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Original Article

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Comparison of D- Dimer & FDP (Fibrin Degradation Product) with APACHE II as marker of Severity of Acute Pancreatitis

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Abstract

Acute Pancreatitis is an acute inflammatory process of the pancreas with varying involvement of regional tissues or remote organ systems. Complex clinical criteria of Severe Acute Pancreatitis include Ranson's criteria for non-gall stone pancreatitis. The Apache II & multiple organ system failure scale provides prognosis at the time of admission. D Dimer Is a fragment produced during the degradation of clot. FDP is Fibrin Degradation Product in blood which results from dissolution of clot. The Study was conducted at Chugh Multispeciality Hospital, Bhiwani in 103 patients fulfilling the inclusion and exclusion criteria for a period of 1 Year. The Result Concludes APACHE II FDP & D – Dimer values significantly correlate with each other in almost all aspects.

Keywords: APACHE–II; D-Dimer; FDP; Pancreatitis; MODS.

Introduction

Acute pancreatitis is an acute inflammatory process of the pancreas with varying involvement of regional tissues or remote organ systems. The incidence of acute pancreatitis in England, Denmark and USA ranges between 5 & 30 per 100,000 population with highest incidence recorded in the United States and Finland.

The estimated incidences are however in accurate because the diagnosis of mild diseases may be missed and death may occur before diagnosis in 10% of people with severe disease [1].

The increased incidence of pancreatitis, coupled with new treatment options, poses a challenge for primary care physicians. Twenty five percent of patient suffer from the severe form of the disease with local or systemic complications, resulting in mortality rate ranging from 2 -10%.

Increased mortality and morbidity are associated with organ failure in 50% of severe acute pancreatitis cases [2].

The two most common causes of acute pancreatitis are choleliathisis and alcohol. The other causes includes drugs (Azathioprine, Corticosteroids, etc.) [3].

Regardless of the etiology, pancreatic enzymes (Including Trypsin, Phospholipase A_2 & Elastase) become activated within the gland itself. The enzymes can damage tissue and activated complement and inflammatory cascade, producing cytokines. This process causes inflammation, edema, and sometimes necrosis. In mild pancreatitis, inflammation is confined to the pancreas; the mortality rate is 10 to 50%. After 5 to 7 days, necrotic pancreatic tissue may become infected by enteric bacteria. The chances of developing infected pancreatic necrosis depend the extent of necrosis [4].

Activated enzymes and cytokines that enter the peritoneal cavity cause a chemical burn and results in exudation of fluid in third space; those that enter the systemic circulation causes a systemic inflammatory response that can result in acute respiratory distress syndrome and renal failure. The systemic effects are mainly the result of

increased capillary permeability and decreased vascular tome, which result from the released cytokines and chemikines. Phospholipasae A_2 is thought to injure alveolar membranes of the lungs [4].

In about 40% of patients, collections of enzymes rich pancreatic fluid and tissue debris form in and around the pancreas and are called acute fluid collection. In about half, the collection resolves spontaneously. In others, the collections become infected or form pseudocysts. Pseudocysts may bleed rupture or become infected [5].

Death during the first several days is usually caused by cardiovascular instability (With refractory Shock & Renal Failure) or respiratory failure (With Hypoxemia and at times adult respiratory distress Syndrome).

Occasionally, death results from heart failure secondary to an unidentified myocardial depressant factor.

Death after the first week is usually caused by Multi organ system failure and / or infected necrosis and septic complication [6].

Pancreatitis is classified as acute unless there are CT/MRCP or ERCP findings of chronic pancreatitis. Mild acute pancreatitis consists of minimal or no organ dysfunction and an uneventful recovery. Severe pancreatitis manifests as organ failure and local complications such as necrosis, abscess and pseudocyst [7].

Complex clinical criteria of severe acute pancreatitis include Ranson's criteria for non gall stone pancreatitis and APACHE 2 (Acute Physiology and Chronic Health evaluation) score more than [8]. The APACHE 2 and the multiple organ system failure scales provides prognostic information at the time of admission and may be repeated daily to monitor disease progression.

It has been shown that coagulation abnormalities are common in acute pancreatitis which might be a part of the inflammatory process or related to early intravascular consumption of coagulation factors secondary to circulating pancreatic enzymes, particularly Trypsin or secondary to any vascular injury [9].

D – Dimer is a fragment produced during the degradation of Clot. The D Stands fort Domain indicates two identical units or domains. D- Dimer level is related to disease severity. FDP is a test measures Fibrin Degradation products in blood which result from dissolution of clots.

As a result of the coagulation process, fibrinogen is split in to Fibrin monomer and Fibrin. Fibrin monomer forms the fibrin polymer clot. By Measuring FDPs and idea about the activity of fibrinolytic system can be achieved [10].

A positive D – Dimer indicates the presence of an abnormally high level of fibrin degradation products in the body. There is sparse literature of D – Dimer and FDP as markers of severe pancreatitis [11].

Aims & Objectives

1. To study the profile of D - Dimer & FDP in acute pancreatitis.
2. To compare serum D Dimer and FDP levels with the APACHE II.

Review of Literature

Acute Pancreatitis is associated with increased concentration of serum fibrinogen, FDPs & D – Dimer, representing acquired dysfibrinoginemia, which is impaired fibrin polymerization. These coagulation abnormalities in patients with AP may be related to early intravascular consumption of coagulation factors secondary to circulating pancreatic enzymes like trypsin or secondary to vascular injury [10].

D Dimer may be expression of pancreatitis involving other organ systems. D- Dimer was found to be a prominent link in the chain of events leading to severe disease and statistically significant difference was found in the level of D – Dimer between uncomplicated and complicated pancreatitis [12].

Berry et al reported elevated levels of FDPs in 45% of patients with acute pancreatitis and suggested that the marked lung damage may be related to pulmonary fibrin deposition. Studies have shown reduced level of protein C, Anti thrombin III, D- Dimer and PAI – 1 in severe necrotizing pancreatitis indicating exhaustion of fibrinolysis and coagulation inhibitors in patients with poor outcome [13].

Material & Methods

This study was conducted at Chugh Multispecialty, Bhiwani for a period of one year.

Inclusion Criteria

All patients with a diagnosis of acute pancreatitis with or without complications were included.

Exclusion Criteria

1. Patient who presented after one week of onset of pain.
2. Underlying significant co morbidities including.
   - Decompensated Liver Disease.
   - Pre Existing Pulmonary infection or active pulmonary pathology.
   - Patients with inherent bleeding disorders or coagulation abnormalities.
   - Pregnant females.
   - Patients with active cardiac diseases e.g acute myocardial infarction, USA & Atrial fibrillation.
   - CKD – Chronic Kidney Disease.

Diagnostic Criteria

Acute Pancreatitis was diagnosed based on presence of characteristic pain and more than 3 times elevation of amylase /Lipase. Patients underwent a thorough history taking and clinical examination.

The following investigation were performed

- Complete Heamogram.
- Serum Amylase & Serum Lipase level.
- Liver Function Test.
- Renal Function Test.
- PT – Prothrombin Time.
- Urine Routine & microscopy.
- ABG – Arterial Blood Gases.
- USG – Abdomen.
- CECT – Abdomen if required.

D- Dimer And FDP Levels were done on 1, 3 & 7th Day of admission and then 7th day till the time of discharge or death.

Similarly APACHE Scores were calculated on the aforementioned days using the computer based APACHE II calculator. CECT was done on 3rd and 5th day in patients where indicated. Day of onset of organ failure from the onset of pain and number of organ involved were recorded.

Severity of pancreatitis was taken as per APACHE II scores of <8 as mild and >8 as severe. FDP <10 and D – Dimer <200 were taken normal.

Statistical Analysis

A database was generated in SPSS software. Correlation between D Dimer And FDP with APACHE II scores were expressed as person's correlation coefficient.

Comparison between the variables was done using mann- Whitney test, Chi square Test. A P-Value <0.05 was considred as statistically significant.

Results & Observations

One Hundred three (103) patients presenting with acute pancreatitis who fulfilled the inclusion criteria were included in the study.

Their details are as follows.

- Alcohol was the commonest etiology of the acute pancreatitis.
- In almost 25% of patients the etiology was not clear during their admission.
- Out of 103 patients taken together, 97 survived and 6 died with an approximate survival rate of 97.17%. Among the organ failure, respiratory failure was most common i.e. in 25 patients (25%), renal failure in 8 patients (8.08%), hypotension was present in 5 patients (5.2%). One patient had episode of gastro intestinal bleed.

Table 1: Organ Failure & Mortality

Organ Failure & Mortality	
Variables	**All patients**
Respiratory Failure	25 (25.0%)
Renal failure	8 (8.08%)
Bleed	1 (1.04 %)
Hypotension	5 (5. 21%)
Death	6 (5.38 %)

In this study we have tried to find any correlation with outcome of the patient i.e. whether the patient survives or dies with the severity of scores of APACHE and with the values of FDP and D- Dimer. Results shown above clearly depict that in the patient who died had higher APACHE scores, higher FDP values and higher D- Dimer values in all the 3 readings taken, which shows that they are correlated significantly with p value < 0.05 on all the occasions.

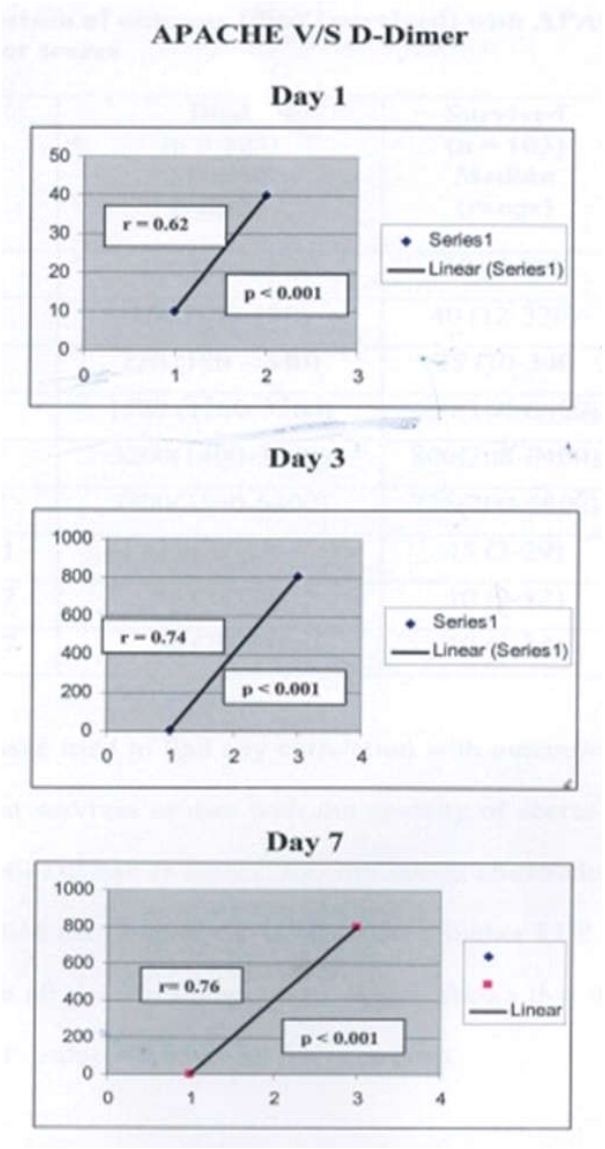

Fig. 1: APACHE V/S D- Dimer

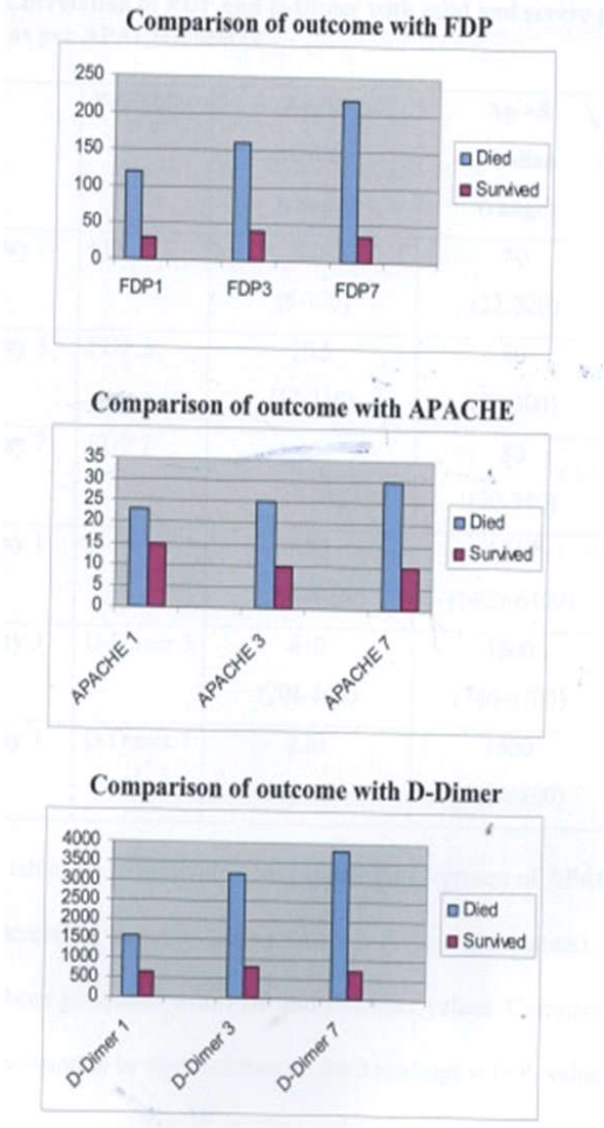

Fig. 2: Correlation of outcome with FDP, APACHE and D-Dimer

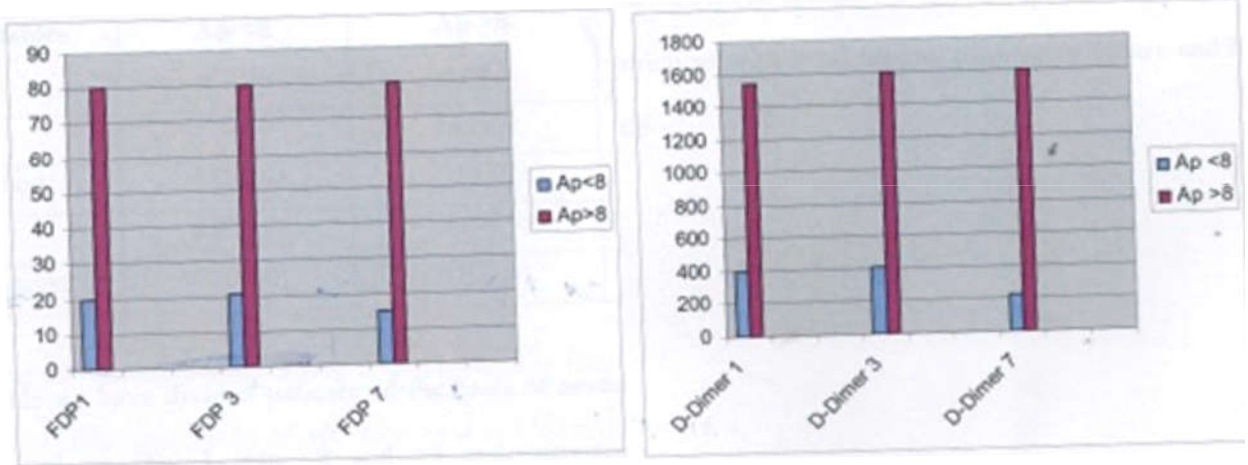

Fig. 3: Correlation of FDP and D-Dimer with mild and severe pancreatitis as per APACHE scores

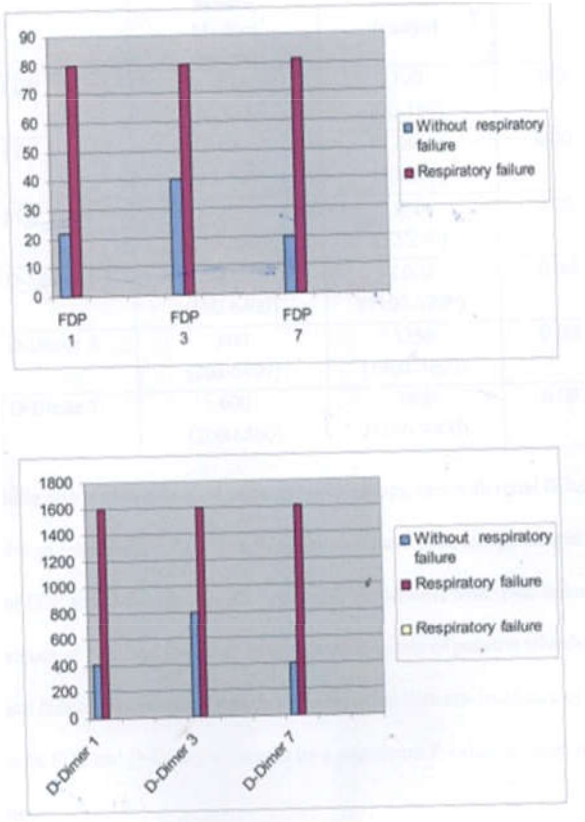

Fig. 4: Comparison of FDP and D-Dimer with respiratory failure

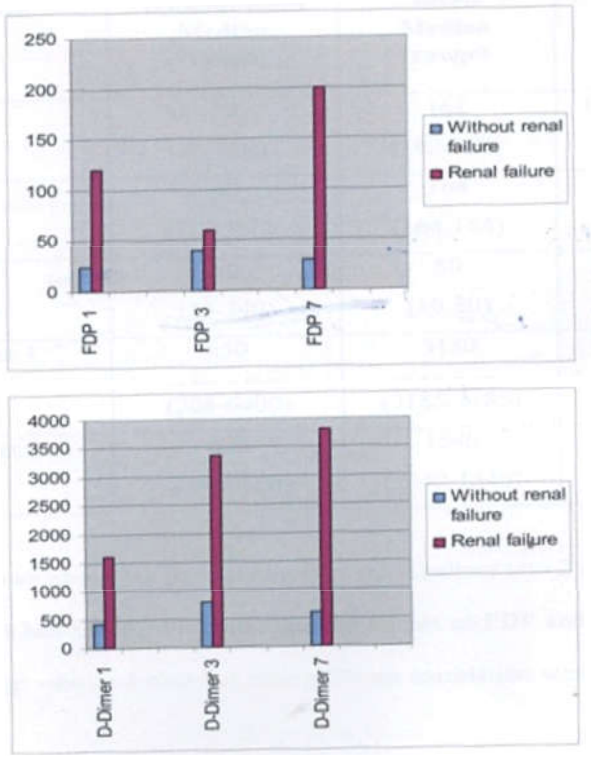

Fig. 5: Comparison of FDP and D-Dimer with renal failure

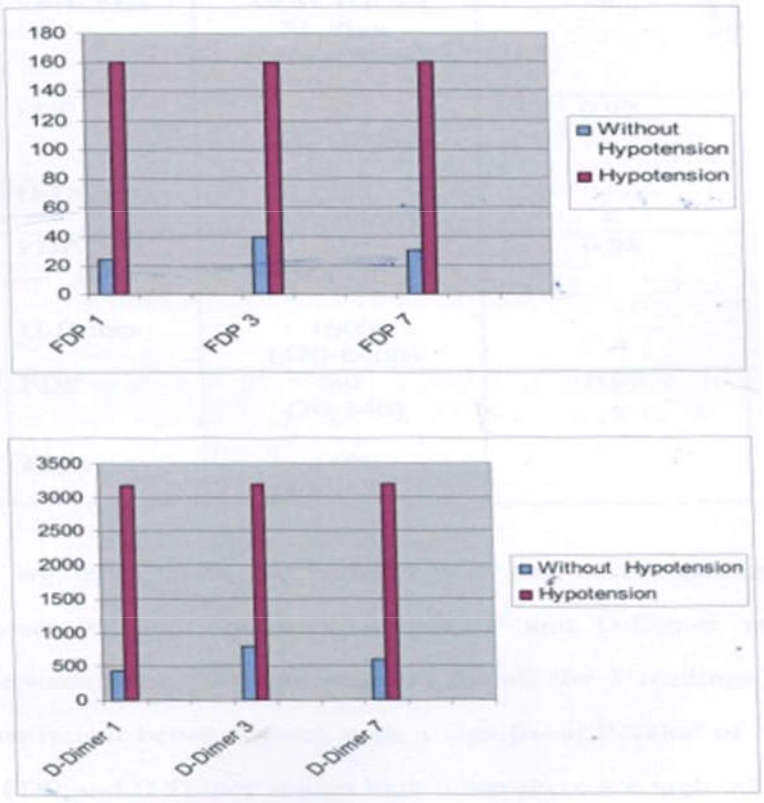

Fig. 6: Comparison of FDP and D-Dimer with Hypotention

Discussion

Acute Pancreatitis is associated with a wide range of clinical presentation varying from mild to severe clinical course with an overall mortality of about 10-15%. There is an need for an easier and more useful marker of severity, which is reliable and as a good as APACHE II scoring in predicting severity, prompted us to conduct this study in our institute.

In our study the average age of presentation of patient with acute pancreatitis was 43 years.

In our group of patients maximum organ failure was seen around 3rd day of onset of pain. In our study we found most common etiological factor was alcohol wgich may be due to high frequency of male patients (70-87%).

We have found in our study that direct correlation exist between APACHE II scores with D- Dimer & FDP value taken on any day. We also found significant correlation between APACHE II scores FDP & D – Dimer values when patients were divided into Mild & Severe Pancreatitis. FDP & D – Dimer values were significantly correlated with incidence of organ failure specifically respiratory, Renal & cardiovascular failure in form of hypotension.

Higher FDP & D- Dimer values directly correlates with severity of pancreatitis because when we compared FDP and D –Dimer with Severe pancreatitis as per APACHE II scores.

We have also found that there is no correlation between FDP & D – Dimer with day of organ failure, CT Severity Index or total duration of stay in hospital for a patient.

Conclusion

Incidence of acute pancreatitis is more common in males, with alcohol consumption is the main etiological factor. In females biliary pancreatitis Is more common. Organ failure are more common in first week after the attack of acute pancreatitis.

Among the organ failures, respiratory problems take the front seat, then comes the renal failure & hypotension.

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Case Report

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An Unusual Cause of Variceal GI Bleed

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Abstract

Background: Mesenteric vein thrombosis due to Mesenteric fibromatosis presenting with ectopic variceal bleed is less common. *Case Description*: 38 year old male presented with history of large volume melena. He was resuscitated and Endoscopy was done, showed multiple ectopic varices-bleeder in Duodenal third portion, hemostasis achieved by injecting Cyanocrylate glue. On reviewing, he had evaluation for pain abdomen, a year back which suggested inoperable retroperitoneal neoplasm. He had taken alternative medicine, being given a poor prognosis. Endosonography guided aspiration from lesion was suggestive of atypical cells. After 1 year, he had re-bleeding. Glue injection was done to another bleeding varix in D3. Repeat CT scan showed similar sized retroperitoneal mass. Diagnostic laparoscopy showed Omental cake like mass, histologically - mesenteric fibromatosis. *Clinical Relevance*: GI Bleed is a rare manifestation of Mesenteric fibromatosis and here it was variceal bleed secondary to thrombosis of the mesenteric vein, managed endoscopically. Patient had spontaneous Mesenteric fibromatosis and is stable for few years now. Hence, all retroperitoneal lesions need complete evaluation before giving a poor prognosis.

Keywords: Mesenteric Fibromatosis; Retroperitoneal Mass; Ectopic Variceal GI Bleeding; Glue Injection; CT abdomen; Endosonography; Diagnostic Laparoscopy; Histopathology.

Introduction

Mesenteric fibromatosis, also known as mesenteric desmoids, is part of the clinical-pathologic spectrum of deep fibromatosis, which encompasses a group of benign fibroproliferative processes that are locally aggressive and have the capacity to infiltrate or recur without metastasis. It is classified according to their anatomical location, whether it is intra-abdominal, from the deep soft tissues of the abdominal wall, or deep within the extra-abdominal soft tissues. It usually affects females and in fourth decade of life. They present with pain abdomen, bloating, weight loss or abdominal mass. It is associated with abdominal trauma, surgery or could be spontaneous. Gardner's syndrome is associated in few of them.

Surgery is definitive management. Chemotherapy is an alternative option. Radiation may be given in selected cases.

We hereby describe a patient whose presentation was unusual as it is less common in males, there was no associated Gardner's syndrome, nor any abdominal surgery nor trauma, but was detected on evaluation of gastrointestinal bleeding. Ectopic variceal bleeding due to thrombosis of Superior Mesenteric Vein due to infiltration from Mesenteric fibromatosis is rare. All retroperitoneal mass lesion patients need complete evaluation before giving a poor prognosis.

Case Report

A 38 year old male, was referred with history of black and maroon coloured stools for 5 days, requiring multiple blood transfusions. Endoscopy done else where was reported normal.

Patient was stabilised with transfusions and Pantoprazole infusion.

After stabilisation, Endoscopy was done which showed multiple bunches of varices and a bleeding varix in deep duodenum (D3).

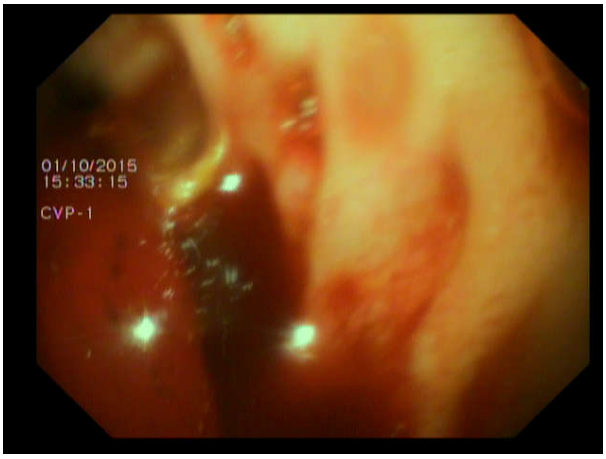

Fig. 1: Endoscopic image in D3: Blood in lumen

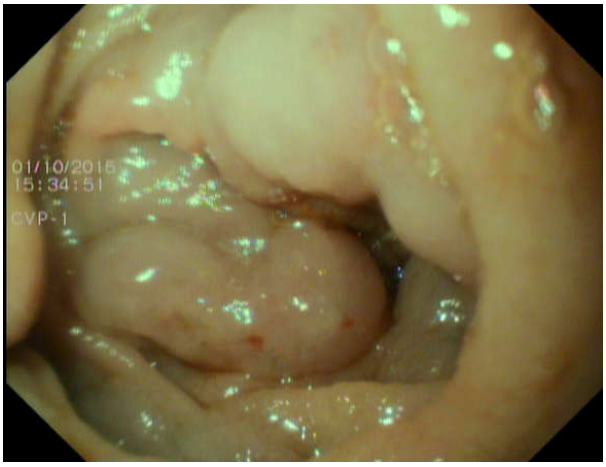

Fig. 2: Endoscopic image- Post glue injection

Two ml of Cyanocrylate glue was injected into the bleeding varix and hemostasis was achieved.

On reviewing history, patient had Episode of pain abdomen 1 year back for which he underwent evaluation elsewhere and found to have a mass in peripancreatic region. Endosonographiy guided fine needle aspiration was suggestive of atypical cells. Contrast enhanced CT abdomen showed inoperable retroperitoneal mass. He had taken alternative medicine-Tibetan medicine for the same.

CT Abdominal angiography was done which showed a poorly enhancing mass with hypodense rim of soft tissue encasing proximal SMA for a length of approximately 8 cms (3.1 x 4.0 cms in AP x Tr dimensions) with mild adjacent desmoplastic reaction- likely neoplastic. Mesentery showed increased perivascular stranding with enlarged mesenteric lymphnodes (average size 8 - 9 mm SAD). Splenoportal venous confluence is attenuated by the mass lesion with non-visualised SMV-? Attenuated / thrombosed. Multiple portovenous collaterals are seen in perigastric region, splenic hilum , mesentery and omentum.

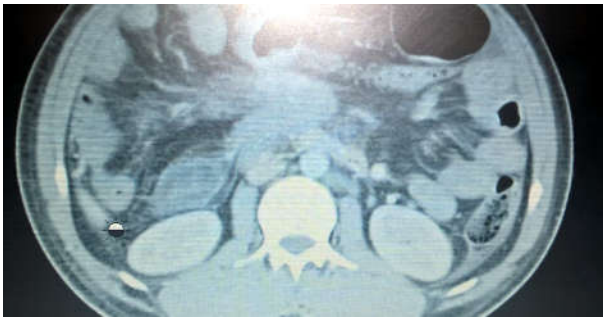

Fig. 3: CT image showing mass encasing vessels with collaterals

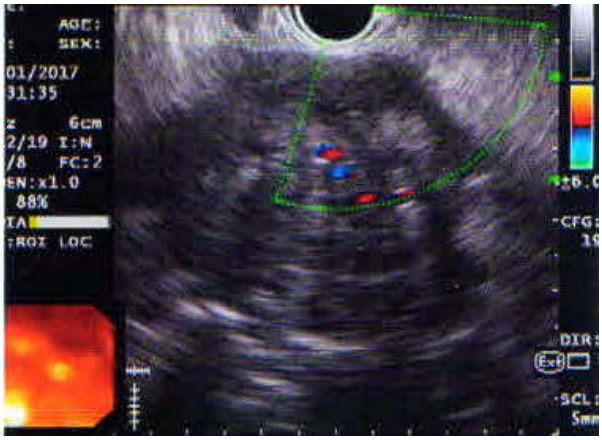

Fig. 4: EUS image of the mass lesion

Serum CEA, CA 19-9 was normal.

He was advised Endosonography guided FNA or diagnostic Laparoscopy after surgical consultation. Patient was stabilised and discharged on request on beta-blocker and hematinics.

After 3 months, patient came back for further management following another course of Tibetan medicine. EUS guided FNA was done which revealed atypical cells.

Antinuclear antibodies were negative and Immunoglobulin-IgG4 levels were within normal

range. He was advised diagnostic Laparoscopy and proceed tumour removal if feasible.

He came back after few months, Laparoscopy done which revealed omental cake like mass from which only biopsy could be taken.

Histopathology showed dense fibrosis with lympoplasmacytic infiltration. Immunohistiochemistry showed negative for lymphoma panel and mesenteric fibromatosis was diagnosed.

In view of association with Adenomatosis Polypsosis, colonoscopy was done which was normal. There was no eye symptom.

Patient was explained about disease prognosis and limited options in view of extensive disease and vascular involement. He was discharged on hematinics.

He came back after 6 months with another episode of bleeding.

Repeat endoscopy was done and another varix in duodenum was showing bleeding stigmata into which glue injection was done and haemostasis was achieved.

He is currently receiving beta-blocker and hematinics and doing well.

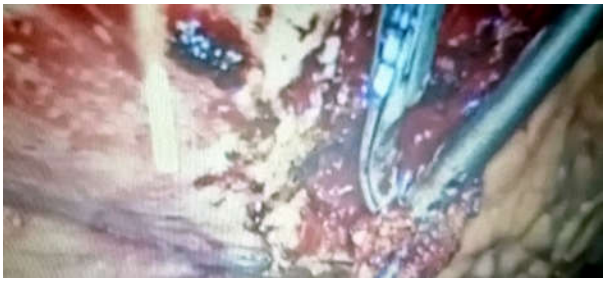

Fig. 5: Laparoscopic picture-omental cake

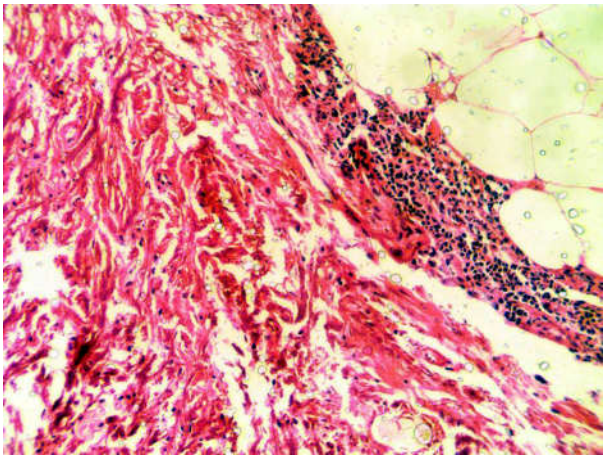

Fig. 6: Histopathology-H&E staining: Fibrosis, Collagen, chronic inflammation, no mitoses

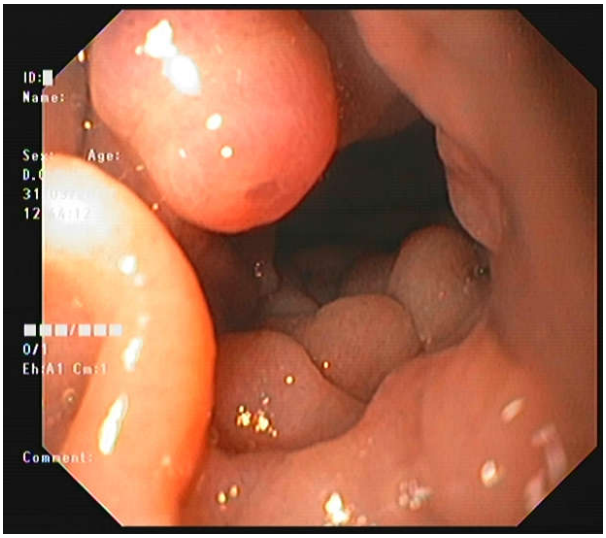

Fig. 7: Endoscopy image: Second varix with ulcer

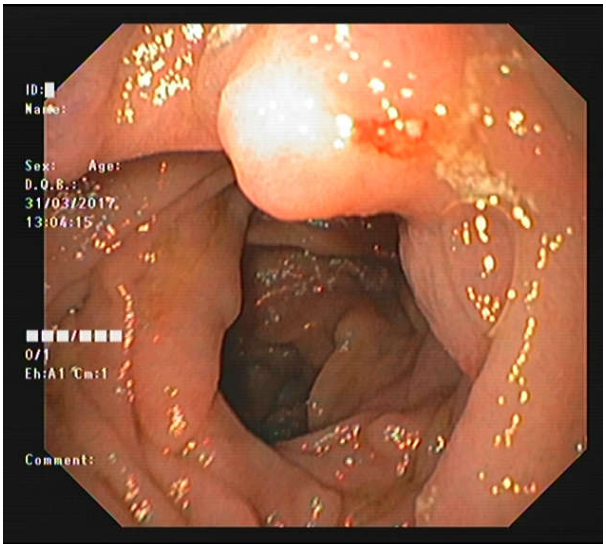

Fig. 8: Endoscopy image- Post Glue injection

Our case was unique because patient was male with no features of Gardner's syndrome and there was no abdominal trauma or surgery. He presented with bleeding due to varices which developed following SMV thrombosis. He is surviving with good functional status after being given a bad prognosis 3 years back. Hence, all retroperitonal lesions need complete evaluation before giving any prognosis.

Discussion

Mesenteric fibromatosis is a fibroblastic proliferation of the mesentery due to surgical trauma or spontaneously. Most of the cases are intra-abdominal and involve mesentery, however mesenteric desmoids account for less than 10% of

sporadic desmoid tumors [1,2]. They can arise spontaneously after surgical trauma or abdominal surgery. Thirteen percent of patients with mesenteric fibromatosis have FAP, specifically the Gardner syndrome variant of FAP [1]. Therefore, patients with FAP and a family history of mesenteric desmoids have a greater than 25% chance of developing this tumor [1]. Additionally, 83% of patients with FAP and mesenteric fibromatosis have a history of abdominal surgery, most commonly a total colectomy [1]. Disease is more aggressive in patients with FAP. Mesenteric fibromatosis is more common in female patients (80%) from 14 to 75 years of age (mean: 41 years), without any preference in race [1]. The female predilection is due to the fact that estrogen, even exogenous estrogen, is a factor that predisposes one to mesenteric fibromatosis and plays a role in its formation. MF occurs more frequently during pregnancy and in premenopausal women compared to postmenopausal women [3]. The patient usually presents with signs and symptoms related to the small bowel such as abdominal pain or a palpable abdominal mass, or clinical complications like small bowel obstruction, fistula formation, or bowel perforation [1]. Gastrointestinal bleeding is rare manifestation of mesenteric fibromatosis and it occurs if it involves the wall of bowel mimicking Gastrointestinal Stromal Tumour (GIST). Immunohistiochemistry can help differentiate mesenteric fibromatosis from GIST. Cd117, PDGFR, CD4 and DOG1 are positive in GIST and beta-catenin staining in mesenteric fibromatosis [13,14,15].

Although the natural course and clinical progression is not predictable, some of them remain stable for long and some regress over time.

The management of mesenteric fibromatosis includes multiple modalities. Surgical resection is definitive in non invading tumours. 53-67% of cases, it is operable [1,5]. Hormonal therapy with Tamoxifen or interferon and NSAIDS with chemotherapy can play a role in the treatment due to the fact that local recurrence is high, mainly in Gardner's syndrome patients. Doxorubicin is the preferred chemotherapy agent [5]. Post operative complications like short bowel and enterocutaneous fistula can occur. If resected incompletely, it tends to recur and are locally aggressive [5,6].

Vinblastine and Methotrexate with Tamoxifen are used if tumour recurs. Tyrosine kinase inhibitor, Imatinib is an alternative if there are multiple tumours.

Indomethacin and sulindac can be tried in unresectable tumours. Prednisolone and Azathioprine have been shown success in case reports [6].

Radiation therapy has a small role in mesenteric fibromatosis treatment in intraabdominal Desmoids and without any involvement of vessels [5,6].

Our patient had locally advanced disease and hence not a candidate for surgery. Radiation is contra-indicated in view of vascular involvement and extensive disease. Chemotherapy was not considered after discussing with patient. He is currently receiving hematinics and beta-blockers and doing well with 3 years of follow up. Mesenteric fibromatosis could have a long term stable course and hence patients with retroperitoneal mass should be thoroughly evaluated for possible surgery or alternative modalities and followed up for disease related complications and not to be labelled as having poor prognosis.

Conclusion

Mesenteric fibromatosis is fibroblastic proliferation affecting the mesentery after surgical trauma or spontaneously and in Familial adenomatous Polyposis. It is more common in females and is locally aggressive. Surgery, Chemotherapy and Radiation therapy are the options for management.

This case was unusual as the patient presented with recurrent ectopic variceal bleeding due to mesenteric vein thrombosis managed endoscopically. Patient is doing well with three years follow up after being labelled as inoperable retroperitoneal neoplasm. All retroperitoneal mass lesions need complete evaluation before giving a bad prognosis.

Acknowledgements

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A Case of Spontaneous Celiac Artery Dissection with Unusual Presentation

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Abstract

Spontaneous dissection of celiac trunk is quiet an uncommon medical condition, with less than forty case reports in the medical literature. Due to rarity of this disease in clinical practice and non-specific nature of symptoms, a high degree of clinical suspicion is needed. Epigastric pain is most common presenting symptom. Symptomatic spontaneous celiac artery dissection is a rare condition that is being detected more often with the use of advanced imaging techniques. The ideal treatment has not yet been established but the available strategies are conservative medical management, endovascular intervention or surgical revascularization. We describe the case of 55 year old man who presented with severe upper abdominal pain , dynamic small bowel obstruction and diagnosed as spontaneous celiac artery dissection on imaging , confirmed on angiography. We provide a review of the current literature about imaging finding and management of this rare entity.

Keywords: Celiac Artery; Bowel Obstruction; Angiography.

Introduction

Isolated spontaneous celiac artery dissection is rare. Most reported cases have occurred in men, and the cause and natural history of the condition are not well understood. Epigastric pain is most common presenting symptom. Symptomatic spontaneous celiac artery dissection is a rare condition that is being detected more often with the use of advanced imaging techniques. The ideal treatment has not yet been established but the available strategies are conservative medical management, endovascular intervention or surgical revascularization.

Case Report

A 55 year old previously healthy man had diffuse upper abdominal pain, three days prior to presentation, which was non radiating and intermittent, intensity ranging from moderate to severe. The patient also had bilious vomiting with abdominal distension and constipation one day prior to presentation. He denied history of trauma, fever, rigors, bright red blood per rectum or melena. Patient had no history of any significant medical or surgical conditions in the past including diabetes and hypertension or heart disease.

On physical examination, his vital parameters were normal with a blood pressure of 130/70 mm Hg and heart rate of 88 bpm. There was mild upper abdominal distension with mild tenderness in epigastric region and reduced bowel sounds without guarding or rigidity. Patient was managed with nasogastric aspiration, analgesics and intravenous fluids for suspected dynamic small bowel obstruction. Complete blood count, blood sugar,renal function tests, liver function tests, electrolytes, ESR, CRP, amylase and lipase, were within normal limits. LDH, HbA1c and lipid profile were also within normal limits. Electrocardiogram and chest x ray was normal.

A helical CT scan of the abdomen and pelvis with IV contrast was performed which showed a three cm long segment of dissection of celiac artery about 6mm from its origin from aorta with possible pseudo aneurysm in distal artery, luminal narrowing and extension of dissection into the bifurcation of artery into splenic artery and common hepatic artery (Figure 1,2). The aorta, superior mesenteric artery and its branches were all normal on CT scanning. Patient underwent celiac artery angiography and showed findings similar to CT scan report. He underwent work up for vasculitis (p-ANCA, c-ANCA, ANA profile) which was negative. The patient was treated conservatively with low molecular weight heparin in the therapeutic doses and supportive treatment. His symptoms improved within two days, he was symptom free five days after hospitalization and discharged in stable condition on oral anticoagulation medications with a target INR of 2.5. He was completely symptom free on outpatients follow up after a month with normal quality of life.

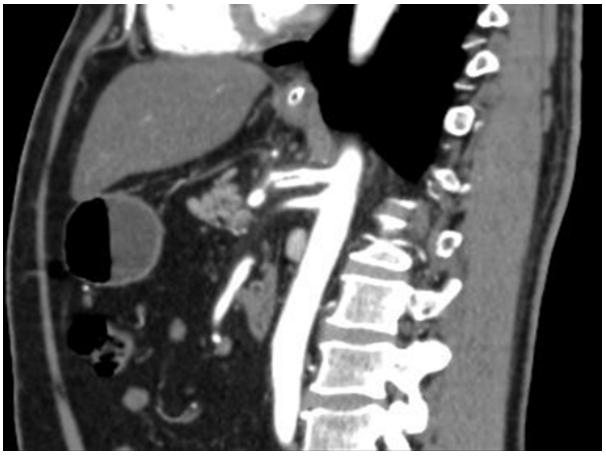

Fig. 1: Dissection of celiac artery about 6mm from its origin from aorta on CT abdomen with contrast

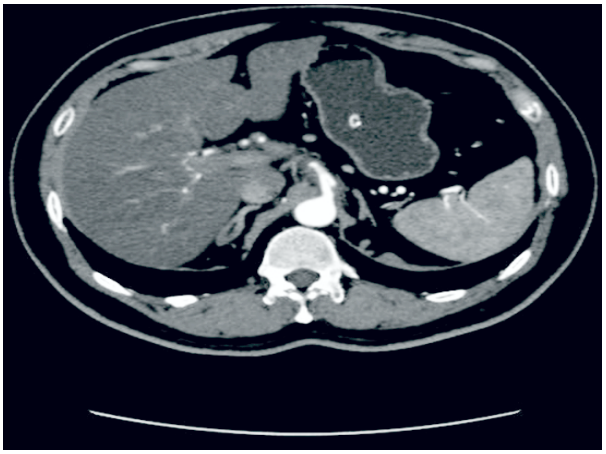

Fig. 2: Dissection of celiac artery - axial images of CT abdomen with contrast

Discussion

Arterial dissection is defined as cleavage of two layers of the arterial wall caused by intramural hematoma [5]. The first reported case of spontaneous celiac artery dissection was described in 1959[3]. Only 13 cases were reported before 2001[4]. A standard search made on Medline database reveals less than 40 cases of isolated celiac artery dissection with aneurysm. Spontaneous arterial dissection is 5 times more common in men than in women, and the average age of the patients is approximately 55 years [1].

Predisposing factors have been suggested to be pre-existing vascular disease, hypertension, pregnancy, trauma and degeneration of arterial wall however, no definite cause was found in many cases [2,4]. Most patients with celiac artery dissection are asymptomatic possibly due to the lack of small bowel involvement. Some patients may present with abdominal pain which may be due to simultaneous involvement of the splenic, renal or superior mesenteric arteries causing infarction and bowel ischemia. Patients with ruptured arterial aneurysms present acutely with bleeding. Chronic dissection can present with symptoms of intestinal angina i.e. post-prandial abdominal pain and weight loss [4]. Magnetic resonance imaging, doppler ultrasonography, and conventional angiography have been used in the diagnosis of splanchnic artery dissections; however, CT angiography is considered to be the imaging technique of choice [1,4].

Diagnostic imaging findings on CT according to Kim et al. include an intimal flap, which is pathognomonic or eccentric mural thrombus in the celiac lumen, which should raise suspicion for dissection [1]. Because the intimal flap is not always visible, mural thrombus may be the only clue to the presence of dissection. The natural progression of spontaneous dissecting celiac artery aneurysm is unclear. Aneurysm rupture with intraperitoneal bleeding, distal propagation of dissection with branch vessel involvement, end-organ infarctions, and intestinal ischemia are some of the serious complications on follow-up [6]. Management of spontaneous dissecting celiac artery aneurysm should be a case-based approach. Conservative medical management, surgical and endovascular techniques are the treatment options available. Medical management consists of anticoagulative therapy. Continuous heparin administration is recommended while the patient is fasting or until the abdominal pain abates [8,9]. Therapy can later be changed to oral warfarin until improvement is

evident. If the need for anticoagulative therapy exceeds 6 months, we recommend that an invasive strategy be considered, because lifelong warfarin therapy has no proven benefit in patients with celiac artery dissection. Some authors have advocated anti platelet therapy during the acute stage of spontaneous dissection, because subendothelial injury can trigger thrombosis, [7,10]. Strict blood pressure control might prevent propagation of the dissection. Potential advantages of stenting over surgery include shorter hospital stays, less need for anticoagulation, and reduced radiation exposure from serial imaging. Potential disadvantages include stent thrombosis, restenosis, and procedure-related sequelae such as access-site complications.

Conclusions

An isolated dissection of the visceral arteries, particularly of the celiac artery, is extremely rare. They typically present with abdominal pain, and are occasionally associated with haemorrhage. Although treatment strategies are somewhat unclear, medical management and close observation is appropriate for uncomplicated lesions. Surgical management is the preferred treatment for those patients who have associated complications or persistent or recurrent symptoms. For those patients who are not good surgical candidates, endovascular techniques provide other potential treatments options for celiac artery dissection. Early diagnosis and treatment is important for better outcome.

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Rarest Complication of Thyroid Surgery-Esophagus Injury Repair with Sternocleidomastoid Flap: A Case Report with Review

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Abstract

The most common complications after thyroidectomy areinjury to the recurrent laryngeal nerve and parathyroid glands. Cervical esophagus perforation is an exceptionally rare complication after thyroidectomy; it can usually be resolved by conservative care. The most common cause of cervical esophagus is anterior spinal surgeries and there are reports of closure using muscle flaps esp. erector colli. In this report we are sharing our experience of cervical esophagus transection with tissue loss following thyroidectomy and presenting in the acute phase. Exploration was done and repair of esophagus by mobilization and reinforcing with sternocleidomastoid muscle from right side. The defect healed well with no stricture for a follow up period of 1 yr.

Keywords: Thyroidectomy Complications; Esophageal Perforation; Esophageal Transection; Sternocleidomastoid Muscle Flap.

Introduction

Thyroidectomy is a common surgery without many complications. The common complications are recurrent laryngeal nerve injury, parathyroid deficiency, and rarely injury to trachea due to adhesions. Usually the esophagus is not coming in the field and no injury is possible. We came across a rare situation where thyroidectomy was associated with a major esophageal injury and leakage of content through the drain reported in 24 hrs of surgery.

Case Report

32 yrs. old female patient presented with solitary nodule of thyroid of the right lobe and FNAC was reported as Multinodular goiter and patient underwent total thyroidectomy in a hospital. The drain was placed and the drainage was minimal. On the 1st postoperative day the fluid takenorally by the patient was drained through the drainage tube. Then suspected esophageal injury and referred to us.

On examination the drain was draining saliva and the fluids taken orally. X-ray of neck and chest followed by CT scan was done to rule out pneumomediastinum. There was no collection or features of inflammation down in the mediastinum.

Exploration of the wound done on the 1st postoperative day through the same incision and it was found that the esophagus was totally divided and a portion was missing. Nasogastric tube was seen through the wound.

Suction and cleaning of the wound done and esophagus mobilized from both ends and anastomosed. The inflamed area was covered with rt. Sternocleidomastoid muscle flap and it is fixed to the prevertebral fascia and wrapped aroundthe anastomotic site.

The patient was put on nasogastric tube feed for 3 wks. Then a barium fluoroscopy done andfound that

there was no leak or stricture.Oral fluid was started first and after 24 hrs. solid or semisolid food was also given. The patient was followed up for 12 months and no stenosis or stricture reported.

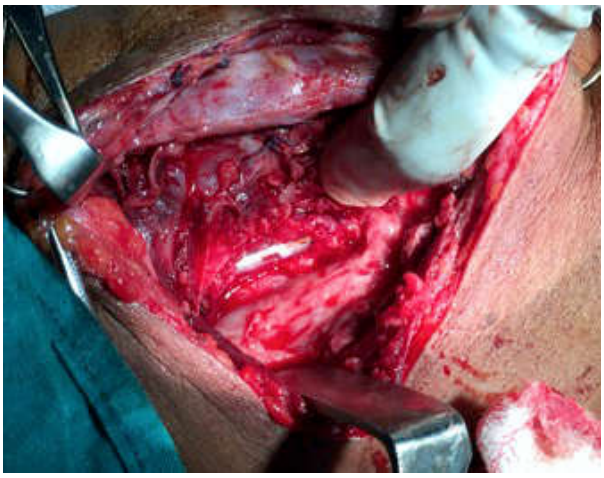

Fig. 1: Nasotracheal tube through the divided esophagus

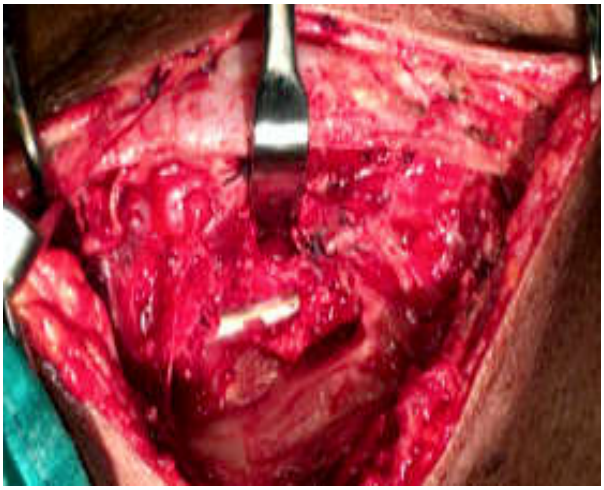

Fig. 2: Mobilized esophagus

Fig. 3: Sternocleidomastoid muscle flap [fig a & b]

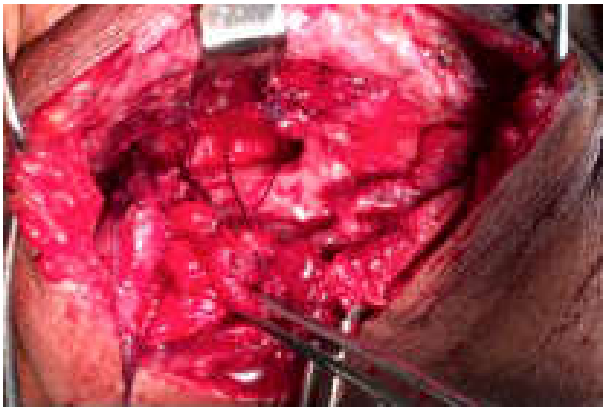

Fig. 4: The sternomastoid is fixed to the prevertebral fascia and wrapped around the anastomosis

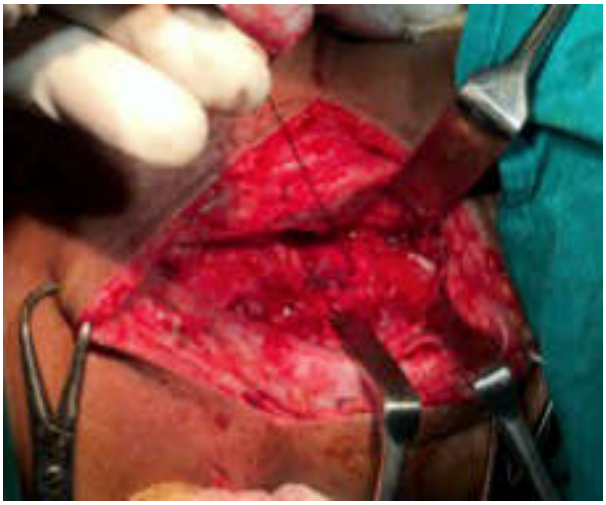

Fig. 5: Final stage of reconstruction

Discussion

Many cases of recurrent laryngeal nerve injury and hypoparathyroidism were reported in thyroidectomies. But injury to trachea and esophagus are very rare.

The esophageal injury were reported were simple tear or opening and preoperative repair with silk were done and they healwell [1,2,3]. There were cases reported as late presentation of esophageal injury with stricture. Cervical esophageal injuries are more common with anterior spinal surgeries [4,5]. They are either repaired or cover with sternocleidomastoid or cervical colli muscleflaps [4].

In our patient the transection of esophagus was happened with loss of some portion. So the esophagus mobilized from both ends and approximated in 2 layers i.e., the mucosa which was edematous and the muscular coat. To avoid leak and to improve the healing the anastomotic area is reinforced with sternocleidomastoid muscle, which

was taken from RT side and fixed to prevertebral fascia. It took three weeks to heal the flap to settle as in a normal case of flap surgery and the leak stopped.

Follow up after 12 months didn't give any history of difficulty in swallowing or evidence of stricture. Barium swallow was done and it was normal.

The sternocleidomastoid muscle is having two heads at the sternoclavicular area and the lower portion is tendinous. The upper part is muscular with multiple blood supply. A single axial blood flow is not there in sternocleidomastoid and so mobilization should be limited and it is detached from the sternoclavicular end and fixed to the prevertebralfascia towards opposite side. Esophagus is having very less blood flow in the neck area and too the posterior part, so that leak is more due to poor healing.

Conclusion

A rare complication thyroidectomy is discussed with method to reconstruct the anastomosis by reinforcing with a muscle flap. The esophageal injuries are very rare and only small injuries are reported which are treated conservatively. There are reports of such cases in literature but total transection is rare and hence we are presenting this article.

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Include summary of key findings (primary outcome measures, secondary outcome measures, results as they relate to a prior hypothesis); Strengths and limitations of the study (study question, study design, data collection, analysis and interpretation); Interpretation and implications in the context of the totality of evidence (is there a systematic review to refer to, if not, could one be reasonably done here and now?, What this study adds to the available evidence, effects on patient care and health policy, possible mechanisms)? Controversies raised by this study; and Future research directions (for this particular research collaboration, underlying mechanisms, clinical research). Do not repeat in detail data or other material given in the Introduction or the Results section.

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[1] Flink H, Tegelberg Å, Thörn M, Lagerlöf F. Effect of oral iron supplementation on unstimulated salivary flow rate: A randomized, double-blind, placebo-controlled trial. J Oral Pathol Med 2006; 35: 540-7.

[2] Twetman S, Axelsson S, Dahlgren H, Holm AK, Källestål C, Lagerlöf F, et al. Caries-preventive effect of fluoride toothpaste: A systematic review. Acta Odontol Scand 2003; 61: 347-55.

Article in supplement or special issue

[3] Fleischer W, Reimer K. Povidone iodine antisepsis. State of the art. Dermatology 1997; 195 Suppl 2: 3-9.

Corporate (collective) author

[4] American Academy of Periodontology. Sonic and ultrasonic scalers in periodontics. J Periodontol 2000; 71: 1792-801.

Unpublished article

[5] Garoushi S, Lassila LV, Tezvergil A, Vallittu PK. Static and fatigue compression test for particulate filler composite resin with fiber-reinforced composite substructure. Dent Mater 2006.

Personal author(s)

[6] Hosmer D, Lemeshow S. Applied logistic regression, 2nd edn. New York: Wiley-Interscience; 2000.

Chapter in book

[7] Nauntofte B, Tenovuo J, Lagerlöf F. Secretion and composition of saliva. In: Fejerskov O, Kidd EAM,

editors. Dental caries: The disease and its clinical management. Oxford: Blackwell Munksgaard; 2003. p. 7-27.

No author given

[8] World Health Organization. Oral health surveys - basic methods, 4th edn. Geneva: World Health Organization; 1997.

Reference from electronic media

[9] National Statistics Online—Trends in suicide by method in England and Wales, 1979-2001. www.statistics.gov.uk/downloads/theme_health/HSQ 20.pdf (accessed Jan 24, 2005): 7-18. Only verified references against the original documents should be cited. Authors are responsible for the accuracy and completeness of their references and for correct text citation. The number of reference should be kept limited to 20 in case of major communications and 10 for short communications.

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