancer: improved outcomes with stringent enrollment criteria. Urology 2011; 77: 980–4.

36. Adamy A, Yee DS, Matsushita K et al. Role of prostate specific antigen and immediate confirmatory biopsy in predicting progression during active surveillance for low risk prostate cancer. J. Urol. 2011; 185: 477–82.

37. Berglund RK, Masterson TA, Vora KC, Eggener SE, Eastham JA, Guillonneau BD. Pathological upgrading and up staging with immediate repeat biopsy in patients eligible for active surveillance. J. Urol. 2008; 180: 1964–7; discussion 1967–8.

38. Motamedinia P, RiChard JL, McKiernan JM, DeCastro

GJ, Benson MC. Role of immediate confirmatory prostate biopsy to ensure accurate eligibility for active surveillance. Urology 2012; 80: 1070–4.

39. Hoeks CMA, Somford DM, van Oort IM et al. Value of 3-T multiparametric magnetic resonance imaging and magnetic resonance-guided biopsy for early risk restratification in active surveillance of low-risk prostate cancer: a prospective multicenter cohort study. Invest. Radiol. 2014; 49: 165–72.

40. Schoots IG, Petrides N, Giganti F et al. Magnetic resonance imaging in active surveillance of prostate cancer: a systematic review. Eur. Urol. 2015; 67: 627–36.

41. Siddiqui MM, Rais-Bahrami S, Truong H et al. Magnetic resonance imaging/ ultrasound-fusion biopsy significantly upgrades prostate cancer versus systematic 12-core transrectal ultrasound biopsy. Eur. Urol. 2013; 64: 713–9.

42. Knezevic D, Goddard AD, Natraj N et al. Analytical validation of the Oncotype DX prostate cancer assay – a clinical RT-PCR assay optimized for prostate needle biopsies. BMC Genom. 2013; 14: 690.

43. Sartori DA, Chan DW. Biomarkers in prostate cancer: what' s new? Curr. Opin. Oncol. 2014; 26: 259–64.

44. Soloway MS, Soloway CT, Eldefrawy A, Acosta K, Kava B, Manoharan M. Careful selection and close monitoring of low-risk prostate cancer patients on active surveillance minimizes the need for treatment. Eur. Urol. 2010; 58: 831–5.

45. Carter HB, Kettermann A, Warlick C et al. Expectant management of prostate cancer with curative intent: an update of the Johns Hopkins experience. J. Urol. 2007; 178: 2359–64; discussion 2364–5.

46. Ross AE, Loeb S, Landis P et al. Prostate-specific antigen kinetics during follow-up are an unreliable trigger for intervention in a prostate cancer surveillance program. J. Clin. Oncol. 2010; 28: 2810–6.

47. Corcoran NM, Casey RG, Hong MKH et al. The ability of prostate-specific antigen (PSA) density to predict an upgrade in Gleason score between initial prostate biopsy and prostatectomy diminishes with increasing tumour grade due to reduced PSA secretion per unit tumour volume. BJU Int. 2012; 110: 36–42.

48. Thomsen FB, Røder MA, Hvarness H, Iversen P, Brasso K. Active surveillance can reduce overtreatment in patients with low-risk prostate cancer. Dan. Med. J. 2013; 60: A4575.

49. Iu PP. ESUR prostate MR guidelines. Eur. Radiol. 2013; 23: 2320–1.

50. Tosoian JJ, Loeb S, Kettermann A et al. Accuracy of PCA3 measurement in predicting short-term biopsy progression in an active surveillance program. J. Urol. 2010; 183: 534–8.

51. Nakanishi H, Groskopf J, Fritsche HA et al. PCA3 molecular urine assay correlates with prostate cancer tumor volume: implication in selecting candidates for active surveillance. J. Urol. 2008; 179: 1804–9; discussion 1809–10.

52. Hirama H, Sugimoto M, Ito K, Shiraishi T, Kakehi Y. The impact of baseline [-2]proPSA-related indices on the prediction of pathological reclassification at 1 year during active surveillance for low-risk prostate cancer: the Japanese multicenter study cohort. J. Cancer Res. Clin. Oncol. 2014; 140: 257–63.

53. The National Institute for Health and Care Excellence (NICE) Guidelines for Prostate cancer; published on 9 May,2019. www.nic

Outcome of surgical repair of complete rupture of distal biceps tendon: a clinical series

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Abstract

Complete distal biceps rupture is a rare injury as compared to proximal biceps tendon rupture. It is usually caused by an eccentric contraction of the muscle, often seen with a sudden unintentional pull or jerk. An epidemiological study showed an incidence of 1.2 ruptures per 100,000 patients per year with an average age of 47 years. It is important not to miss the diagnosis initially, since delay in surgery does affect the outcome. In low-demand patients with complete distal biceps tendon tears, non-operative treatment may be entertained provided the patient understands the potential for residual weakness, particularly of forearm supination. The surgical repair is the treatment of choice especially in high demand male patients. There are a variety of fixation methods including bone anchors, suspension techniques, bone tunnels with interference screws, and transosseus sutures. No one technique has emerged as the gold standard and the choice remains that of surgeon. In this case series we present couple of patients with distal biceps tendon rupture one with acute rupture and another with chronic rupture treated surgically. We conclude patients do benefit from surgical repair

Keywords: Distal biceps tendon, Acute, Chronic, Tendon tear, Elbow, Repair

Introduction

Nearly 90% of biceps tendon ruptures occur in the proximal biceps and involve the long head of biceps. The remaining ruptures occur in the distal biceps tendon representing only 3% of biceps muscle injuries. The mechanism of injury in distal rupture involves a strong eccentric contraction of the biceps tendon against unanticipated resistance. The injury is most common in the dominant arm of middle-aged men. Symptoms include weakness in elbow flexion (by 30%) and forearm supination (by 40%).[1]

Some individuals may maintain reasonable function after non-operative treatment of a ruptured distal biceps tendon, biomechanical and clinical studies suggest that most individuals benefit from surgical repair or reconstruction.[2]

The surgical treatment of distal biceps tendon tears has been studied extensively. The techniques available for repair involve a three-level distinction: anatomic *versus* non-anatomic repair, single-incision *versus* double-incision exposure and fixation method (most commonly the use of cortical button, interference screws, transosseous sutures or suture anchors).[2,3]

The number of reported distal biceps tendon tears seems to have increased over the last few years, likely related to better understanding and improved diagnostic methods.[4] However in a number of instances, the diagnosis is initially missed. In this case series we present couple of patients with complete distal biceps tendon rupture; one with acute rupture and another with chronic rupture. We discuss the diagnosis, treatment, outcome and complications of surgical repair of the tendon using suture anchor.

Clinical series

Case 1

63 year old left dominant handed male patient sustained injury to left elbow at work site one month before he presented to us,- while he was trying to cut a banana tree

it was about to fall on his feet, while he tried to catch the tree before it fell. He had a painful 'pop' at the time of injury. He noticed painful range of movement of left elbow and difficulty in using left elbow. Initially he consulted an orthopaedic surgeon and was managed with a sling and analgesics, when he presented to us he had developed painful restriction of range of movement and was so unhappy about the initial treatment. On inspection there was flattening of the distal contour of the arm as compared to opposite arm (fig. 1), mild tenderness at antecubital fossa. He had weakness of flexion and supination., On 'hook test'(fig. 2) we could not hook finger around any anterior structures with elbow in flexed and supinated position. Pre operative quick DASH score was 57. X-ray showed no bony abnormality and an MRI confirmed complete rupture (fig. 3) and marked retraction of distal biceps tendon from bicipital tuberosity of the radius. Since the duration of injury was just one month we planned for reinsertion of tendon to bicipital tuberosity using fibre wire and an anchor screw. After pre anaesthesia evaluation patient was taken up for surgery. We went through anterior approach using a curved single incision over antecubital

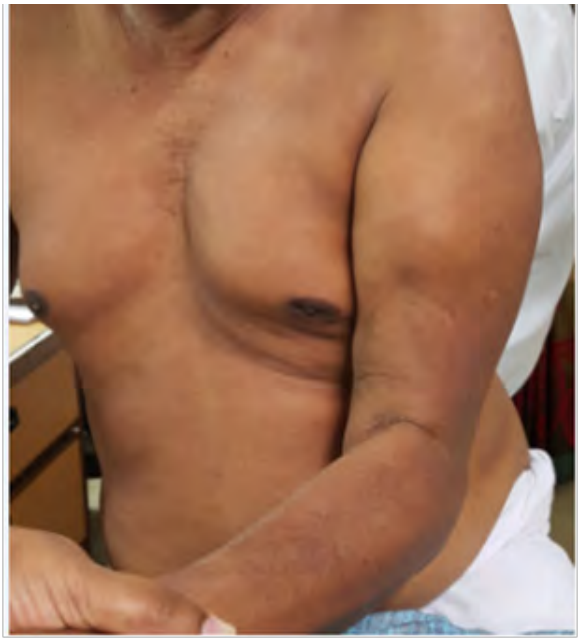

Fig. 1: At presentation note the flattening of the distal contour of the arm

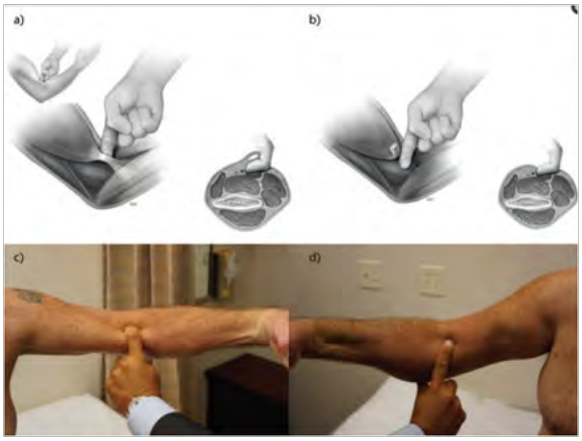

Fig. 2: The hook test for distal biceps tendon, as described by O'Driscoll.6 a) The normal test in which the examiner's fingers can be hooked under the biceps tendon. b) The abnormal test, in which the examiner is unable to hook the distal biceps tendon. c) Demonstration of a normal hook test. As shown, a cord-like structure is felt under the index finger. d) Clinical picture demonstrating an abnormal hook test. The examiner is unable to feel the cord-like structure corresponding to the distal biceps tendon of the arm.

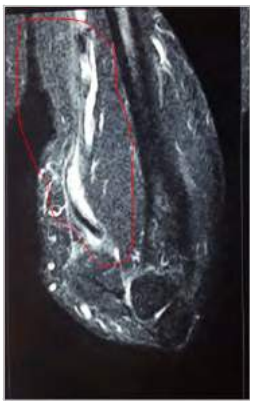

Fig. 3: MRI showing complete rupture and marked retraction of distal beceps tendon from bicipital tuberosity of radius

fossa without a tourniquet, and retrieved the tendon from the superior part of the incision (Fig. 4). On the distal part of the incision further dissection was done and

Fig. 4: Retrieval of the ruptured tendon from the proximal part of incision

recurrent branch of radial artery was ligated (fig. 5) to avoid injury to it. Bicepital tuberosity on radius was identified and bed prepared (fig. 6) An anchor screw was passed into it and with the fibre wire, tendon was re attached(fig. 7), as we expected there was enormous tension on tendon and hence a long arm slab in 110^0 of flexion and supination was given. Patient was discharged on second post op day and at one month follow up slab and sutures were removed, elbow mobilisation started (fig. 8).

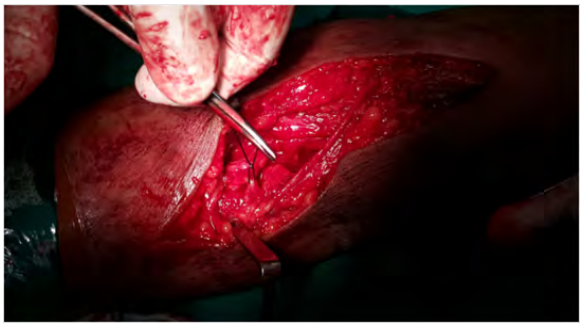

Fig. 5: Ligation of recurrent branch of radial artery

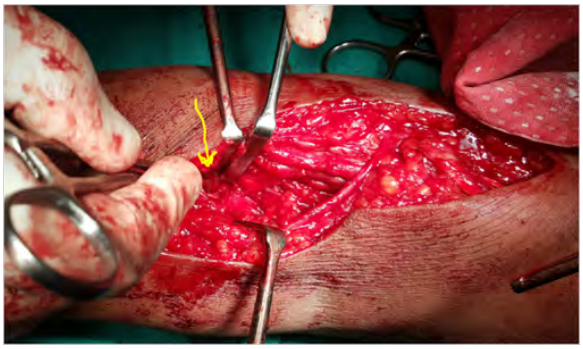

Fig. 6: Bicepital tuberosity on radius

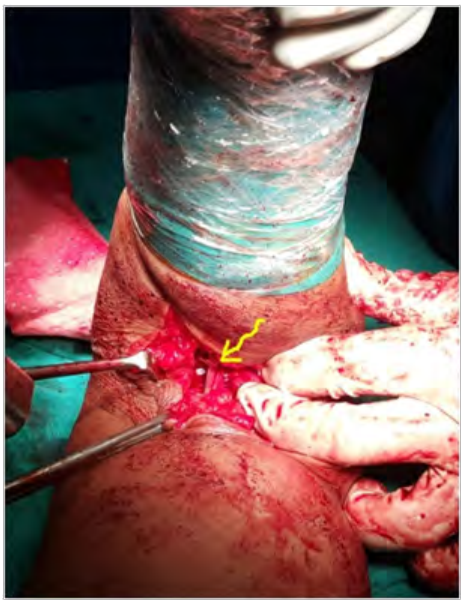

Fig. 7: Reattachment of tendon using suture anchor and fibre wire

Fig. 8: At 1 month follow up POP slab was removed and sutures removed wound healed well

Wound was healed well and he had near normal range of movement and without any pain and quick DASH score improved to 11 at 2 months post op Figure 9(a) and 9(b) and it is improving further.

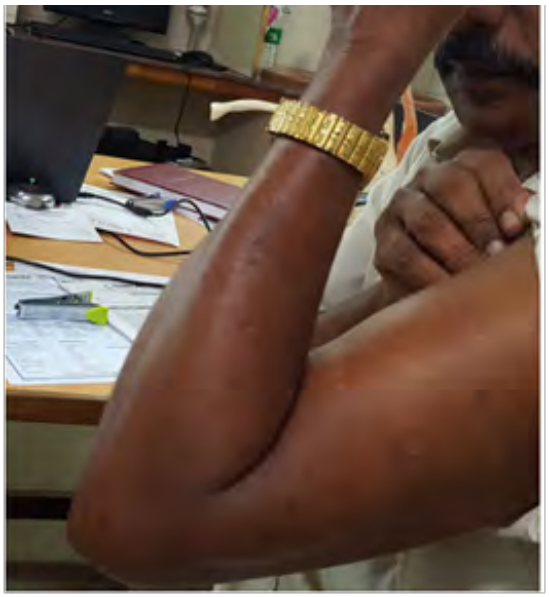

Fig. 9(a): At 2 months follow up

Fig. 9(b): At 2 months follow up

Case 2

A 44 year old right hand dominant male farmer patient sustained distal bicepital tendon rupture following a fall from tree while he was trying to cut a branch of tree and he lost control and fell down and tried to catch another branch of the tree to save himself and he heard a painful pop at that time and finally he fell on the ground. Fortunately he did not have any major fractures but noticed pain and swelling around the elbow. On the same day he presented to us. On examination swelling and ecchymosis in the distal arm and proximal forearm. Severe tenderness was noted. X ray of right elbow was normal and an MRI showed complete tear of distal biceps tendon. He was treated similarly as described in case one and biceps was reattached using fibre wire and suture anchor. Post operative protocol was also similar as case one and at 3 months follow up his quick DASH score improved to zero and he returned back to work.

Discussion

Distal biceps rupture is a rare injury as compared to proximal biceps tendon rupture usually caused by an eccentric contraction of the muscle, often seen with a sudden unintentional pull or jerk. An epidemiological study showed an incidence of 1.2 ruptures per 100,000 patients per year with an average age of 47 years.[5] Unlike proximal biceps tendon rupture which leave only a cosmetic deformity (with little or no functional disability), the distal biceps tendon injury affects functional outcome of elbow resulting in stiffness, chronic pain, weakness of flexion and supination. Unfortunately, the implications of this injury is high, in demanding male labourers. In a number of instances, the diagnosis is initially missed. In such situations patients are extremely unhappy. As seen in our patient (case one) his diagnosis was missed initially and when he presented to us at one month post injury he was very unhappy about initial treatment and outcome.

The diagnosis of complete distal biceps tendon tears can be established based on patient history and physical examination. X ray elbow may be normal, Ultrasonogram (USG) and magnetic resonance imaging (MRI) provide more valuable information. Patients may report a painful 'pop' at the time of injury. A useful clinical test was described by O'Driscoll et al, the so-called 'hook test'.[6,7] The patient is asked to look at the palm of his hand on the affected side with the shoulder elevated, the elbow flexed at 90° and the forearm in supination. An intact distal biceps tendon allows the examiner to hook his fingers around the cord-like structure. If the bicep is torn, since the distal brachialis is flat, the examiner will not be able to hook his finger around any anterior structures. This test was elicited in patient with chronic distal biceps tendon rupture (case 1) We did not try this test in other patient with acute tendon rupture because it was painful.

Nonoperative treatment in symptomatic patients has been shown to result in a 30–50% loss in supination strength and 20% loss in elbow flexion strength.[8] Thus the surgical repair is the treatment of choice especially in high demand male patients. So, the consensus of opinion is that acute ruptures should be repaired primarily if possible, and there are a variety of fixation methods including bone anchors, suspension techniques, bone tunnels with interference screws, and transosseus sutures.[9] No one technique has emerged as the gold standard, and the choice remains that of surgeon.

The techniques available for repair involve a three-level distinction: anatomic *versus* non-anatomic repair, single-incision *versus* double-incision exposure and various fixation methods. Randomised controlled trial was conducted to compare acute DBT tears treated surgically with a single-incision technique (fixation with two suture anchors) or double-incision technique (fixation with

transosseous bone tunnels).[10] The authors found that both techniques provided similar results in terms of pain, American Shoulder and Elbow Surgeons (ASES) elbow scores, and functional sub-scores, DASH score, patient-rated elbow evaluation (PREE) score, and isometric extension, pronation or supination strength. However, the double-incision technique resulted in significantly higher strength for elbow flexion when compared to the single-incision technique (104% *vs* 94%, respectively). We have used single incision technique in both cases and used anchor screw for fixation. In acute cases this approach gives excellent exposure for retrieval, preparation and fixation at radial tuberosity. In chronic cases if there is associated *lacertus fibrosus* tear, tendon retracts so much and generate lot of tension when it is brought down and hence it requires immobilisation in flexion and supination.. In situation where severe retraction of tendon requires reconstruction this is best done with autograft. Outcome of our patients is excellent. Quick DASH score improved from 57 to 11 at 2 months post op and it is improving further in patient with chronic rupture and quick DASH score improved to 0 at 3 months post-op in patient with acute rupture.

Injuries to posterior interosseous nerve and the lateral antebrachial cutaneous (LABC) nerve have been reported in 5% to 40% of elbows respectively, more commonly with a single anterior incision.[10,11] Heterotopic ossification may be seen on radiographs after distal biceps tendon repair using any exposure or fixation technique, but it seems to be more common and tends to interfere more with forearm rotation using a two-incision technique. Care should be taken not to expose the ulna to prevent cross union.[3] We don't have such complication in either cases.

Conclusion

Distal biceps tendon rupture is a relatively rare injury usually caused by an eccentric contraction of the muscle in middle aged men. It is common on the dominant side. It is important not to miss the diagnosis initially, since delay in surgery does affect the outcome. In low-demand individuals acute complete tears are occasionally treated non-operatively, but most patients benefit from surgical repair.

References

1. Loannis Sarris, Dean G Sotereanos, Distal biceps tendon ruptures; Journal of the American Society for Surgery of the Hand, Volume 2 , Issue 3 , 121 – 128, 2002
2. Miyamoto RG, Elser F, Millett PJ; Distal biceps tendon injuries. J Bone Joint Surg Am. 2010 Sep 1; 92(11):2128-38.
3. Sutton KM, Dodds SD, Ahmad CS, Sethi PM. Surgical treatment of distal biceps rupture. J Am Acad Orthop Surg 2010;18:139-148.
4. Database.Kelly MP, Perkinson SG, Ablove RH, Tueting JLAm Distal Biceps Tendon Ruptures: An Epidemiological Analysis Using a Large Population J Sports Med. 2015 Aug;3(8):2012-7.
5. Safran MR, Graham SM; Distal biceps tendon ruptures: incidence, demographics, and the effect of smoking. Clin Orthop Relat Res. 2002 Nov; (404):275-83.
6. O'Driscoll SW, Goncalves LB, Dietz P ;The hook test for distal biceps tendon avulsion.;Am J Sports Med. 2007 Nov; 35(11):1865-9.
7. Alentorn-Geli, Eduard, Andrew T. Assenmacher, and Joaquín Sánchez-Sotelo. "Distal Biceps Tendon Injuries: A Clinically Relevant Current Concepts Review." EFORT Open Reviews 1.9 (2016): 316–324. PMC. Web. 21 Oct. 2017.
8. Baker BE, Bierwagen D ;Rupture of the distal tendon of the biceps brachii. Operative versus non-operative treatment. J Bone Joint Surg Am. 1985 Mar; 67(3):414-7.9. Distal biceps tendon injuries. Miyamoto RG, Elser F, Millett PJJ Bone Joint Surg Am. 2010 Sep 1; 92(11):2128-38.

10. Grewal R, Athwal GS, MacDermid JC, et al. Single versus double-incision technique for the repair of acute distal biceps tendon ruptures: a randomized clinical trial. J Bone Joint Surg [Am] 2012;94-A:1166-1174

11. Morrey BF. Repair of distal biceps tendon rupture. In: Morrey BF, editor. , ed. Master's technique in orthopaedic surgery. The elbow. Philadelphia, PA: Wolters Kluwer; 2015:267-289.

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Effect of planned teaching programme regarding oral anticoagulation therapy and its therapeutic outcome among patients

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Abstract

Objective: To assess the relation between knowledge level and compliance in maintaining therapeutic INR.

Keywords: OAC – Oral anti-coagulation

OAT – Oral anti-coagulation therapy

OAK test – Oral anti-coagulation Knowledge test. The OAK test is a questionnaire consisting of 20 multiple choice questions and 5 open ended questions that assess the patients' knowledge on Oral anticoagulation therapy.

Introduction

Oral anticoagulants are highly effective for the treatment and prevention of thromboembolic disorders. However, anti-coagulation control and prevention of complication has been a long standing challenge. The level of knowledge of patients regarding oral anticoagulation plays an important role in maintenance of therapeutic INR and in prevention of drug related adverse events which are controversial[1]. Most studies conducted on this subject had small patient sample sizes and did not use the validated questionnaires to assess patients' knowledge on anti-coagulation therapy[2, 3].

Oral anti-coagulation therapy is the cornerstone in the prevention of morbidity and mortality due to venous thromboembolic events, such as the presence of venous thrombosis, stroke or cardiac arrhythmia. Oral anticoagulant therapy (OAT) comprises Vitamin K antagonist drugs, which act by increasing the blood clotting time of the individual (assessed by the INR - International Normalized Ratio). The vitamin K antagonists have a vast clinical experience in addition to the published evidence, which has proven its efficacy, and thus, they remain the mainstay of OAC therapy[4]. Monitoring OAC therapy is imperative in maintaining the appropriate levels of anticoagulation, balancing the risk of thrombosis and bleeding.

Patients who start using OAT remain hospitalized until the dose adjustment to their clinical condition, and, even after discharge, frequent tracking is needed to monitor the treatment. Numerous factors influence blood coagulation and may lead the patient to a higher risk of bleeding or thrombi[5,6].To minimize the risk of complications due to OAT use, specialized clinics in the management of such treatment have become common on the world stage. Specialized health professionals (physicians, pharmacists, nurses) develop actions for the realization of educational programs with verbal and written guidance, and the use of instructional videos; support groups, home visits, follow-up by telephone and quality indicators for the service provided[7]. Many studies highlighted the positive significant correlations between patients' adherence to OAC and their level of knowledge[8]. Studies suggest that patient education promotes better clinical outcomes, such as greater compliance[9,10], better INR control with values within the expected therapeutic range, better understanding of the signs and symptoms of complications[11], significant reduction in readmissions and decreased health costs.[12,13,14,15]

Methodology

Study design : Quantitative approach. Descriptive analytical design

Period of study : April 2017 – March 2018. (1 Year)

Population : Patients on regular anti- coagulant therapy

Sample : The patients who had started oral anticoagulation therapy from outside hospitals, now on regular follow up with KIMS OAC clinic

Sample size : 80

Data Collection Tools : Questionnaire to assess Knowledge regarding oral anticoagulation therapy

INR values from EMR

Inclusion Criteria : The patients, who had started oral anticoagulation therapy from outside hospitals, visited KIMS on OP or IP basis during the study period and consented for study.

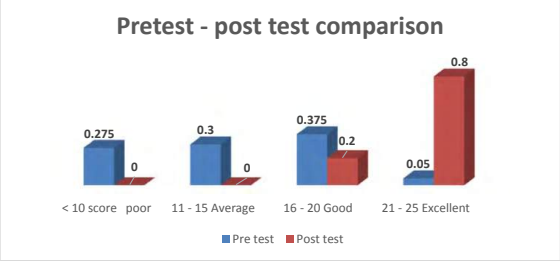
Exclusion Criteria : All patients who started OAC therapy and received education through OAC clinic.

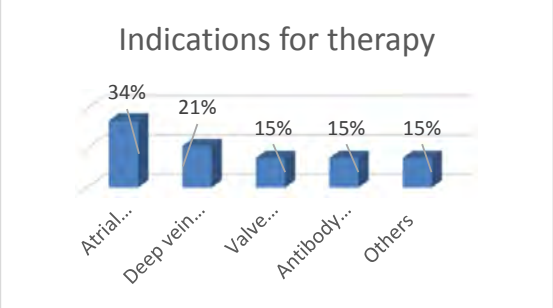
The study commenced in the month of April 2017. A standardized questionnaire (Zeolla et al.) to assess the knowledge on OAT was adopted (Annexure 1). All the patients who had started OAT from outside hospitals were being tracked during their visit to our institution as IP and OP. These patients were registered and followed up through the Oral Anticoagulation Clinic led by the OAC nurse. During visit, these patients were consented to participate in the study and were asked to complete the OAK test. After that a detailed education on OAT was given to these patients by the OAC Nurse. Then they were asked to undergo the OAK post- test during the next visit. The pre and post test scores were compared.

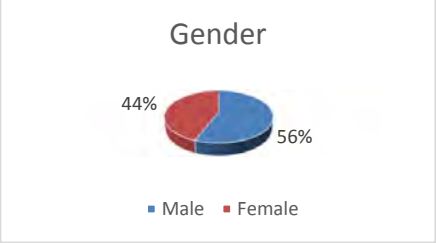
Correspondingly the therapeutic INR value prior to the training session and after the training sessions was compared.

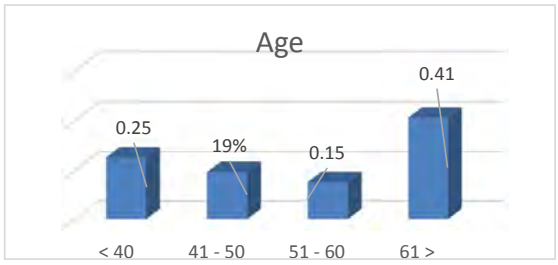
Results

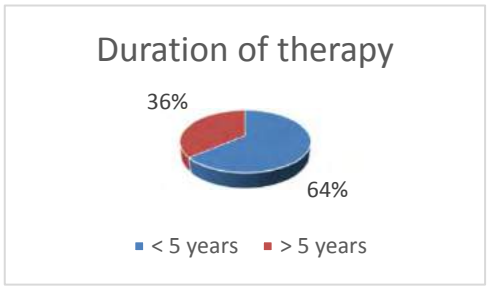
1. Among the 80 patients studied, 41% of the patients were above 60 years of age, 44% were male and 56% were female and 64% were on OAT for > 5 years.
2. 42.5 % were having a pretest score < 60% but in posttest none had a score less than 60%.
3. Only 5 % had excellent score > 80%, where as in posttest 80% had excellent score.
4. 68.75% were having INR value not in therapeutic range prior to training session whereas it has come down to 5% & 2.5% in the 1st and 2nd consecutive visit after training session.
5. Indication for OAT were Atrial fibrillation 34%, Deep vein thrombosis-21 %, valve replacement 15%, Antibody (Anti phospholipid , Lupus Anticoagulant) highly positive 15% and others (Embolectomy, pulmonary embolism, CVT, Ectacia coronaries) 15%.











Result and Discussion

1. There is a significant statistical association between pretest and posttest knowledge (P = 0.001*).
2. There is a significant statistical association between therapeutic INR value before and after planned teaching program (P = 0.001*)
3. There is a positive correlation (r = 0.142) between knowledge level and compliance in maintaining therapeutic INR value.

The reasons identified for noncompliance in maintenance of therapeutic INR even after training sessions were the following:

1. Attitude issues shown by alcoholic patients.
2. Patient and caretaker are old and forget to take medicine.
3. Financial constraints in regular follow up of INR values.

Areas for further study

1. The study can be done with a bigger sample, so that the result can be generalized more.
2. The effect of Knowledge and attitude of caretakers regarding the OAT on maintenance of therapeutic INR needs to be studied.
3. The effect of knowledge on prevention of complications needs to be studied.

Limitation of study

1. The study was limited to patients on traditional warfarin therapy and not to the patients on new generation OAC; hence the findings cannot be generalized.

Conclusion

The detailed education on OAT given to the patients by the OAC Nurse is found to be effective in the Maintenance of therapeutic INR. As knowledge on OAT increased, the compliance to therapeutic INR improved.

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We take this opportunity to extend our sincere gratitude and appreciation to all those who made this study possible.

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References

1. Omair S, Musallam N, Deghaither N, AlSadoun N , Bayoumy N. Compliance with and awareness about longterm oral anticoagulant therapy among Saudi patients in a University Hospital, Riyadh, Saudi Arabia. J. Applied Hematology2016 ;(7):1.
2. Zeolla MM, Brodeur MR, Dominelli A, Haines ST, Allie ND.Development and validation of an instrument to determine patient knowledge: the oral anticoagulation knowledge test. Ann Pharmacother.2006; 40(4):633-38.
3. Poupak Rahmani, Charlotte L. Guzman, Mark D Blostein, Ashley Tabah, Alla Muladzanov, and Susan R. Kahn Patients' Knowledge Of Anticoagulation and Its Association With Clinical Characteristics, INR Control and Warfarin-Related Adverse Events Article 'Blood' 2013 122:1738;
4. Efird LE, Chasler J, Alexander GC, McGuire M (Jun 21, 2016). "Prescribing Patterns of Novel Anticoagulants within a Statewide Multispecialty Practice". American Journal of Pharmacy Benefits. 8 (3): 97 – 102.
5. Vrijens B, de Geest S, Hughes DA, et al., "A new taxonomy for describing and defining adherence to medications," British Journal of Clinical Pharmacology2012; 73(5): 691–705.
6. Kumari MJ, Amirthavalli A, Dhananchezhian K, Jennifer D, Elakkia G, MathumalarN, Sangeetha M, Santhi G, Sreevastha D. Assessment of Knowledge on Oral Anticoagulation Therapy among Valve Replacement Patients. Inter J. Advanced Research 2015; 3(4): 1236-1243.
7. Emily M Hawes, University of North Carolina School of Medicine, Chapel Hill, NC 27599, USA. Patient Education on Oral Anticoagulation, Pharmacy (Basel). 2018 Jun; 6(2): 34.Published online 2018 Apr 20. doi: 10.3390/pharmacy6020034
8. IOSR Journal of Nursing and Health Science (IOSR-JNHS) e-ISSN: 2320–1959.p- ISSN: 2320–1940 Volume 6, Issue 3 Ver. II (May. - June. 2017), PP 19-29 www.iosrjournals.org

   DOI: 10.9790/1959-0603021929 www.iosrjournals.org 19 | Page Knowledge and Adherence to Oral Anticoagulant Therapy among Patients with Mechanical Heart Valve Prosthesis
9. Wang Y, Kong MC, Lee LH, Ng HJ, Ko Y. Knowledge, satisfaction, and concerns regarding warfarin therapy and their association with warfarin adherence and anticoagulation control. Thromb Res 2014; 133:5504.
10. Kim JH, Kim GS, Kim EJ, Park S, Chung N, Chu SH. Factors affecting medication adherence and anticoagulation control in Korean patients taking warfarin. J CardiovascNurs 2011; 26:46674.
11. Rocha H, Rabelo E, Aliti G, and de Souza E Knowledge of Patients with Mechanical Valve Prostheses Concerning Chronic Oral Anticoagulant Therapy. Rev. Latino-Am. Enfermagem 2010 Jul-Aug; 18(4): 696-702.
12. Holbrook A., Schulman S., Witt D.M., Vandvik P.O., Fish J., Kovacs M.J., Svensson P.J., Veenstra D.L., Crowther M., Guyatt G.H. Evidence-based management of anticoagulant therapy: Antithrombotictherapy and prevention of thrombosis, 9th ed: American College of Chest Physicians evidence-based clinical practice guidelines. Chest. 2012; 141(Suppl. 2):e152S–e184S. doi: 10.1378/chest.11-2295.[PMC free article] [PubMed] [Cross Ref]
13. Wittkowsky A.K. Impact of target-specific oral anticoagulants on transitions of care and outpatient care models. J. Thromb. Thrombolysis. 2013; 35:304–311. doi: 10.1007/s11239-013-0879-y. [PubMed] [Cross Ref]

14. Zdyb E.G., Courtney D.M., Malik S., Schmidt M.J., Lyden A.E. Impact of Discharge Anticoagulation Education by Emergency Department Pharmacists at a Tertiary Academic Medical Center. J. Emerg. Med. 2017; 53:896–903. doi: 10.1016/j.jemermed.2017.06.008. [PubMed] [Cross Ref]

15. Dharmarajan T.S., Gupta A., Baig M.A., Norkus E.P. Warfarin: Implementing its safe use in hospitalized patients from nursing homes and community through a performance improvement initiative. J. Am. Med. Dir. Assoc. 2011; 12:518–523. doi: 10.1016/j.jamda.2010.04.007. [PubMed] [Cross Ref]

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Perioperative nutrition intervention in post liver transplant - A case study

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Abstract

LT is an effective therapeutic option for irreversible ALD & ESLD for which available therapies have failed. The objective of MNT in acute post Liver Transplant phase is to provide adequate nutrition for replenishment of lost nutrients & to promote recipient recovery, it emphasizes the need for continuous, patient centric, aggressive nutrition support.

Key words: LT-Liver Transplant, ALD-Acute Liver Disease, HRS-Hepato Renal Syndrome, HCC-Hepato Cellular Carcinoma, MNT-Medical Nutrition Therapy, ONS-Oral Nutrition Supplement

Introduction

Liver Transplant(LT) is the only treatment for ESLD. Patients with cirrhosis should be considered for transplant evaluation when they have a decline in hepatic synthesis or excretory function, ascites, HE or complications such as HRS,HCC, recurrent SBP or variceal bleeding. Medical Nutrition Therapy is necessary during all phases of LT for improved surgical outcome.

Transplant process/treatment itself can be considered in 2 phases- pre transplant phase and posttransplant phase. The post transplant phase is again divided into acute phase(2-6 weeks post LT) & chronic phase (3 months post LT).

Its estimated that malnutrition occurs in 65-100% of patients with ESLD. Reasons for malnutrition in patients with ESLD is multifactorial. Major determinants are decreased nutrient and calorie intake, intestinal malabsorption& overloaded catabolism. The presence of impaired absorption function due to portal hypertension (PHT). Loss of protein & trace elements is a common clinical phenomena in ESLD patients resulting from complications of diarrhea.

Pre Transplant phase

A 61 year old male diagnosed with DCLD-PHT under went DDLT, with a CHILD C score and MELD Na 23. Medical history showed the patient is known Diabetic for 20yrs, suffered from jaundice, ascites associated with pedal edema with recurrent episodes of HE & UTI.

Assessments

Nutrition assessment & intervention are challenging pre operatively because of difficulties such as ascites, altered biochemical parameters, nausea, GI related problems and gustatory sensation. Perioperative assessment which include history, physical examination, lab tests & instrumental examination. Subjective Global Assessment showed a moderate malnutrition, NRS score of 2. There was a loss of subcutaneous fat mass & skeletal muscle mass due to protein catabolism. Up to 10 Lt of ascitic fluid tapping was done prior to surgery.

Nutrition assessment of the patient

Parameters	Observation
Anthropometric Evaluation	Pre OP
Weight(Kg)	89
Height(cm)	169
Ideal Body Weight(Kg)	69
Dry Weight(Kg)	81
SGA	19
NRS	2

Biochemical Examination

Parameters	Pre LT	Post LT
Hb	10	9.2
Platelets	97	202
Alb	3.2	3.5
BT	2.8	0.5
BD	1.3	0.3
AST	25	10
ALT	38	11
Na	126	136
K	4.3	4.6
Cr	0.7	0.6

Nutrition Intervention Pre LT

The objective of Medical Nutrition Therapy is to educate & provide the requirements according to the present disease condition. The patient was advised to follow 25-30Kcal/kg bodyweight ,with moderate carbohydrate and protein 1.2-1.5gm/kg body weight with salt restricted to 2gm/day with a fluid restriction of 1.25Lt/day. In order to meet the high caloric demand a hepatic friendly Oral Nutrition Supplement(ONS) was advised to take as TDS.

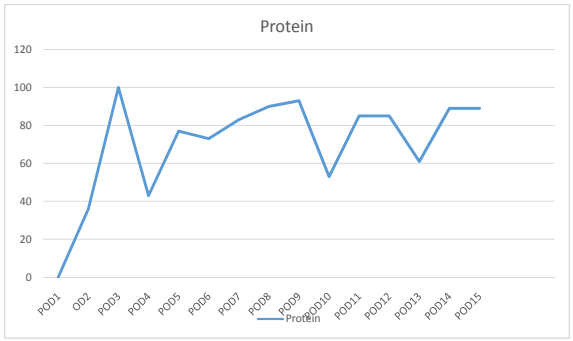
Nutrition Intervention Post LT

The post op period could be more conductive to nutritional support as the patient was under continuous Medical & Nutritional monitoring. Acute post LT phase is characterized by catabolism due to surgery & corticosteroid administration which are anti-inflammatory & immunosuppressive drugs. Timely & adequate nutrition may replenish lost nutrients & to promote patient recovery in a faster rate.

After LT, the patients were recommended disease specific diets through various routs of feeding so that patients could maintain intake according to the recommended requirements. For this patient we initiated on NJ feed (POD1) within 24hr after LT, initially it was started as 50ml/hr - clear feeds along with oral sips of water. As the patient tolerated the feed it was switched to normal feeds and the feed volume was increased to 75ml/hr . Meanwhile the patient's oral intake improved , on POD4 the patient was off NJ feeds & started taking full oral diet. The patient requirements were 2100Kcal & 103gm protein. He was started with a high protein Oral Nutritional Supplement(ONS) in a complete neutropenic manner.

It is quite challenging to meet the nutrient requirements in acute post LT phase because of excessive nausea after surgery stress, pain, lack of appetite or slow GI functioning, metabolic complications, regular tests requiring NPO's which would impact on the daily food intake, hospital foods, functional inability etc would make it difficult for the patient to consume adequate quantities of food.

Information regarding dietary intake was computed from 24hrs diet recall.

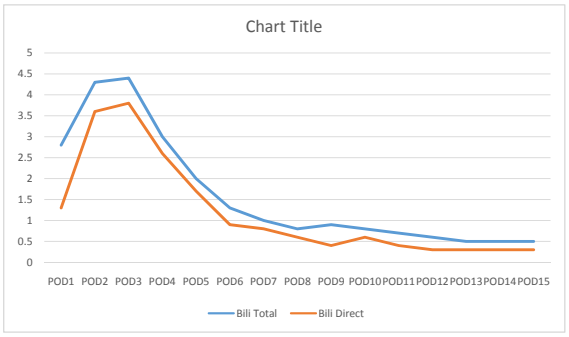


Graph representing the daily calorie intake which was met through NJ & Oral Feeds

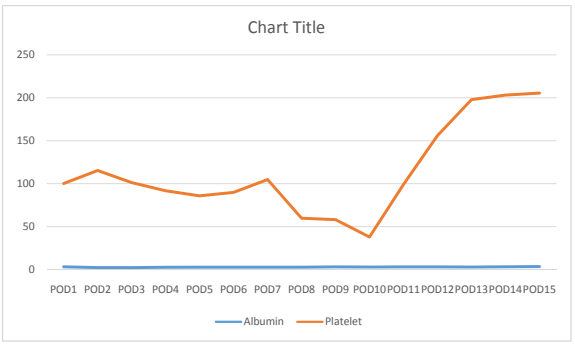
Graph representing the daily protein intake

For this patient he met 105% of his daily calorie & 97% of daily protein requirement when he was on NJ feed. As the patient started taking orally 78% of his daily calorie & 87% of his daily protein requirements were met.

The patient was advised to include foods from all the food groups, in order to provide wide variety of food choice



& to receive all the macro & micro nutrients, there by promoting a healthy gut micro biota.



Graph representing Changes in Total & Direct Bilirubin during post LT

Graph representing the daily albumin & platelet count

During the post LT period the patient received couple of blood transfusion, 20% albumin infusion was given on daily basis.

Conclusion

Continuous observation by medical and nutrition experts helped to fullfill nutritional needs through various feeding routes. A gradual improvement in nutrition, biochemical, and functional parameters were able to observe from this study. Each patient is specific and each diet formulated are specific in order to achieve their present demands. A cautious patient centric approach with continuous monitoring helps in better post LT outcome. A gradual improvement in nutrition, biochemical and functional parameters are seen after LT.

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References

- Plauth M, Cabre E, Riggio O, Assis-camilo M, Pirlich M, Kondrup J et al. ESPEN guidelines on enteral nutrition: Liver Disease. Clin Nutrition 2006
- Bakshi N, Singh K, Nutrition Assessment in patients undergoing Liver transplant. Indian J Crit Care Med 2014
- Bakshi N, Singh K. Effect of malnutrition on health related quality of life of patients awaiting liver transplantation. Indian J Transplant 2015
- Bakshi N, Singh K, Soin AS. Effect of pre-transplant malnutrition on outcomes of liver transplant. J Clinic ExpHepatol 2016

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MIRA: A new vista in skull base surgery

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Neurovascular compression syndrome (NVCS) is a disease caused by presence of contact between a vascular loop in the cerebellopontine angle (CPA) and one of the cranial nerves. Normally, the CPA is characterized by presence of many vascular and neural structures, which are normally in contact with each other without causing a problem; however, sometimes this contact causes a problem to the patient and becomes symptomatic leading to the so-called NVCS, the symptom of which depends on the compressed cranial nerve and the compressing vessel is called an off ending vessel. Vascular compression syndrome of the cranial nerves, first suggested in 1934 by Dandy and popularized by Jannetta in the 1970s , are gaining acceptance with the improvement in MRI assessment and the success of endoscope-assisted microvascular decompression (MVD) procedures; they are commonly described in trigeminal neuralgia and hemifacial spasm. Minimally invasive retrosigmoid approach (MIRA) was first described by Jacques Magnan in1974. Unlike the conventional approach the morbidity of the procedure is very low and using endoscopes will give a panoramic view of the complex anatomy of CPA.

Case

A 39-year-old female, tailor by occupation presented with an embarrassing symptom of left eye winking and twitching of left side of face and lips involuntarily. She also had

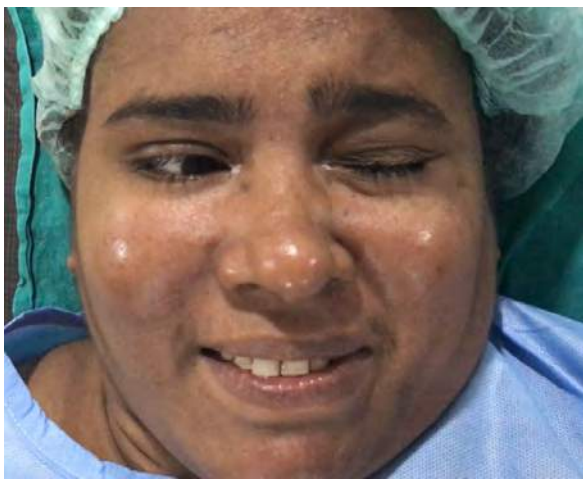

Fig.1: Pre op

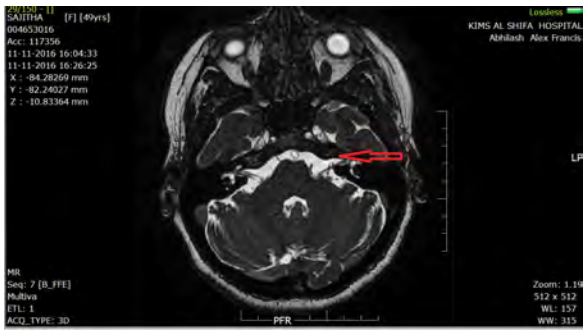

Fig. 2: MRI

severe facial pain during attacks and had taken multiple medicines with no effect. Whenever she stoops downs for some time for her work she experiences excruciating headache which made her quit her job. Her MRI showed a vascular loop compressing the left facial nerve as the culprit of her symptoms.She was diagnosed to have Left

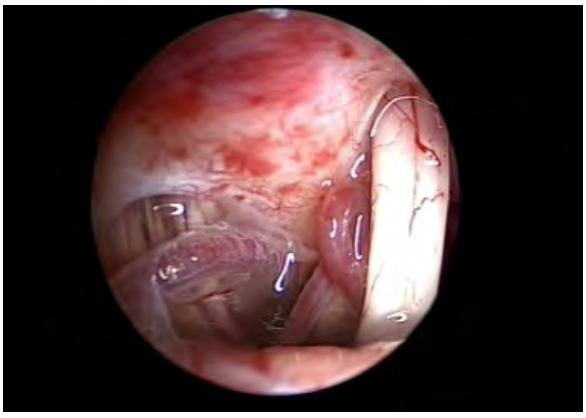

Fig. 3: Vascular loop over the Facial N

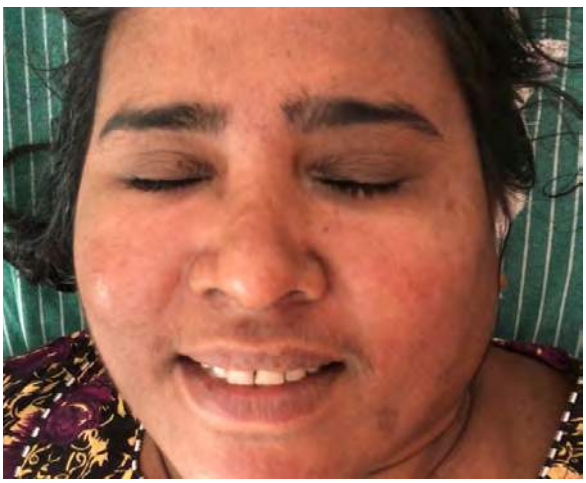

Fig.4: Post op

Disclosure: Patient photos published with their permission

hemifacial spasm and a decision to surgically decompress the vascular insult was taken.

We did endoscopic MIRA approach for left hemifacial spasm; a keyhole procedure in the skull and under endoscopic assistance the culprit blood vessel was diverted from the facial nerve at its entry point to brain by separating it by micromanipulation and keeping a Teflon felt between the vessel and the facial nerve. She had only one day of ICU stay and left the hospital the very next day alleviated of this misery and has been symptom free for the last 5 years.

Trigeminal neuralgia, hemifacial spasm, intractable vertigo, tinnitus, and glossopharyngeal neuralgia are disabling functional cranial nerve disorders that can impair severely the quality of the life of patients. The efficacy of conservative management usually is limited over time, and patients ultimately seek a definitive surgical solution to their problem. Although MVD is gratifying for the patient and the surgeon, it is the surgeon's duty to prevent any devastating complications in these functional disorders. In our neuro-otological experience using MIRA surgeries for indications such as vascular compression syndromes, we have very little morbidity; this should be the rule in all functional surgical indications. The most common surgical causes of incomplete cure are misjudgment of the real off ending vessel and incomplete or improper replacement of the conflict vessel. The use of endoscope intraoperatively enhances the chances of visualization of the offending vessels without increasing the amount of retraction of the cerebellum, and thus reducing the chances of complications such as hearing loss and facial weakness.

Thus,MIRA has revolutionized the management of vascular compression disorders and has provided a smiling face to many with less morbidity.

Appreciations

Dr. Rajeev C, Resident, Dept. of General Surgery, secured first prize in the Quiz competition held at the conference of the Association of Surgical Gastroenterologists of Kerala, on 11th and 12th Jan 2020 in Palakkad.

Dr. Prabhnoor Singh & Dr. Anand Vakade, Residents, Dept. of Urology secured second prize in USI URO Quiz competition held at the Annual Conference of the Urological Society of India from 23rd to 25th Jan 2020 in Kochi.

Dr. CC Kartha, Sr. Advisor – SOCOMER received Lifetime Achievement Award in Cardiovascular Sciences, Medicine and Surgery from the International Academy of Cardiovascular Sciences on 21st Feb 2020 in New Delhi.

Dr. GV Sujith Kumar Reddy, Resident, Dept. of Neonatology, secured first prize in the All India Quiz at NORM 2020 held in Delhi.

Scientific Publications

Sl.No.	Name of the Author/s	Title	Name of the Journal
1	Van Vollenhoven RF, Navarra SV, Levy RA, **Mathew Thomas (Internal Medicine, KIMS)**, Heath A, Lustine T, Adamkovic A, Fettiplace J, Wang M-L, Ji B, Roth D.	Long-term safety and limited organ damage in patients with systemic lupus erythematosus treated with belimumab: a Phase III study extension.	Rheumatology (Oxford). 2019 Jul 13. Pii: kez279. Doi: 10.1093/rheumatology/kez279 *(International)*
2	Kumar AA, Satheesh G, Vijayakumar G, Chandran M, Prabhu PR, Simon L, Kutty VR, **Kartha CC(SOCOMER),** Jaleel A.	Postprandial Metabolism is Impaired in Overweight Normoglycemic Young Adults without Family History of Diabetes.	Scientific Reports.2020;10:353. https://doi.org/10.1038/s41598-019-57257-2. *(International)*
3	Kartha CC (SOCOMER)	Annual Review of Medicine, 2019	Current Science. 2020; 118: 2 *(National)*
4	Praveen Murlidharan (Nephrology), Sreelakshmi K (Clinical Research), Satish Balan (Nephrology), Kartha CC(SOCOMER).	Mechanisms for obesity related kidney disease.	Chapter in the book entitled "Pathophysiology and obesity induced health complications, Advances in biochemistry in health and disease 19". Springer International. 2020.19;193-216.
5	Anandkumar A (AIMS, Cochin), Kartha CC (SOCOMER)	Is ambient space a determinant of human health?	Current Science. 2020; 118(4):511-212. (Guest Editorial).

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