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Amyloidosis of the colon

Yvonne M. Dawkins, Barrie Hanchard, Michael G. Lee

ABSTRACT

Introduction: Gastrointestinal amyloidosis is rare in western countries. The most frequent clinical manifestation in all patients with amyloidosis is weight loss and gastrointestinal bleeding. However, diarrhea is seen in patients with secondary amyloidosis. Case Report: A 76-year-old male presented with a six-month history of diarrhea, and had three episodes of bloody diarrhea over a two-day period prior to admission. There was associated anorexia and weight loss. Colonoscopy revealed pancolitis and a presumptive diagnosis of ulcerative colitis was made. He had resolution of his diarrhea after four days on prednisone. However, biopsy revealed colonic amyloid deposition. He also had non-nephrotic range proteinuria and cardiomyopathy and likely had systemic amyloidosis, affecting the heart and kidney in addition to colonic involvement. Conclusion: The intestinal presentation may be similar to several conditions as a result of the protean manifestations of gastrointestinal amyloidosis. A high index of suspicion should be maintained in any patient presenting with chronic diarrhea and edema to avoid delay in diagnosis and treatment.

Keywords: Amyloidosis, Colitis, Colonoscopy, Diarrhea

INTRODUCTION

Amyloidosis may involve the gastrointestinal tract, mainly the upper intestinal tract but involvement of the colon may occur. Gastrointestinal amyloidosis is relatively rare in western countries occurring in 3–8% of patients with systemic amyloidosis, but a recent report from Korea found 15.5% gastrointestinal involvement [1]. The most frequent clinical manifestation in all patients with amyloidosis are weight loss and gastrointestinal bleeding [2]. However, diarrhea is seen in patients with secondary amyloidosis [1]. The majority of patients with amyloidosis (80%) have primary amyloidosis, and most of these patients are over 50 years old [2]. In primary amyloidosis, amyloid deposition occur in the muscularis mucosae, submucosa, and muscularis propria leading to the thickening of intestinal folds, and usually presents with constipation, mechanical obstruction, or chronic intestinal pseudo-obstruction [3].

We present the case of an elderly male with chronic diarrhea and weight loss. Colonoscopy revealed pancolitis and colon biopsy revealed amyloid deposition.
CASE REPORT

A 76-year-old Jamaican male presented to the University Hospital of the West Indies, Jamaica with a six-month history of diarrhea, which he describes as four episodes of loose non-bloody stools daily. This was associated with anorexia and 10 pounds weight loss in six months. He presented to hospital after experiencing three episodes of bloody diarrhea over a two-day period. He denied urgency, tenesmus, abdominal pain, nausea, vomiting or symptoms of anemia. He has not experienced similar symptoms in the past. He is an ex-smoker and denies any history of recent antibiotic exposure, recent travel or a personal or family history of colon cancer or inflammatory bowel disease. His past medical history was significant for congestive cardiac failure which was thought to be due to ischemic heart disease diagnosed four weeks earlier after presenting with a history of bilateral lower limb edema. The echocardiogram revealed moderate concentric left ventricular hypertrophy with ejection fraction 65–70%, mild diastolic dysfunction, moderately enlarged right atrium, moderately dilated left atrium and small generalized pericardial effusion with no evidence of cardiac tamponade.

On examination he was noted to have pale mucous membranes, moderate generalized wasting and moderate pedal edema. His cardiovascular examination was significant for low volume pulses but was otherwise normal. His abdominal examination revealed shifting dullness but no organomegaly. His rectal examination showed a moderately enlarged firm prostate with no focal lesion. His respiratory and nervous system examinations were unremarkable.

Investigations showed hemoglobin 8.9 g/dl (normal range: 11.5–16.5 g/dl), MCV 83.9 fl (normal range: 81.1–96.0 fl), MCH 27.6 pg (normal range: 27.0–31.2 pg), platelets 229x10^9/L (normal range: 150–450 x10^9/L), white blood cell count 5.2x10^9/L (normal range: 3.75–11.0 x10^9/L), total protein 37 g/L (normal range: 68–84 g/L), albumin 19 g/L (normal range: 38–52 g/L), globulin 18 g/L (normal range: 18–38 g/L), alkaline phosphatase 240 U/L (normal range: 15–105 U/L), AST 16 U/L (normal range: 7–32 U/L), total bilirubin 11 umol/L (normal range: 4–18 umol/L), ferritin 273 ng/ml (normal range: 16–294 ng/ml), vitamin B12 506 pmol/L (normal range: 156–698 pmol/L), folic acid 6 pg/ml (normal range: 2–20 pg/ml), PSA 3.74 ug/L (normal range: 0.00–4.00 ug/L). His spot urine protein/creatinine ratio was 1.67 g/day. He had normal clotting indices, urea, creatinine and electrolytes.

The abdominal ultrasound revealed prostatomegaly with features of chronic bladder outlet obstruction, features suggestive of bilateral renal parenchymal disease, small volume ascites and small bilateral pleural effusions. Colonoscopy showed mild to moderate pancolitis characterized by granularity of the mucosa, blunting of the normal vascular pattern, friability of the mucosa with scattered superficial ulcerations. This was thought to be suggestive of ulcerative colitis. Biopsies, however, revealed colonic mucosa in which the general architecture is distorted by the deposition in the lamina propria of amorphous eosinophilic material. This stained positively with Congo red stains, confirming the presence of amyloid (Figure 1). He was assessed as having colonic amyloidosis as part of secondary amyloidosis.

After his presumptive diagnosis of ulcerative colitis he was started on prednisone 40 mg daily with complete resolution of his diarrhea after four days but he continued to experience persistent lower limb edema, ascites and hypoalbuminemia. He was treated symptomatically but he demised in hospital.

DISCUSSION

Amyloidosis is a rare disorder and refers to the extracellular deposition of insoluble amyloid fibrils in the tissues of the body leading to end organ damage. Depending on the precursor protein, clinical manifestations may vary significantly. Amyloid can be confined to an organ or may be systemic. In systemic amyloidosis, the heart, kidneys, and nerves are most commonly affected, resulting in congestive heart failure, arrhythmia, nephrotic syndrome, renal failure, and peripheral and autonomic neuropathies [4]. Isolated gastrointestinal amyloidosis is rare [5]. In one series, the most common presentations for gastrointestinal amyloidosis were weight loss (45%) and gastrointestinal bleeding (36%) [5]. In another report, the most common symptoms of gastrointestinal amyloidosis were diarrhea (45.8%), anorexia (37.5%), weight loss and nausea and/or vomiting (29.2%) and the histologically confirmed gastrointestinal tract site was the stomach (55.0%), colon (45.0%) and rectum (35.0%). Patients with gastrointestinal involvement had a greater frequency of organ involvement [1].

The endoscopic appearance of gastrointestinal amyloidosis is non-specific and may include a fine granular appearance of the mucosa, erosions, ulcerations, mucosal friability and polypoid protrusions [6]. These findings may reflect amyloid deposition in the mucosa or submucosa. In one study, the degree of amyloid deposition was most marked in the duodenum and...

Figure 1: Congo red stain of colon mucosa showing deposition of amorphous eosinophilic material in the lamina propria, distorted architecture and positive staining with Congo red (black arrow) confirming the presence of amyloid.
significantly correlated with the frequency of endoscopic findings of fine granular appearance and polypoid protrusions [6]. In secondary amyloidosis (serum acute phase-reactant, amyloid A protein), endoscopic findings have been related to deposition occurring mainly in the propria mucosae with symptoms of diarrhea, as in our patient [3]. The endoscopic findings in our patient were similar to ulcerative colitis. There are no pathognomonic radiologic or endoscopic findings, and diagnosis is usually delayed. Ultimately, a biopsy of the affected region of the gastrointestinal system is required to confirm the diagnosis and exclude other causes. Characteristically, tissue biopsy has positive staining of amyloid by Congo red or amyloid fibrils on electron microscopy. Further evaluation should thereafter be performed to determine the type of amyloid and the underlying cause. Our patient had biopsy proven colonic amyloidosis but in light of his non-nephrotic range proteinuria and cardiomyopathy, he likely had systemic amyloidosis, affecting the heart and kidney in addition to colonic involvement.

The intestinal presentation may be similar to several conditions. As a result of the protean manifestations of gastrointestinal amyloidosis, the diagnosis is often delayed and requires a thorough history, physical examination and appropriate investigations including an upper endoscopy and/or colonoscopy. A high index of suspicion should be maintained in any patient presenting with diarrhea and edema so as not to delay diagnosis and treatment.

Current therapies suppress or stabilize the precursor protein formation and interfere with fibrillogenesis [4]. The treatment of gastrointestinal amyloidosis is aimed at symptomatic control. Patients with severe hypoalbuminemia and chronic diarrhea due to protein-losing enteropathy may respond to combination therapy with a somatostatin analogue, like octreotide and corticosteroid [7, 8]. This therapeutic option should be considered not only in AA amyloidosis, but also in systemic amyloidosis, because of the lack of specific therapies in this serious condition [7]. The underlying cause of the amyloidosis should be treated and this may result in lasting regression of the gastrointestinal amyloidosis [9–11]. The prognosis of patients with AL amyloidosis (amyloid light-chain amyloidosis) and gastrointestinal involvement was poorer than those without gastrointestinal involvement, and they presented with more organ involvement and more advanced disease than those without organ involvement [1].

**CONCLUSION**

The intestinal presentation may be similar to several conditions as a result of the protean manifestations of gastrointestinal amyloidosis. A high index of suspicion should be maintained in any patient presenting with chronic diarrhea and edema to avoid delay in diagnosis and treatment.

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**Author Contributions**

Yvonne M. Dawkins – Substantial contributions to conception and design, Acquisition of data, Drafting the article, Revising it critically for important intellectual content, Final approval of the version to be published. Barrie Hanchard – Substantial contributions to conception and design, Acquisition of data, Revising it critically for important intellectual content, Final approval of the version to be published. Michael G. Lee – Substantial contributions to conception and design, Drafting the article, Revising it critically for important intellectual content, Final approval of the version to be published.

**Guarantor**

The corresponding author is the guarantor of submission.

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**REFERENCES**

Early laparoscopic treatment of an obstructed paracecal hernia in an octogenarian: A case report

Robert Cooke, Nicholas Heywood, Abhiram Sharma, Velauthan Rudralingham

ABSTRACT

Paracecal hernias are a rare cause of small bowel obstruction. The management of these has been predominantly by laparotomy. An 84-year-old male was presented who underwent early laparoscopic management of a paracecal hernia. This resulted in avoidance of laparotomy and a short postoperative hospital stay.

Keywords: Paracecal hernia, Bowel obstruction, Laparoscopy

INTRODUCTION

Small bowel obstruction is a common presentation to the acute surgical take. It is most commonly due to adhesions, however up to 5.8% of cases are caused by internal hernias. Of which paracecal hernias account for up to 6.6% [1, 2]. Clinical diagnosis can be difficult especially in the virgin abdomen as signs are non-specific. Computed tomography scan is the current main tool for diagnosis but the exact findings are only usually identified during surgery. Laparotomy has been the mainstay for management for these patients [3].

CASE REPORT

An 84-year-old male with no previous history of abdominal surgery presented to accident and emergency department with a one day history of intermittent abdominal pain. He denied any nausea or vomiting, however, noted a reduced stool frequency in the preceding two weeks. His past medical history included ischemic heart disease, cerebrovascular disease, diabetes, hypertension, pulmonary fibrosis, benign prostatic hyperplasia and he had an abdominal aortic aneurysm under surveillance.

Initial assessment of the patient’s abdomen revealed no tenderness or distension and physiological observations were within normal limits. Blood results were unremarkable except for a mildly elevated C-reactive protein of 42. During a period of observation he experienced increasing pain and developed tenderness in the right iliac fossa.

A computed tomography scan was performed, revealing a localized incarcerated segment of small bowel lateral to the cecum in the right paracolic gutter with upstream dilatation of small bowel loops lying anterior to the ascending colon. The associated localized mesenteric congestion and fluid surrounding the localized segment of small bowel in the right iliac fossa were suggestive of a paracecal hernia.

The patient was taken to theatre for laparoscopy within 24 hours of admission. A paracecal hernia containing an
obstructing loop of jejunum was identified between the cecum and lateral abdominal wall. Multiple adhesions in this area created a narrow necked blind ending ‘hernia cave’ in the right paracolic gutter. An adjacent internal hernia contained an unobstructed vermiform appendix. Laparoscopic reduction of the small bowel from the hernia was performed and adhesiolysis undertaken to prevent recurrence. The patient had an uncomplicated postoperative course, returned to baseline function and was discharged on the third postoperative day.

DISCUSSION

A hernia is defined as the protrusion of a viscus through a defect in the wall of the cavity in which it resides. When bowel passes through a peritoneal defect or mesenteric aperture, it is known as an internal hernia [4]. Internal hernias account for up to 5.8% of presentations of small bowel obstruction, of which paracecal hernias make up only 6.6% [1].

The paracecal peritoneum develops once the midgut has completed migration. The result is formation of four paracecal recesses being formed. These are the superior ileocecal recess, inferior ileocecal recess, retrocecal recess and paracolic sulci. These all have the potential to form hernial orifices [5].

Symptoms of abdominal pain and vomiting from paracecal hernias are variable and depend on the degree of incarceration or obstruction. Often due to their anatomical location, they may be mistaken for other right iliac fossa pathology, such as appendicitis [1, 4].

When there is diagnostic uncertainty, or if internal hernia is suspected, computed tomography scan is the preferred investigation of choice. Aside from confirming the diagnosis, this readily available imaging provides information about the presence of strangulation and ischemia as an aid to preoperative planning. The presence of a cluster of dilated small bowel loops, a transition

Figure 1: Cluster of small bowel loops incarcerated lateral to the cecum in the right paracolic gutter.

Figure 2: Stretched mesentery, disproportionate edema and inter-loop fluid in affected segment-concern for vascular compromise.

Figure 3: (A, B) Intraoperative images of the paracecal hernia caused by multiple adhesions.

Figure 4: Laparoscopic reduction of the hernia.
point lateral to the cecum and congestion of mesenteric vessels raise suspicion for an obstructing paracecal hernia. The cecum can sometimes be seen to have been displaced anteromedially. Other imaging modalities such as plain X-ray films and contrast studies often provide limited information and may result in delayed diagnosis subsequently increasing the risk of complications [2, 4, 6].

Laparotomy has generally been the preferred approach for the presence of small bowel obstruction, however, many studies including O’Connor et al. review of over 2000 cases have shown the safety and efficacy of laparoscopic surgery [7]. The debate over the approach is on-going because of the technical difficulties and potential complications associated with laparoscopic surgery in these patients. These include damage to distended small bowel loops and visualization of the transition point, making a high conversion rate to laparotomy and morbidity. However, benefits of laparoscopic surgery in small bowel obstruction have been shown, these are reduction in postoperative hospital stay, reduction in morbidity, reduction in adhesion formation and faster return to bowel function [7].

A recent review of published cases of paracecal hernias by Ogami et al. [3] highlighted that the vast majority of reported cases have been managed by laparotomy and that only three cases have been managed laparoscopically, none of which were performed within the UK. On reviewing these three case reports [3, 8, 9] all were performed after several days of conservative treatment which had not responded, in comparison to our patient who was operated on within 24 hours. All the cases showed viable bowel upon reduction of the hernia and no resections were required. The postoperative courses were uneventful with patients being discharged between 9 and 10 days postoperatively. In our case, the prompt diagnosis and early laparoscopic intervention had a reduced length of stay in comparison to the previously discussed cases.

CONCLUSION

We have demonstrated the safe use of early laparoscopic reduction of paracecal hernia and division of adhesions in an elderly patient. We have avoided the need for laparotomy in this patient resulting in early return to function, avoiding the morbidity associated with open abdominal surgery. Early computed tomography scan and laparoscopic approach should be considered in this group of patients.

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Author Contributions

Robert Cooke – Substantial contributions to conception and design, Acquisition of data, Analysis and interpretation of data, Drafting the article, Revising it critically for important intellectual content, Final approval of the version to be published

Nicholas Heywood – Analysis and interpretation of data, Revising it critically for important intellectual content, Final approval of the version to be published

Abhiram Sharma – Analysis and interpretation of data, Revising it critically for important intellectual content, Final approval of the version to be published

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Guarantor

The corresponding author is the guarantor of submission.

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REFERENCES

Report of a case of pancreatic hemangioma: A difficult preoperative diagnosis

AL Hashmi Al Warith, Lagrange Xavier, Fara Régis, Camerlo Antoine

ABSTRACT

Hemangiomas can be found in various organs in the gastrointestinal tract but are rarely described in the pancreas. We report here a case of 71-year-old female who presented on abdominal computed tomography (CT) scan an incidental finding of cystic lesion in the tail of the pancreas. Follow-up magnetic resonance imaging scan after three months showed well demarcated multi loculated lesion increasing in size comparing to the last CT scan. The patient underwent laparoscopic distal pancreatectomy with splenectomy. The pathological analysis of the specimen showed a pancreatic hemangioma with no features of malignancy. The clinical presentation, radiological features and the modalities of diagnosis are here discussed.

Keywords: Endoscopic ultrasound, Hemangioma, Pancreatic cyst

INTRODUCTION

Pancreatic hemangioma is a rare cystic lesion of the pancreas. A few cases are reported in literature. The radiological features of pancreatic hemangioma overlap with other cystic lesions of the pancreas like mucinous cystadenoma and intrapapillary ductal mucinous neoplasm of the pancreas [1–4]. Thus, most of the cases of pancreatic hemangioma end up in surgical resection due to the uncertainty of the diagnosis. We discuss here the ways to avoid pancreatic resection of pancreatic hemangioma.

CASE REPORT

A 71-year-old female with a past medical history of thyroideectomy and diverticulosis presented to the emergency department with left iliac fossa pain. Computed tomography scan showed signs of diverticulosis without any complication and incidental finding of cystic lesion in the tail of the pancreas. The patient was transferred to our center for further follow-up and investigation.

Clinical examination on admission revealed healthy looking women, comfortable, abdomen soft and no abdominal masses palpable. Blood tests including lipase, CEA and CA 19-9 were normal. Computed tomography scan and magnetic resonance imaging (MRI) scan showed a 19-mm cystic multi loculated lesion in the tail of the pancreas which was initially thought to be a serious cystadenoma. We decided to follow-up the lesions with MRI scan in three months’ time because of atypical characteristics of the lesion. Magnetic resonance imaging
(MRI) at three months showed a 24 mm cystic loculated lesion (increasing in size comparing to the last CT scan), well demarcated with a thick and contrast-enhanced septa (Figure 1). No infiltration to the surrounding structure and no communication with the main pancreatic duct were described. Endoscopic ultrasound showed a 25-mm cystic lesion with same characteristics as on MRI scan and particularly did not find intramural nodule (Figure 2). For technical reason the puncture biopsy was not possible.

Since a diagnosis of pancreatic mucinous neoplasia could not be ruled out, a decision to perform pancreatic resection was made. Laparoscopic distal pancreatectomy with splenectomy was done. Postoperative course was uneventful and the patient was discharged without complications five days after surgery. The histopathological report revealed hemorrhagic cystic lesion measuring 2 cm, pathological features resembling pancreatic hemangioma without any features of malignancy (Figure 3).

**DISCUSSION**

Hemangioma is a vascular tumor composing of blood vessels lined by epithelial tissue. They can be found in various organs including brain, liver, kidney. Vascular tumors of the pancreas are very rare. Only few cases were reported in literature. They account for 1% of the visceral hemangioma and are mostly found in females. Until now there are 14 cases of pancreatic hemangioma reported in the literature. It is difficult to establish the diagnosis preoperatively, because of the rarity of the disease and the overlapping other cystic lesions of the pancreas. Usually, patients are strictly asymptomatic and abdominal imaging showed an incidental finding of pancreatic cystic lesion.
Rarely, they present with pancreatitis or abnormalities in the liver function test [1–7].

There are several radiological modalities to diagnose pancreatic hemangioma. Ultrasound is helpful to diagnose the pancreatic hemangioma especially large size lesions (> 5 cm) as reported in nine cases. In the ultrasound they look like cystic lesion, hyper echogenic comparing to the rest of the pancreas with no Doppler signal comparing to malignant lesion which is well vascularized. In the endoscopic ultrasound they appear as cystic mass with thick septations with no Doppler signal. Most of the reported cases share the same ultrasonographic features [1].

In computed tomography scan, hemangiomas are strongly contrast enhancing in the arterial phase, peripheral irregular enhancement with central non-enhancement in venous phase, and progressive filling-in during the delayed phases [5]. Pancreatic hemangiomas appear in the CT scan as well demarcated cystic lesion enhanced in the arterial phase with no communication with main pancreatic duct. The enhancement in the arterial phase is not found in all reported cases of pancreatic hemangioma. This is explained by the slow blood flow due to the presence of AV shunting. On MRI scan it appears as a lobulated, hypo-intense mass in T1-weighted images, and shows moderate hyperintensity signal in T2-weighted image [1, 2].

As we mentioned earlier, the features of pancreatic hemangioma can overlaps with other cystic lesions of the pancreas. For that in reviewing literature, only five of the reported cases were diagnosed preoperatively. The differential diagnosis for pancreatic hemangioma includes pancreatic pseudocyst, branch duct IPMN, serous cystadenoma or mucinous cystadenoma [6].

The role of the biopsy in the pancreatic hemangioma remains controversial. Endoscopic ultrasound guided FNA has been reported in some cases with no risk of bleeding but often non contributive results. Interest of ponction would be to eliminate diagnosis as IPMN or mucinous cystadenoma.

Concerning the treatment, pancreatic hemangioma can be observed when the diagnosis is certain. In reviewing the reported cases pancreatic hemangioma have been treated with surgical resection because of uncertainty of the diagnosis in 80% of cases.

CONCLUSION

Pancreatic hemangioma is a rare benign tumor. Current imaging techniques cannot reliably differentiate it from other neoplasm of the pancreas. Most of the cases end up in surgical resections due to uncertainty of the diagnosis.

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REFERENCES


Thoracic aortic aneurysm/dissection as an indication for family screening in younger patients: A case report

Javad Savoj, Heather Chen, Gregory Guldner, Rajesh Gulati

ABSTRACT

Introduction: Familial thoracic aortic aneurysm/dissection (TAAD) is a potentially lethal condition with a rising incidence which displays familial clustering in more than 20% of cases. Familial TAAD (FTAAD) refers to patients who have TAAD with a family history of aneurysmal disease who do not meet strict criteria for known connective tissue diseases. The FTAAD generally presents at an earlier age and has faster rate of aortic expansion. Case Report: A 45-year-old male was presented to the emergency department with acute neck and chest pain. Computed tomography detected an aortic root aneurysm and type one aortic dissection. His brother reported an aortic dissection at age 55 and his uncle succumbed to sudden death in his fifties. Conclusion: Family history may yield important clues to a catastrophic diagnosis for the acute care clinician and prompt primary care providers to discuss issues of possible screening during office visits. Most TAADs are sporadic and occur in association with atherosclerosis in older patients compared to familial TAAD, which has a mean age of 56.8. Up to 50% of susceptible relatives develop aortic dissection in FTAAD cases.

Keywords: Aortic aneurysm, Aortic dissection type one, Familial thoracic aortic aneurysm/dissection (TAAD), Familial thoracic aortic aneurysm/dissection (FTAAD)

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INTRODUCTION

Ascending thoracic aortic dissections typically present with severe chest pain and later the development of acute hemodynamic compromise. Early diagnosis and treatment is crucial for survival. The incidence of acute aortic dissection in the general population is estimated to range from 2.6 to 3.5 per 100,000 person-years [1–3]. In a review of 464 patients from the International Registry of Acute Aortic Dissection (IRAD), 65% of all types of aortic dissection were men, with a mean age of 63 years. Women presenting with aortic dissection were generally older than men with a mean of 67 years [4].

Etiologies differ between older adult patients and younger patients with dissections involving the ascending aorta. In an IRAD review, 32% of patients were ≥70 years of age and were significantly more likely to have atherosclerosis, prior aortic aneurysm,
iatrogenic dissection, or intramural hematoma. In a review of patients under age of 40 years, only 34% had a history of hypertension, and only 1% had a history of atherosclerosis. Marfan syndrome was present in 8.5% of younger patients (mean age 55 years), and was not diagnosed in any of the older adult patients [5, 6].

Familial thoracic aortic aneurysm/dissection (FTAAD) refers to patients who have thoracic aortic artery disease coupled with a family history of aneurysmal disease and who do not meet strict criteria for known connective tissue diseases. Patients with FTAAD generally present at an earlier age (56.8 years old) compared with patients with sporadic TAA (65.7 years old), and also have faster rate of aortic expansion [7].

CASE REPORT

A 45-year-old male presented to a community hospital emergency department with acute neck and chest pain. His pain first started as odynophagia and then extended to his chest and abdomen. He had no history of hypertension, coronary artery disease, diabetes mellitus, or smoking. His family history was significant for a type one aortic dissection in his brother three years earlier at age 55 years and a questionable history of the sudden death of his uncle while he was asleep. Computed tomography angiogram showed an aneurysmal dilation of the aortic root and an acute Stanford type one aortic dissection involving the ascending thoracic aorta, aortic arch, and descending thoracic aorta extending to the abdominal aorta (Figure 1–2). His dissection also involved the left brachiocephalic artery extending to the origin of the vertebral artery. Further investigations revealed that his brother had the same type of aortic dissection with extension to his left carotid artery and abdominal aorta, which had been treated with aortic valve replacement and aortoplasty. However, his brother had a history of smoking and diabetes, and his course had been complicated with stroke and right lower extremity compartment syndrome. The patient was transferred for aortoplasty, and after one year follow-up, did not have any complications. He and his brother did not meet any criteria of known connective tissue diseases such as Marfan or Loeys-Dietz syndrome.

DISCUSSION

Familial thoracic aortic aneurysm/dissection (FTAAD) presents at a younger age than sporadic patients, but at a significantly older age than patients with Marfan syndrome or Loeys-Dietz syndrome. Familial TAAD is primarily inherited in an autosomal dominant manner with decreased penetrance and variable expression. About 20% of patients with TAAD have a first-degree relative with a similar disease [8]. Mapping studies have firmly established that there is significant genetic heterogeneity for familial thoracic aortic aneurysm and dissection (i.e., many different genes can be mutated and cause the same clinical condition).

Our patient’s brother had a history of diabetes and atherosclerotic risk factors, which increase the chance of having sporadic TAAD. However, the age of onset was relatively lower than that for the average sporadic TAAD patient.

The international consensus guidelines recommend screening by aortic imaging for all first-degree relatives of patients with sporadic TAAD and all first- and second degree relatives in families with TAAD in which the genetic cause is not known [9]. However, these criteria would include a large proportion of the general population, which may not be cost effective. Furthermore, there are no strict guidelines on the age cut-off to commence screening and screening intervals, since there is no definitive age of onset in patients with familial TAAD and the rate of aortic expansion is faster in these patients. Genetic tests are expensive and not well established for clinical use. A negative genetic test for known mutations such as TGFBR2, ACTA2 and MYH11 does not rule out familial TAAD since each year new gene mutations are identified. Screening with thoracic
echocardiography is feasible, but it does not capture the entire aorta. Computed tomography scan or magnetic resonance angiography are the optimal imaging tests to detect TAAD, but computed tomography angiography exposes patients to radiation, and magnetic resonance angiography might not be cost effective for identifying patients with dilated aortas who should enter a follow-up program.

Currently, there is no ideal single screening test, particularly for younger individuals. Recent studies have shown ultra-low dose (ULD) chest CT scan can be used for detection of solid nodules, asbestos-related pleural diseases screening, and for monitoring of infectious pneumonia [10]. However, the sensitivity of ULD chest CT scan for screening of TAAD is still unclear. Limited one sequence magnetic resonance angiography can be another reasonable option that needs to be investigated as a screening method for TAAD.

CONCLUSION

Familial thoracic aortic aneurysm/dissection (FTAAD) is a catastrophic disease that is inherited mainly in an autosomal dominant mode. The average age of presentation is less than 60 years old based on previous studies. There are a few known mutations that are responsible for FTAAD. However, unknown mutations and modes of inheritance likely still exist. Acute care providers treating patients with symptoms possibly reflecting a thoracic aortic dissection may wish to inquire about family history of TAAD or sudden unexplained death if the initial evaluation has not made the need for definitive imaging clear. Primary care providers reviewing family medical histories should discuss screening options and controversies with patients who have had family members with TAAD. Although routine screening of patients’ family members with TAAD is recommended, there are no established guidelines for modality of screening, age cut-off for screening, and frequency of follow up of these patients. Our experience suggests that first degree families of patients with known risk factors of sporadic aortic aneurysm and dissection who are relatively younger than 60 should be screened for familial TAAD. Further studies are required to establish a cost effective and clinically feasible screening method based on patient age and family history.

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REFERENCES


A forgotten double-J stent with missing shaft and unusual large stone formation at its both the J end: A case report

Rajesh Kumar Maurya, Vikash Katiar, Vijay Kannaujiya

ABSTRACT

Introduction: Use of ureteric stents is accepted standard practice in the management of ureteric obstruction, ‘forgotten’ indwelling stents can cause encrustation, pyelonephritis, recurrent obstruction, and stent migration and breakage. A stent register should be maintained to check follow-up of such patient to prevent this urological travesty. Case Report: We are reporting a case of encrustation and unusual large stones formation at both the J end of a forgotten double-J stent in a 35-year-old female presented in our outpatient department for complaints of left flank and lower abdominal pain, burning and increased frequency of micturition for four months. Conclusion: Indwelling stents can result in complications such as encrustation, pyelonephritis, recurrent obstruction, and stent migration and breakage so their use should be done with caution.

Keywords: Bladder stone, Forgotten Stent, Indwelling Stent, Stenturia, Staghorn calculus, PCNL

INTRODUCTION

The double-J ureteric stent has become one of the most basic and valuable tool in urology [1]. The use of ureteric stents is accepted standard practice in the management of ureteric obstruction [2]. ‘Forgotten’ indwelling stents can result in complications such as encrustation, pyelonephritis, recurrent obstruction, and stent migration and breakage [3, 4] and hence can result in significant morbidity and financial loss to the patients. We herein report a case of encrustation and large stones formation at both the J end of a forgotten double-J stent.

CASE REPORT

A 35-year-old female presented in our outpatient department for complaints of left flank and lower abdominal pain, burning and increased frequency of micturition for four months. Six years back she had undergone left sided ureteroscopy at some other centre for ureteric stone clearance and after the procedure a double-J stent was placed. Patient did not turn up for stent removal. Two years back she also passed fragments of stent in the urine (stenturia).
Ultrasound kidney, ureter, and bladder of the patient revealed essentially right sided normal kidney, while in left kidney there was a staghorn calculus of size about 25 mm, besides it there was a large bladder stone of size about 49 mm. Digital X-ray kidney, ureter, and bladder showed coiled ends of a double-J stent in the left renal pelvis and bladder region with formation of stones over it however entire shaft of the stent was missing (Figure 1). Her KFT was within the normal limit.

Patient was managed by antibiotics according to urine culture sensitivity and by open cystolithotomy for bladder stone (Figure 2), and after one month of open cystolithotomy she underwent PCNL for the staghorn calculus, her postoperative recovery was uneventful both the time. Her latest digital X-ray KUB (Figure 3) dated 01/06/2017 showing complete clearance of stone and double-J stent in situ.

**DISCUSSION**

It has became a routine practice in urology to use a double-J stents for various indications, double-J stent was introduced in the year 1978 since then many improvements have been made in its design and composition to minimize patient discomfort, because of it patient can forget regarding the indwelling stent. The rate of complications like fragmentation, encrustation, pyelonephritis, recurrent obstruction and stent migration are significantly related to indwelling time.

El-Faqih et al. reported encrustation rate of 9.2%, 47.5% and 76.3% for indwelling time of less than six weeks, 6–12 weeks and more than 12 weeks respectively, they emphasized need for maintaining computerized stent register [3].

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**Figure 1**: Digital X-ray kidney, ureter, and bladder of the patient showing A large bladder stone and a large staghorn calculus (in left renal pelvis) formed over J ends of double-J stent while central shaft has been disappeared due to stenturia.

**Figure 2**: Bladder stone after its removal from urinary bladder.

**Figure 3**: Digital X-ray kidney, ureter, and bladder of patient showing complete clearance of stone with DJ stent insitu.
Monga et al. concluded that management of such complicated ureteral stents requires a multimodal therapeutic approach, a computerized tracking registry of ureteral stents may help prevent this urological travesty [4].

Kawahara et al. reported similar results, they also concluded that although ureteral stent encrustation was related to the indwelling time, heavily encrusted ureteral stents necessitating additional procedures for removal occurred within an indwelling time of 3 months, the exact interval for removal of an indwelling ureteral stent to avoid additional procedures for removal is therefore difficult to determine [5].

Spontaneous fracture of indwelling ureteral stents can be prevented by careful examination of the stents prior to insertion, by following the instructions of manufacturers on maximum time limits [6]. Successful management of retained ureteral stents requires careful planning and may entail a combination of endourologic approaches [7].

Tunney et al. reported that Silicone was least prone to struvite encrustation, followed by polyurethane, silitek, percutflex and hydrogel-coated polyurethane, in rank order. Similarly, silicone was least prone to hydroxyapatite encrustation, followed by silitek, polyurethane, percutflex and hydrogel-coated polyurethane [8].

Kumar et al. showed that mean indwelling time for spontaneous stent fragmentation was 3.5 months only [9]. In case of our patient indwelling time was six years, so we conclude that due to encrustation and stone formation, both ends of the stent were retained in situ and the central shaft was fragmented and passed along with urine (stenturia) and disappeared [10–13].

**CONCLUSION**

In a nutshell, we conclude that although double J stent is an essential tool in quiver of an urologist, their use should be done with caution, patient should be well informed regarding their benefit and harm, A stent register should be maintained to check follow up of such patient, and if there is need of prolong stenting it should be changed after every 12 weeks.

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Rajesh Kumar Maurya – Substantial contributions to conception and design, Revising it critically for important intellectual content, Final approval of the version to be published

Vikash Katiar – Analysis and interpretation of data, Revising it critically for important intellectual content, Final approval of the version to be published

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**REFERENCES**

Tubercular thyroid abscess: A case report

Bhavinder Arora

ABSTRACT

Introduction: Thyroid gland tuberculosis is a very rare extrapulmonary presentation of tuberculosis even in countries where pulmonary tuberculosis is endemic. Thyroid gland tuberculosis was reported in 19th century. Thyroid gland tuberculosis presenting as abscess is very rare with occasional case reports in literature. A case of thyroid gland abscess is presented here. Case Report: A young girl presented with swelling in right lobe of thyroid. Ultrasound (USG) revealed a swelling in right lobe of thyroid with central necrosis. Contrast-enhanced computed tomography scan (CECT) and magnetic resonance imaging (MRI) scan of neck could not make a definite diagnosis. Fine needle aspiration (FNA) from the swelling could provide the diagnosis of tubercular thyroid abscess. Pus aspirated from the swelling did not reveal any acid fast bacilli. The patient was treated by antitubercular therapy, thus avoiding surgery of thyroid. Conclusion: The accurate diagnosis of tubercular thyroid abscess is essential by using USG, CECT, MRI scan and FNA. The surgery of thyroid gland has been limited, because of availability of antitubercular therapy.

Keywords: Abscess, Antitubercular therapy, Fine needle aspiration, Thyroid, Tuberculosis

INTRODUCTION

Tuberculosis of thyroid is a rare manifestation of extrapulmonary tuberculosis. The incidence is very low even in countries where pulmonary tuberculosis is endemic [1]. The incidence of this disease varies from 0.1–1.15% [1, 2]. This low incidence of thyroid tuberculosis is attributed high vascularity of thyroid gland and its ability to resist infection [2]. The thyroid gland gets infected by hematogenous and lymphogenous route or direct spread from tubercular cervical lymph nodes [3]. The clinical presentation of tubercular infection of thyroid can be chronic or cold abscess, subacute thyroiditis and rarely as acute abscess. However, the most common manifestation is cold or caseous abscess and a solitary thyroid nodule. Tubercular thyroid can be confused malignant thyroid [4]. The accurate diagnosis needs help of radiological investigations besides elaborate clinical history and examination [5]. Tissue diagnosis either by fine needle aspiration or histopathology is essential [6]. Tubercular thyroid abscess are occasionally available in medical literature. One such case report of tubercular abscess has been presented here.
CASE REPORT

A 16-year-old girl presented with swelling in the right lobe of thyroid. There were no generalized symptoms like fever, malaise, night sweats and weight loss. There was no history of difficulty in deglutition or voice change. On clinical examination there was a single abscess of size 4x4 cm in right lobe of thyroid moving with deglutition. The margins were well demarcated, smooth surface, non-tender and overlying skin was normal with a tattoo mark on it (Figure 1). There were no clinical features of hypothyroidism or hyperthyroidism. Routine blood investigations were done; hemoglobin 11.0 g/dl, total leucocytes count 8600/mm³, neutrophils 67/mm³, lymphocytes 31/mm³ and eosinophils 2/mm³. The erythrocyte sedimentation rate was 20 mm. The Mountax test was highly positive more than 10 mm in diameter. Thyroid function tests T3, T4, TSH were normal. X-ray chest was normal. Ultrasonography of neck revealed a 50x45x20 mm solitary nodule in the right lobe of liver. This solitary thyroid nodule was showing thick irregular wall with central necrosis reported as suspected thyroid abscess. Magnetic resonance imaging scan of the neck showed a lesion of intermediate signal intensity due to presence of dense inflammatory cells and granulomas with central necrosis (Figure 2). A doubt about carcinoma of thyroid was placed as differential diagnosis. The CECT scan of neck was done to rule out carcinoma of thyroid gland. This was helpful in diagnosis of tubercular thyroid abscess as localized caseous lesion in right lobe of thyroid (Figure 3). Fine needle aspiration from this solitary thyroid nodule was done to confirm the diagnosis. The stained smears revealed degenerated and intact neutrophils, and macrophages in serofibrinous background. A few epithelioid granuloma and multinucleated giant cells are also seen suggestive of tuberculosis with central caseous necrosis. Ziehl–Neelsen staining with 20% H₂SO₄ was noncontributory (Figure 4). From the central part of swelling about 2 ml of thick yellow color pus was aspirated as shown in Figure 5. The smears prepared from this pus did not show any acid-fast bacilli. After aspiration the swelling decreased in size. The cytological diagnosis of tubercular abscess was made. The patient was put on antitubercular treatment with four drug regimens. The swelling decreased in size in next three months (Figure 6). She was asked to continue on three drug regimens for another six months leading to complete resolution of swelling.

DISCUSSION

Tuberculosis of thyroid is rare diagnosis reported since early 19th century [7]. It is a rare extrapulmonary manifestation of tuberculosis, the true incidence of tuberculosis of thyroid is unknown. This rare involvement of thyroid gland to tubercular infection is attributed to high vascularity of thyroid gland and bactericidal property of colloid material [8]. Thyroid tuberculosis may be primary or secondary concurrent with pulmonary tuberculosis. The primary involvement of thyroid without pulmonary involvement is extremely rare [9].

There are two routes of infection by which thyroid gland get infected; generalized dissemination by hematogenous route as in miliary tuberculosis and a focal spread to thyroid gland. Focal spread may be primary of thyroid gland called primary tuberculosis of thyroid gland [10]. However, it may be secondary from adjacent lymph node [11]. Occasionally, there may be lymphogenous spread.

The clinical presentation of tubercular thyroid abscess is generally as a solitary thyroid nodule or a cold abscess rarely presenting as acute abscess [12, 13]. Solid thyroid nodule can mimic clinically as thyroid carcinoma [14].

Figure 1: Thyroid abscess right lobe.

Figure 2: Magnetic resonance imaging scan showing central necrosis in right thyroid lobe swelling.
Presenting symptoms in tubercular thyroid abscess are variable. Most of the patients present as solitary thyroid nodule with no sign of acute inflammation. All main diseases of thyroid gland should be considered including carcinoma thyroid [15]. The accurate diagnosis of tubercular thyroid has to be made by using investigation. The ultrasound is the basic investigation for a solitary thyroid nodule. Radiological imaging techniques of MRI scan and CT scan make the diagnosis of thyroid tuberculosis and also rule out malignancy of thyroid [16]. However, confirmation of diagnosis can only be done using tissue diagnostic technique of fine needle aspiration cytology [17]. The cytological diagnosis can be made by presence of tubercular granuloma. Acid-fast bacilli staining may not detect tubercular bacilli [18]. In such cases PCR can be done [19]. The confirmatory diagnosis of tuberculosis of thyroid by fine needle aspiration cytology can avoid unnecessary thyroid surgery for histopathological confirmation.

Antitubercular therapy is the preferred method of treatment of tuberculosis of thyroid nowadays [20]. If thick pus is present in central part it can be aspirated using thick needle. Repeated aspiration may be necessary.
A few cases in which pus cannot be aspirated open drainage may be necessary. Repeated needle drainage and antitubercular drug therapy is the treatment of choice as being the least invasive method [21]. However, very large thyroid abscess may need open drainage and excision is required rarely. Those patients who do not respond to antitubercular therapy in three months duration may need surgical excision of thyroid nodule or hemithyroidectomy [22].

CONCLUSION

Tubercular thyroid abscess is a rare clinical diagnosis as pulmonary tuberculosis may not be associated with pulmonary tuberculosis in most of these patients. Tubercular thyroid abscess can be diagnosed only with a very high degree of clinical suspicion. Imaging techniques like magnetic resonance imaging scan and computed tomography scan are useful in making the diagnosis of tubercular thyroid abscess. Definitive diagnosis can be made by cytological examination by presence of tubercular granuloma. Tubercular thyroid abscess can be treated by aspiration of pus followed by antitubercular treatment thus avoiding surgery of thyroid.

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Author Contribution
Bhavinder Arora – Substantial contributions to conception and design, Acquisition of data, Analysis and interpretation of data, Drafting the article, Revising it critically for important intellectual content, Final approval of the version to be published

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REFERENCES

Inflammatory paradental cyst on the distobuccal aspect of an impacted mandibular third molar: A case report


ABSTRACT

Introduction: The paradental cyst is an inflammatory odontogenic cyst usually associated with distal and buccal aspect of partially impacted mandibular third molars. An associated history of pericoronitis, as a consequence of an inflammatory process in the periodontal pocket has been suggested as the route of inflammation. The pathogenesis of these cysts is most likely to be originated from the inflammatory proliferation of epithelial rests of Malassez. This cyst has been under reported due to the lack of sufficient clinical information to establish the diagnosis and many may have been misdiagnosed as dentigerous cysts, pericoronitis, lateral radicular cysts or inflamed dental follicles. Case Report: We present a case of a 36-year-old male presented to the department of oral and maxillofacial surgery with a complaint of discomfort seen in relation to distal aspect of mandibular third molar for the last six months. The presence of swelling in the buccal and distal aspect, radiolucrency seen distally of mandibular third molar with intact lamina dura and unwidened periodontal ligament space, positive response to vitality tests and classic histopathologic findings confirmed the diagnosis of paradental cyst. Conclusion: A proper insight or knowledge into the clinicoradiographical presentation and histological findings would help us to differentiate the inflammatory paradental cyst from other odontogenic cysts.

Keywords: Epithelial rests, Odontogenic, Paradental cyst, Pericoronitis

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INTRODUCTION

Hofrath, in 1930, was the first author to report on several cases of jaw cysts located distally to third mandibular molar with pericoronitis [1]. Main, in 1970, understanding the inflammatory nature of these lesions, put forward the term ‘inflammatory collateral...
cyst’ [1]. Based on Hofrath’s description of the clinical, radiological and histological features of these cysts, the lesions were later termed paradental cysts by Craig in 1976 [2]. He described it as a cyst of inflammatory origin, occurring on the lateral aspect of the roots of partially erupted mandibular third molars, where there was an associated history of pericoronitis, as a consequence of an inflammatory process in the periodontal pocket [3]. The term ‘inflammatory paradental cyst’ was suggested by Vedtofte and Praetorius, because of its inflammatory origin and also due to its location at the side of the tooth [4]. Stoneman and Worth, in 1983 described a lesion that was similar to paradental cyst but occurred primarily in relation to mandibular first and second molar. This entity was called mandibular infected buccal cyst to emphasis its origin in inflamed periodontal tissues of partially or fully erupted molars [5].

According to Craig, paradental cysts constituted 4.7% of 1051 odontogenic cysts [3]. Philipsen et al. reported frequencies ranging between 0.9 and 4.7% of odontogenic cysts [5]. The clinical manifestation of the paradental cyst usually presents with a history of recurrent inflammatory periodontal process or pericoronitis. Presentation of few signs and mild symptoms like discomfort, tenderness, moderate pain and in some cases, suppuration through the periodontal sulcus is seen. Paradental cysts are present commonly on buccal or distal aspects and rarely on mesial aspect of partially or fully erupted vital teeth [2, 6]. These are commonly involved with mandibular third molars. According to their review of literature, Philipsen et al. reported that the mean age of occurrence for inflammatory paradental cysts, was in the third decade [5]. Craig and Philipsen recorded most lesions in males; with a male to female ratio as 1:0.4. Radiographically, the lesions are usually superimposed on buccal root surface as well demarcated radiolucencies. Corticated margin with intact lamina dura is seen around the roots along with absence of widening of periodontal ligament space [3, 5].

Keeping in mind the various clinical variations, the present article aims to discuss the differential diagnosis and its various aspects by presenting a case report to illustrate the findings.

**CASE REPORT**

A 36-year-old male patient reported to the department of oral and maxillofacial surgery with a discomfort seen in relation to the distal aspect of impacted left mandibular third molar for the last six months. Clinically, the patient was asymptomatic, reported no paresthesia and showed no suppuration in the affected site. On extraoral examination, a hardened increase in volume could be observed in the mandibular body and ramus area by palpation. Intraoral examination revealed a partially impacted left mandibular third molar with pericoronitis distal to it. Panoramic radiographic examination revealed a radiolucent lesion on the distal aspect of impacted mandibular third molar of size approximately 2 cm in diameter (Figure 1). The periodontal ligament space and the lamina dura were intact and continuous around the root.

Based on the clinical and radiographic findings, a differential diagnosis of developmental odontogenic cyst, odontogenic keratocyst and periapical cyst were suspected. Due to the presence of radiolucency on the distal aspect of tooth 38, which appeared in the panoramic radiograph, a dentigerous cyst was suspected. Fine needle aspiration revealed blood tinged fluid. Tooth extraction along with the enucleation of the cystic lesion was performed under local anaesthesia. The cystic lesion was excisioned. On grossing and macroscopic examination, tooth specimen with associated soft lesional tissue as well as the excised lesional tissue measuring 2.2x1.7x1 cm and 1.5x0.9x0.6 cm respectively was observed (Figure 2).

![Figure 1: Presence of radioluency distal to 38 with intact lamina dura and unwidened periodontal ligament space.](image1.png)

![Figure 2: Gross specimen image showing mandibular third molar tooth with associated lesional tissue.](image2.png)
Microscopic examination revealed a hyperplastic non-keratinized stratified squamous epithelium of varying thickness (Figure 3). Epithelium exhibited proliferation in an arcading pattern. An intense inflammatory cell infiltrate was observed in the adjacent fibrovascular connective tissue capsule (Figure 4). The connective tissue was moderately collagenous with diffuse dense chronic inflammatory cells, predominantly lymphocytes and plasma cells. Numerous endothelium lined blood vessels, extravasated RBCs, calcifications and trabeculae of bone were also seen in the connective tissue. The findings were compatible with the diagnosis of inflammatory paradental cyst.

DISCUSSION

In 1992, the World Health Organization (WHO) included the paradental cyst for the first time in the histologic typing of odontogenic tumors [5, 6]. Since the prevalence of paradental cyst varies between 1–5% in all odontogenic cysts, the paradental cyst has been included in the group of rare lesions [7]. It is believed that paradental cysts are under reported and due to the lack of sufficient clinical information to establish the diagnosis, many may have been misdiagnosed as dentigerous cysts, pericoronitis, lateral radicular cysts or inflamed dental follicles [2].

Most often lesions are located in a buccal or distobuccal location and cover the root surface, frequently involving the bifurcation. According to Colgan et al., the actual site of the lesion may depend on angle of impaction of the associated tooth [5]. Cysts were located on the mesial aspect of mesioangular impacted tooth, buccal to vertical impactions, distal or distobuccal to distoangularly impacted teeth. In Craig's series of 49 cases, 26 cysts were located on buccal aspect of roots, 19 were distal and four were mesial [3]. In this case, the cyst was also located on the distobuccal aspect of impacted mandibular third molar.

Regarding the pathogenesis, Craig (1976) believed that either the reduced enamel epithelium or the cell rests of Malassez could be the key to the formation of paradental cysts. Craig preferred reduced enamel epithelium as the cells of origin because, in his study, the rests of Malassez always seemed inactive and the cyst should be uniformly distributed around the root surface if the development of the cyst were to be from the rests of Malassez [3]. Craig also came up with an interesting finding, in 20 of 28 cases, where the associated tooth was available for study, that the removal of cyst from the buccal root surface unveiled a developmental enamel projection extending from the amelocemental junction towards the root bifurcation. Many authors have suggested the presence of this small enamel projection, within the bifurcation area of the roots on the buccal aspect of teeth, as part of the etiology of paradental cysts [2, 3]. Craig attributed the presence of an extension of reduced enamel epithelium over these enamel projections, to be the cause for the frequent buccal location of the cyst [3].

Many cystic lesions were included under the differential diagnosis. The possibility of a lateral radicular cyst was not considered as the tooth appeared vital [5]. Lack of superimposition of the lesion on the roots and an intact periodontal ligament space ruled out the possibility of a periapical pathology [8]. Lateral periodontal cysts are present in a much older age group and is usually located in the mandibular canine-premolar region [9].

The histological features exhibited by odontogenic keratocysts and unicystic ameloblastoma are usually different from paradental cysts. In this case, lack of appearance of mucoid changes in the connective tissue or remnants of odontogenic epithelium in the walls of the
cyst ruled out the possibility of dentigerous cyst or dental follicular cyst [9]. Colgan’s sign (preservation of a distal follicular space which indicates that most of the follicle is not implicated in the cyst development process) helps to distinguish between dentigerous and paradental cysts radiographically [2, 5].

In the current case, the association of the cyst with a mandibular molar, buccal and distal bony cavitation and positive response to vitality tests suggested a paradental cyst. This was also confirmed by the classic histopathological examination [2, 7, 8, 10]. Although these lesions present with the same etiology and histologic features as that of mandibular infected buccal cyst (first and second molar) and juvenile paradental cyst (involving first and second molars of younger age group), differences in the teeth involved and the differences in the ages of the individuals may well dictate the necessary treatment [1, 2, 5]. The surgical removal of teeth and cyst is considered the treatment of choice when the involved tooth is a third molar [9]. When only first or second molars are involved; enucleation of the cyst without the removal of associated tooth is suggested [2].

CONCLUSION

The inflammatory paradental cyst is a relatively rare underdiagnosed lesion frequently associated with the distal and buccal aspects of mandibular molars. Since the histological features of paradental cysts mimics other inflammatory odontogenic cysts, correlations of the incorporated findings of surgical, radiographic and histologic findings are needed to obtain a definitive diagnosis of paradental cyst.

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ABSTRACT

Introduction: Acute appendicitis is a common surgical emergency that requires intervention. The accurate diagnosis remains challenging in some cases despite advances in both minimally invasive surgery and radiology. Stump appendicitis is a rare complication after appendectomy. It is defined as the acute inflammation of the residual appendix. A small number of stump appendicitis cases have been reported. Case Report: We report a case of stump appendicitis in a 42-year-old female, nine months following a laparoscopic appendectomy. She presented with a 24-hour history of abdominal pain, which started periumbilically and then localized to the right lower quadrant. Physical examination showed tenderness in the right iliac fossa with evidence of rebound and guarding. Laboratory studies were remarkable for leukocytosis. Computed tomography scan of the abdomen and pelvis showed a remnant appendicular segment with a maximum cross diameter of about 1.2 cm, associated with local inflammatory changes and surrounding fat stranding. An open stump appendectomy was performed uneventfully. Conclusion: Stump appendicitis is a rare but serious complication of appendectomy. It can represent a diagnostic dilemma if the treating clinician is unfamiliar with this rare clinical entity. Prompt recognition is important to avoid serious complications. Proper identification of the appendicular base intraoperatively and leaving the appendix stump shorter than 5 mm decrease the risk of stump appendicitis.

Keywords: Acute appendicitis, Completion appendectomy, Stump appendicitis

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INTRODUCTION

Acute appendicitis is a common surgical emergency that requires intervention. The lifetime risk of developing appendicitis is about 7% [1]. The accurate diagnosis of appendicitis remains challenging in some cases despite advances in both minimally invasive surgery and radiology [2]. One rare complication after appendectomy is stump appendicitis, which is defined as the acute inflammation of the residual appendix [3]. Although the signs and symptoms do not differ from those of acute appendicitis, the diagnosis is often not considered because of the history of previous appendectomy [4]. A small number of stump appendicitis cases have been reported [5]. We report a 42-year-old female with preoperatively diagnosed stump appendicitis by computed tomography scan, who underwent a laparoscopic appendectomy nine months ago.
CASE REPORT

A 42-year-old female presented to Mubarak Al-Kabeer hospital on 13/4/2017 with a 24-hour history of abdominal pain. The pain started periumbilically and then localized to the right lower quadrant. It was associated with nausea and episodes of chills and rigors. Her medical history was noncontributory, but surgical history was notable for a laparoscopic appendectomy that was performed nine months earlier at the same hospital.

On admission, the patient was afebrile and her vital signs were otherwise normal. Physical examination revealed tenderness in the right iliac fossa with evidence of rebound and guarding. Routine laboratory studies were remarkable for a white blood cell count of 16x10^9/L with 84% neutrophils. Urinalysis was negative.

Computed tomography (CT) scan of the abdomen and pelvis was performed with rectal and intravenous contrast, which showed a remnant appendicular segment at the base with a maximum cross diameter of about 1.2 cm (Figure 1). It also showed local inflammatory changes and surrounding fat stranding (Figure 2). A preoperative diagnosis of stump appendicitis was made on the basis of the CT study.

Surgical exploration performed after completion of the CT scan showed a 1–2 cm long inflamed appendiceal stump. An open stump appendectomy was performed uneventfully. Stump appendicitis was also confirmed on gross pathologic and histologic examination of the resected specimen. No evidence of gross perforation was present. The postoperative course was uneventful, and the patient was discharged 72 hours later.

DISCUSSION

Appendectomy is one of the most commonly performed emergent surgical procedures. The first appendectomy was performed by Claudius Amyand in 1735. The clinical features and pathological abnormalities of appendicitis were described by Reginald Fitz in 1886. In 1945, Rose was the first to describe stump appendicitis in two patients who had undergone appendectomy for acute appendicitis [6].

The appendix arises from the postero-medial wall of the cecum about 3 cm below the ileocecal valve. The base of the appendix can be misidentified intraoperatively. The variable position and subserous length of the appendix, combined with acute inflammation, may result in this misidentification. Following the teniae coli on the cecum helps in identifying the true appendicular base. Generally, an appendix stump shorter than 5 mm is associated with a lower risk of stump appendicitis [7, 8].

Stump appendicitis can represent a diagnostic dilemma if the treating physician is unfamiliar with this rare clinical entity. Patients present with signs and symptoms of appendicitis or acute abdomen along with a history of previous appendectomy. The presence of an appendectomy scar does not rule out the possibility of stump appendicitis [9]. Prompt recognition is important to avoid serious complications like perforation and peritonitis [8].

Radiological evaluation by ultrasound and CT scan helps in the preoperative diagnosis of stump appendicitis.
Computed tomography scan of the abdomen is more specific than ultrasound for the accurate preoperative diagnosis of stump appendicitis because it excludes other causes of acute abdomen. Computed tomography findings include pericecal inflammatory changes, abscess formation, fluid in the right paracolic gutter and cecal wall thickening [7]. Completion appendectomy either by open or laparoscopic technique is the treatment of choice for stump appendicitis [11].

CONCLUSION

Stump appendicitis is a rare but serious complication of appendectomy. Patients present with signs and symptoms of appendicitis or acute abdomen along with a history of previous appendectomy. The diagnosis can be missed or delayed if the physician is unaware of this rare clinical entity. Prompt recognition is important to avoid serious complications. Proper identification of the appendicular base intraoperatively and leaving the appendix stump shorter than 5 mm decrease the risk of stump appendicitis.

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REFERENCES
Superior pancreaticoduodenal artery pseudoaneurysm complicated with fistulation into common bile duct and duodenum: A case report

Samarasinghe A.S., Chathuranga L.S., Niyas S.M.M.

ABSTRACT

Introduction: Visceral artery aneurysms are a rare clinical entity with life-threatening complications such as rupture, associated with high mortality. Case Report: A 50-year-old male presented with a five weeks history of persistent epigastric pain associated with indigestion, weight loss and melena. Ultrasonography showed a mass in the region of pancreatic head suggestive of a pseudoaneurysm. Computed tomography scan confirmed a large pseudoaneurysm supplied by superior pancreaticoduodenal artery exerting direct pressure over the head of the pancreas and second part of the duodenum. An explorative laparotomy performed and aneurysmal dilatation of superior pancreaticoduodenal artery complicated with fistulation into common bile duct and duodenum identified and repaired. Conclusion: High index of suspicion and timely intervention helps in optimal outcome.

Keywords: Duodenum, Fistulation, Pseudoaneurysm, Superior pancreaticoduodenal artery

INTRODUCTION

True and false aneurysms of the visceral arteries form an important entity of vascular pathology though it is rare. Visceral artery aneurysms (VAAs) represent 0.1–0.2% of all vascular aneurysms and most commonly found in splenic (60%), hepatic (20%) and superior mesenteric (9%) arteries [1–3]. Pancreaticoduodenal artery aneurysms (PDAA) are rare, accounting for only 2% of all splanchnic artery aneurysms, but it is an important vascular disease because of its potential for fatal rupture [4]. False pancreaticoduodenal artery aneurysms occurs due to surrounding inflammatory response in cases like pancreatitis, abdominal trauma, septic emboli or laparoscopic cholecystectomy. They often rupture into the gastrointestinal tract, whereas true aneurysms are usually associated with coeliac axis stenosis, and rupture into the retroperitoneal space [3, 4]. We present a case with pseudoaneurysm of superior pancreaticoduodenal artery fistulized into the duodenum.
CASE REPORT

A 50-year-old male was presented to our hospital with a five-week history of persistent epigastric pain associated with indigestion, weight loss, and melena. On physical examination, he was icteric and had an epigastric tenderness but no palpable masses detected. Biochemical investigations revealed obstructive jaundice with elevated total (135.8 μmol/l) and direct bilirubin levels (114.2 μmol/l). Abdominal ultrasonography reported an aneurysmal dilatation (5.1x5.8x4.8 cm) in the epigastric region with wall thickness of 1.4 cm. Upper gastrointestinal endoscopy demonstrated an ulceration of mucosa of the second part of the duodenum with active bleeding (Figure 1).

Contrast enhanced computed tomography scan of the abdomen showed a pseudoaneurysm in the superior pancreaticoduodenal artery. Superiorly there was a linear high density within the hypodense wall which could be contrast leak into intramural thrombus. Pancreatic head is compressed and there are multiple foci of coarse calcifications within the head of the pancreas. Pancreatic duct is dilated (9.1 mm). Second part of the duodenum is compressed by the lesion. Gallbladder is distended and common bile duct is dilated (1.3 cm). Hepatic artery originates directly from the aorta.

An explorative laparotomy performed and aneurysmal dilatation of the superior pancreaticoduodenal artery identified. Aneurysm sac opened and feeding artery ligated from inside. Two fistulous openings into second part of the duodenum and to the common bile duct identified and repaired (Figure 2 and Figure 3). The patient was discharged one week later after complete recovery.

DISCUSSION

Aneurysm of the pancreaticoduodenal artery is rare accounting for about 2% of all splanchnic aneurysms [4]. Clinical manifestations depend on the type of aneurysm. True aneurysms are associated with coeliac axis stenosis, while pseudoaneurysms are known to rupture into gastrointestinal tract as in this case [4, 5]. Long standing alcohol use favors chronic pancreatitis as being the underlying cause. Aneurysm had exerted sufficient pressure to cause both common bile duct and pancreatic duct dilatation. Acute hemorrhage is a life-threatening complication of pseudoaneurysm, with a high mortality rate of up to 37% [6].

Brocker et al. reviewed 93 cases of PDAAs with celiac stenosis or occlusion and reported both that 52% were ruptured at the time of presentation and that aneurysm size did not correlate with rupture [7]. These results emphasize that PDAAs should be treated at the time of diagnosis. Surgical (e.g., ligation, resection, or pancreaticoduodenectomy) or endovascular embolization are the two main treatment modalities practiced. Surgical correction of visceral aneurysm considered to be the gold standard [8, 9] while endoluminal procedures have now become the first line of therapy instead of surgery in most instances, especially in an emergency setting.

CONCLUSION

We report a rare case of a patient with pancreaticoduodenal artery aneurysm complicated with fistulation into common bile duct and duodenum. High index of suspicion and timely intervention helps in optimal outcome.
Author Contributions
Samarasinghe A.S. – Substantial contributions to conception and design, Acquisition of data, or analysis and interpretation of data, Drafting the article, Final approval of version to be published
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REFERENCES
Neurofibroma of the cervical part of the vagus nerve: A case report

Samarasinghe A.S., Chathuranga L.S., Niyas S.M.M., Sugathadasa W.D.P.

ABSTRACT

Introduction: Neurofibroma of the vagus nerve in the cervical region is an extremely uncommon benign tumor. Only ten reported cases were found in literature. It is slow growing and most are asymptomatic neck lumps. Preoperative imaging aids in arriving at a differential diagnosis and planning the surgery. Complete surgical excision is the standard of care. Case Report: A 39-year-old female presented to the general surgical clinic with a painless lump on the right side of the neck for twelve months duration with recent rapid enlargement. Past medical history was unremarkable. She did not have a family history of neurofibromatosis. Physical examination revealed a 4x3.5 cm lump in the upper neck with well-defined margins and smooth surface. Contrast-enhanced CT scan confirmed a 3.6x3.2x6.9 cm hypodense mass with no contrast enhancement. The lesion was in-between right internal and external carotid artery and extending to the base of the skull. Right internal jugular vein was displaced anteriorly. Right common carotid artery (R/CCA) was stretched. The patient underwent exploration of the neck under general anesthesia with loupe magnification. The gross pathology showed a greyish homogenous cut surface of a 55x40 mm tumor. Pathological examination confirmed the diagnosis of neurofibroma of the vagus nerve. Conclusion: Vagus nerve neurofibromas are rare nerve sheath tumors. Gold-standard treatment of symptomatic benign vagus nerve mass is an attempt at GTR with minimal loss of nerve function.

Keywords: Neurofibroma, Tumor, Vagus nerve

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INTRODUCTION

Neurofibroma of the vagus nerve in the cervical region is an extremely uncommon benign tumor [1]. Only ten reported cases were found in literature [2]. It is slow growing and most are asymptomatic neck lumps [3]. Preoperative imaging aids in arriving at a differential diagnosis and planning the surgery. Complete surgical excision is the standard of care. Here we present a case of sporadic neurofibroma of the vagus nerve in the cervical region of a 39-year-old lady. She underwent successful surgery without disability such as vocal cord palsy.
CASE REPORT

A 39-year-old female presented to the general surgical clinic with a painless lump on the right side of the neck for twelve months duration with recent rapid enlargement. Past medical history was unremarkable. She did not have a family history of neurofibromatosis. Physical examination revealed a 4x3.5 cm lump in the upper neck with well-defined margins and smooth surface. Upon palpation paroxysmal cough was not elicited.

Ultrasound of the neck showed a well-defined solid soft tissue mass with internal vascularity. Contrast enhanced computed tomography scan confirmed a 3.6x3.2x6.9 cm hypodense mass with no contrast enhancement. The lesion was in-between right internal and external carotid artery and extending to the base of the skull. Right internal jugular vein was displaced anteriorly. Right common carotid artery (R/CCA) was stretched (Figure 1).

The fine needle aspiration cytology (FNAC) of the mass was inconclusive showing only stromal tissue fragments. The patient underwent exploration of the neck under general anesthesia with loupe magnification. A vertical incision is made along the anterior border of the sternocleidomastoid muscle achieving vascular control. An ovoid whitish tumor was identified in between the right internal jugular vein and the carotid artery displacing them. Both the superior and inferior ends of the mass appeared in continuity with the vagus nerve stretching its nerve fibers. Plane of the tumor was approached splitting the nerve fibers in the longitudinal direction. Tumor was completely excised preserving the continuity of the vagus nerve (Figure 2).

The gross pathology showed a greyish homogenous cut surface of a 55x50 mm tumor. Pathological examination confirmed the diagnosis of neurofibroma of the vagus nerve. Microscopy showed loosely arranged spindle cells containing slender wavy nuclei with pointed edges. Scattered cells showed enlarged hyperchromatic nuclei resembling degeneration. Mitoses were not increased (Figure 3).

Patient recovered well without hoarseness and discharged home in postoperative day-2. She was followed-up in the clinic with no major complications.

DISCUSSION

Neurofibromas of the vagus nerve are an extremely rare peripheral nerve sheath tumor. There occurrence in the cervical region is even uncommon. There are only ten reported cases of such lesions found in literature [2]. Majority of neoplasms of vagus nerve are schwannomas [4, 5].

Neurofibromas of the vagus nerve are slow growing peripheral nerve sheath tumors [3, 6]. Generally they are asymptomatic. Patients present with a painless neck lump with no significant disability. Some case studies have documented dysphagia and cough with percussion of the mass [4]. The most common age range is 20–40 years with no sex predisposition [7]. There may be an association (60%) with neurofibromatosis type 1 [8, 2].

Growth takes the fusiform shape surrounded by a pseudo-capsule with the stretched neural tissue. Most are benign with a malignant counterpart less frequently.
Preoperative imaging with MRI scan is preferred, to assess the tumor characteristics and extension and arriving at a differential diagnosis. Ultrasound scan and CT scan are also valuable imaging modalities especially in limited resource setting. Optimal surgical approach and planning of surgery is made according the findings.

Cytological assessment with FNAC is non-specific. Biopsy is discouraged due to risk of damaging the surrounding structures, bleeding and because of its effects on future definitive surgery.

Gross total resection is the definitive treatment (GTR). Neurofibromas prove to be most difficult nerve sheath tumors to achieve GTR. Removal of the tumor by internal decompression is preferred as it preserves the function and minimizes the damage to closely packed structures in the vicinity. If the neurofibroma is removed en bloc, microsurgical repair of the nerve is indicated with a nerve graft [5, 9–13].

Following the excision complications such as vocal cord palsy, dysphagia and arrhythmia could occur. Donner et al. previously reported a 16% decrease in motor function using the intra-capsular enucleation technique. Among them up to 10% developed pain syndromes [8]. Green et al. reported postoperative complications like occurrence of dysphagia (40%) and aspiration (46%) after vagus nerve tumor surgery [7]. Gilmer-Hill and Kline stated that nearly all patients who have vagus nerve tumor resections will develop transient hoarseness postoperatively [14]. Our patient did not develop any of these major complications. Patients should be followed-up for local recurrence because of its higher possibility [7].

CONCLUSION

Vagus nerve neurofibromas are rare nerve sheath tumors. Gold-standard treatment of symptomatic benign vagus nerve mass is an attempt at gross total resection with minimal loss of nerve function. Postoperative hoarseness and vocal cord paralysis can be avoided with meticulous surgical technique.

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Chathuranga L.S. – Substantial contributions to conception and design, Acquisition of data, Analysis and interpretation of data, Drafting the article, Revising it critically for important intellectual content, Final approval of the version to be published
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REFERENCES

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Accidental foveal burn following pan retinal photocoagulation and its long-term outcome

Khan Perwez, Pandey Kankambari, Khan Lubna, Saxena Nutan

ABSTRACT

Introduction: Changing lifestyle has led to rising trend of diabetes and its complications. There is an increased incidence of diabetic retinopathy with subsequent increased use of double frequency Nd:YAG lasers. Knowledge about the consequences of accidental exposure of these lasers and long-term prognosis will help us in better management of such accidents.

Case Report: A 65-year-old female patient who sustained accidental double frequency Nd: YAG laser foveal burn while undergoing left eye panretinal photocoagulation (PRP) for diabetic retinopathy is presented with subsequent five years follow-up. Upon initial evaluation, best-corrected visual acuity (BCVA) of affected eye was count finger at 1 foot (CF 1') left eye. Immediate funduscopic examination revealed foveal laser burn. Corticosteroids, in the form of 40 mg prednisolone, were administered orally for two weeks followed by 10 mg per week taper along with long-term topical nepafenac three times a day. Fifteen days after exposure, funduscopic examination revealed a distinct foveal scar followed by epiretinal membrane formation at six months follow-up. The BCVA of the affected eye improved to 20/60 over a period of five years. This clinical course is different from those of previously reported cases where visual acuity did not recover. Conclusion: Accidental exposure of double frequency Nd:YAG laser to fovea can lead to grievous injury with loss of vision. Prompt use of high dose systemic steroids, topical non-steroidal anti-inflammatory drops and natural healing can have better visual outcome.

Keywords: Double frequency Nd:YAG laser, Foveal burn, Panretinal photocoagulation

INTRODUCTION

Double frequency Nd:YAG laser (532 nm), is a solid state laser which emits green wavelength. It has good absorption in the melanin of retinal pigment epithelium with high absorption in oxyhemoglobin, thereby causing direct closure of microaneurysms. Its propensity for low absorption in xanthophyll reduces risk of neuroretinal damage [1, 2].

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An increase in the incidence of diabetic retinopathy and other retinal vasculopathies is noted in recent years due to changes in lifestyle and better diagnostic modalities leading to increased use of double frequency Nd:YAG lasers for therapeutic interventions. Hence there is a substantial risk of accidental exposures, whose long-term natural history is uncertain.

Case report of accidental exposure of fovea to frequency doubled Nd:YAG laser burn with subsequent five years follow-up is presented.

CASE REPORT

Panretinal photocoagulation was advised in a 65-year-old female having proliferative diabetic retinopathy (PDR) with best corrected visual acuity of 20/125 in left eye. It was performed using a double frequency Nd:YAG Laser by a medical trainee in a medical institute. Laser was intended for nasal retina but due to misinterpretation of temporal retina as nasal retina, entire macula including fovea was lasered by high energy burns of 200 milijoules, 150 duration and 500 micrometer spot size. Immediately after the exposure, patient experienced sudden loss of vision in her left eye. Best corrected visual acuity deteriorated to finger counting at 1 foot (CF 1') or 20/8000. Central scotoma in the affected eye was noted on Amsler grid test. Direct ophthalmoscopy and 90 D biomicroscopy revealed a circular greyish patch over the fovea and laser marks involving whole macula.

Corticosteroids, in the form of 40 mg prednisolone, were administered orally for two weeks followed by 10 mg per week taper along with long-term topical nepafenac three times a day. No improvement was noted in first two weeks.

Funduscopic examination at sixth month follow-up revealed a distinct epiretinal membrane which was confirmed by optical coherence tomography (OCT). Fundus fluorescein angiography (FFA) revealed multiple circular scar marks suggestive of laser treatment at macula and foveal region (Figure 1).

Follow-up at weekly scheduled visits highlighted that visual recovery in affected eye was 20/200 after two months.

At sixth month funduscopic examination along with an OCT revealed presence of epiretinal membrane and a prominent foveal scar (Figure 2) and visual acuity improved to 20/100.

Subsequent quarterly visual acuity assessment, funduscopic photography and OCT did not reveal significant changes. Four years later, an OCT revealed spontaneous dehiscence of epiretinal membrane along with improvement in patient’s visual acuity of 20/80 (Figure 3). There was continued presence of a foveal scar with central pigmentation on fundus examination. However, neither choroidal neovascularization nor macular hole formation was evident. Best-corrected visual acuity (BCVA) improved to 20/60 at fifth year follow-up.

DISCUSSION

Patients of proliferative diabetic retinopathy (PDR) and severe non-proliferative diabetic retinopathy (NPDR) have high risk of gross vision loss due to recurrent vitreous hemorrhage and fibro-proliferative changes consequent to neovascularization [3]. The regression of neovascularization achieved by pan retinal photocoagulation (PRP) by double frequency Nd:YAG
laser reduces risk of vision loss by 50% in comparison to untreated eye [2, 3].

New laser modalities, expanding delivery systems, and novel applications of laser energy have vastly expanded our armamentarium for the treatment of diabetic retinopathy. This has led to a concomitant increase in laser-induced injuries, particularly ocular injuries as energy focuses on the retina.

The natural history of laser induced foveal injury remains uncertain as such type of injury is uncommon in experienced hands. However, in training institutes such mishaps can occur by trainees especially in the absence of side observer scope, as it was, in the present case. It is not unusual to observe some visual recovery in cases of laser injury in macular area outside central fovea [4–6]. However, our patient showed subsequent improvement in visual acuity over a period of five years despite direct injury to the fovea by high intensity PRP laser burn.

Potential benefit of initial systemic corticosteroid or long term topical nepafenac in laser induced foveal injury needs to be substantiated by larger cohort studies [7].

Panretinal photocoagulation is avoided in a small oval area of macula, whose superior and inferior boundaries are formed by the respective arcades, nasal boundary by disc and temporal boundary lies two disc diameter temporal to fovea, as excessive energy of PRP burn is detrimental to its sensitive tissue. It is possible to accidently stray in this area during PRP as only a narrow strip of retina, which is to be lasered, is illuminated. So to avoid this complication one should intermittently refer back to fovea. Foveal burns can be avoided by finding the fixation point by projecting a fluorescein angiogram, using akinesia if cooperation is poor, meticulous technique and experience.

Diabetic macular edema was not noted following laser therapy and no anti-VEGF (vascular endothelial growth factor) injection was required in the follow-up of last five years. Thus, emphasizing the utility of laser therapy in treatment of diabetic retinopathy.

We concluded that in spite of direct laser injury to fovea, visual acuity may improve significantly in due course of time.

Although surgical intervention is the main modality of treatment for epiretinal membrane, spontaneous dehiscence may occur in long duration leading to improved visual acuity.

CONCLUSION

This case illustrates the importance of deep knowledge and vigorous training before laser can be handled independently. The minimal invasiveness of laser treatment has significant appeal. Nevertheless, complications are possible, but many can be avoided with meticulous technique and experience.

Author Contributions

Perwez Khan – Substantial contributions to conception and design, Acquisition of data, Analysis and interpretation of data, Drafting the article, Revising it critically for important intellectual content, Final approval of the version to be published

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Unusual third cranial nerve palsy presentation with unexpected distant departure point

Filipa Caiado Sousa, André Diogo Barata, Filipa Teixeira, Vítor Silva

ABSTRACT

Introduction: Oculomotor nerve palsy can arise as a result of a number of different conditions, and the differential diagnosis should take into account patient’s age, past medical history and clinical presentation. Diplopia, ptosis, restricted ocular movements, exotropia, with or without pupil involvement are common symptoms at presentation. Case Report: We present the clinical case of a 72-year-old Caucasian male with ophthalmological history of right eye amblyopia caused by an untreated esotropia, who went to the emergency room presenting holocranial headache, ptosis and temporary diplopia. The pupils were symmetric with a present but slowed down right direct reflex, and a doubtful Marcus Gunn pupil. The patient was orthotropic. Ocular motility examination showed a limitation in adduction, elevation and depression of the right eye. The patient had a computed tomography (CT) of the brain and orbits, a laboratory study, a CT angiography, a brain magnetic resonance, a lumbar puncture, a CT scan of the neck, chest, abdomen and pelvis, a flexible cystoscopy, and a transurethral resection. Once made, the histology of the biopsies of TUR revealed fragments of an infiltrative urothelial carcinoma of high grade. Conclusion: The oculomotor nerve palsy may be the first manifestation of a serious systemic disease and appear in an atypical form in a patient with a previous esotropia. Careful assessment and investigation should be taken.

Keywords: Oculomotor nerve, Third nerve palsy, Urothelial carcinoma

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INTRODUCTION

Oculomotor nerve palsy can arise as a result of a number of different conditions, and the differential diagnosis should take into account patient’s age, past medical history and clinical presentation. Oculomotor nerve injuries may be located at different points of its path and the anatomical relationship of the various portions of this nerve are responsible for many of the clinical features of third nerve palsy. Diplopia, ptosis, restricted ocular movements, exotropia, with or without pupil involvement are common symptoms at presentation [1].

CASE REPORT

We present the clinical case of a 72-year-old Caucasian male with ophthalmological history of right eye (oculus dexter) amblyopia caused by an untreated esotropia.
No relevant medical history was known, with irregular surveillance by his general doctor and no regular medication.

The patient presented with holocranial headache, right eye ptosis and temporary diplopia seven days prior to emergency room observation. He denied ocular pain, fever, previous trauma or other systemic or neurologic symptoms.

Ophthalmic exam disclosed right eye best corrected visual acuity of the right eye of 20/200 and of the left eye of 20/25. A right ptosis (Figure 1) with attainment of the visual axis, without any inflammatory signs. The pupils were symmetric with a present but sluggish right direct reflex, and a doubtful Marcus Gunn pupil. The patient was orthotropic (Figure 1). The ocular motility examination showed a limitation in adduction, elevation and depression of the right eye (Figure 2). No diplopia was present during observation neither other neurological defects. The slit lamp examination revealed a cataract and funduscopic examination was unremarkable.

Computed tomography (CT) scan of the brain and orbits did not reveal acute changes and a analytical study revealed raised inflammatory parameters with neutrophilic leukocytosis (2000, 92.9) and protein chain reaction PCR slightly elevated (4.69). CT angiography that did not reveal aneurysmatic dilations. Brain magnetic resonance imaging (MRI) scan showed countless small foci of enhancement leptomeningeals with cerebellar predominance and additional cerebral foci and in the the internal acoustic canal on T1 acquisition. These findings suggest a possible meningeal carcinomatosis.

Lumbar puncture revealed atypical cells (69 mm³), cerebrospinal fluid (CSF) cytology showed neoplastic cells suggesting infiltration by carcinoma (CK 18+). Cerebrospinal fluid serology was negative for Cryptococcus, Lyme disease and syphilis. Cerebrospinal fluid bacteriology was negative.

The patient underwent investigation for primary tumor site with CT scan of the neck, chest, abdomen and pelvis. The examination showed multiple lymph nodes on level III and IV referred mainly because of their number and size, the larger one with approximately 21x8.5 mm, and a mass with starting point on the left lateral wall of the bladder, with calcifications, measuring approximately 4x2 cm.

The patient was submitted to a flexible cystoscopy that showed a sessil lesion with 3 cm and with probable invasion of the meatus; for a best description a transurethral resection (TUR) of the bladder was proposed. Once made, the histology of the biopsies of TUR revealed fragments of a infiltrative urothelial carcinoma of high grade. The patient started treatment with corticosteroids and methotrexate. The final diagnosis was a palsy of the oculomotor nerve by meningeal carcinomatosis in the context of urothelial carcinoma. The patient was hospitalized in the oncology department and started a systemic treatment, unfortunately with a poor prognosis.

**DISCUSSION**

The oculomotor nerve supplies somatic: superior rectus, inferior rectus, inferior oblique, medial rectus and levator palpebrae superioris as well as autonomic (papillary sphincter and ciliary) muscles of the eye. Oculomotor nerve palsy may be congenital or acquired, complete or partial, pupil sparing or pupil involving, isolated or accompanied by neurological signs. Precise knowledge of its origin and course from nuclear level to terminal muscles is essential to localize the site of involvement [2].

Third cranial nerve palsy may be the first manifestation of a serious systemic disease and accounts for about one third of presenting cranial nerve palsies. The pupil involvement is commonly related to compressive lesions, in particular aneurysms, on the other hand, pupil sparing third nerve palsy suggest microvascular etiologies [3].

This case reveals an atypical presentation in a patient with previous esotropia and amblyopia and therefore diplopia was not an important symptom for the patient as well as exotropia was not present. For this reason, the patient was orthotropic. The pupil involvement was not obvious so we could consider it as an incomplete palsy
(anisocoria was not present), but since there was a sluggish right direct reflex, and a doubtful Marcus Gunn pupil, neuroimaging was mandatory to exclude life-threatening compressive causes. The management of acute partial pupil-sparing is controversial, some suggest that the incidence of aneurysms in this group is low enough that imaging is not necessary, others advocate angiography for all and others suggest a period of observation, since a pupil-sparing partial third nerve palsy from an aneurysm usually develops papillary involvement within a few days [4].

Once excluded aneurysmatic causes further investigation was needed. In this case, the oculomotor nerve palsy appears as a direct compression or infiltration of the oculomotor nerve in his pathway. There are some reports in literature of isolated oculomotor nerve palsy as a paraneoplastic manifestation: gastric diffuse large B cell lymphoma with neoplastic infiltration, although isolated neuropathy is rare, non-small cell lung cancer and Burkitt lymphoma. The majority of the patients with paraneoplastic manifestations and oculomotor nerve palsy exhibited cavernous sinus involvement rather than oculomotor nerve infiltration [5–7].

Literature has described a case of a multiple cranial palsies associated with gallbladder cancer [8].

Brain MRI scan combined with cerebrospinal fluid cytology examination is considered optimal for evaluating the cause of oculomotor nerve palsy, but may not be diagnostic in every case.

CONCLUSION

In conclusion, this case demonstrates that isolated oculomotor nerve palsy demands a careful assessment and complete investigation. This case of isolated nuclear oculomotor nerve palsy with atypical features may mimic oculomotor ischemic nerve palsy usually associated with diabetes mellitus and hypertension. The outcome of oculomotor nerve palsy is related to its cause.

**REFERENCES**

A case of collagenous colitis with cryptogenic organizing pneumonia

Yasuyuki Taooka, Yuka Ide, Yusuke Higashi, Gen Takezawa

CASE REPORT

A 73-year-old female during the treatment of cryptogenic organizing pneumonia (COP) consulted our outpatient clinic complained of chronic diarrhea lasting more than six months. There was no body weight loss, and she did not feel severe abdominal pain. Twelve months prior consultation, prednisolone and lansoprazole were started to be administrated against COP. Since the patient had a past history of peptic ulcer, proton pump inhibitor (PPI) was also started against the adverse effect of corticosteroid therapy. Administration of corticosteroid and lansoprazole was also continued, because of repeating recurrence and improvement of COP. On physical examination, abdomen showed no significant abnormal findings. Stool color was normal, and stool occult blood examination was also negative. And stool bacterial culture did not show abnormal finding. Blood laboratory examination showed as following; white blood cell count 4,890/μL, hemoglobin 15.0 g/dL, platelet count 199,000/mL, total protein 6.7 g/dL, albumin 4.1 g/dL, AST 34 IU/mL, ALT 31 IU/mL, LDH 196 U/mL (normal range: 106–211) fasting blood sugar level 78 mg/dL, amylase 118 IU/L, Na 142 mEq/L, K 3.8 mEq/L, Cl 108 mEq/L, Ca 9.6 mg/dL C-reactive protein 0.0 mg/dL, CEA 1.1 ng/mL, CA19–9 12.4 U/mL, and KL-6 302 U/mL (normal range: 0–499). Esophagogastroduodenoscopy showed peptic ulcer scar formation. Colon endoscopic examination showed diffuse cloudiness with small fine granule-like surface change from rectum to transverse colon (Figure 1). Biopsy samples of colon and rectal mucosa revealed infiltration of inflammatory cells into mucosal layer. And submucosal layer and homogenous, amorphous deposition of collagen fiber was recognized under the surface epithelial cells (subepithelial collagen band) (Figure 2). The diagnosis of collagenous colitis was performed and administration of lansoprazole was discontinued. A few weeks later, watery diarrhea spontaneously discontinued. Six months later, treatment of COP was finished. No recurrence of COP and collagenous colitis after three year.

DISCUSSION

Collagenous colitis is known as one of microscopic colitis, and induces watery diarrhea and chronic colorectal inflammation [1]. As the cause of collagenous colitis with this case, involvement of lansoprazole was suspected. After discontinue of administration...
of lansoprazole, chronic diarrhea was also stopped. Although it is rare, collagenous colitis is known as the one of the adverse effect of PPI. The COP is classified into one of idiopathic interstitial pneumonias. Etiology of COP is still remained uncertain. And interstitial pneumonia and COP sometimes complicates other organ disorders and connective tissue disease [2, 3]. As long as we examined, only two case reports discussing about interstitial pneumonia and collagenous colitis were reported so far [4, 5]. Recently, aberrant T cell response and Th1 cytokine were reported to be involved in inflammation of collagenous colitis [6]. Therefore, we could not rule out the possibility of mutual relationship between collagenous colitis and COP. Although both interactions are unclear, there are no reports to deny their interaction. In this case, collagenous colitis occurred during the treatment of COP, and collagenous colitis did not relapse since after remission of COP. Finally, this case was diagnosed as having lansoprazole induced collagenous colitis. Already there are many reports about relevance between PPI and collagenous colitis [5, 7–9]. This case had a past history of administration of rabeprazole against peptic ulcer, and there was no diarrhea during administration. Furthermore, this case was treated with omeprazole one year later after remission of COP, and there was not an episode of recurrence of watery diarrhea. After having the diagnosis of collagenous colitis, lansoprazole did not used again in this case. These means collagenous colitis of the case was highly associated with lansoprazole, not other kinds of PPI.

CONCLUSION

A rare case of collagenous colitis was reported. When having chronic diarrhea during treatment with proton pump inhibitors (PPI) including lansoprazole, possibility of adverse effect of PPI should be considered. Colon fiberscopic examination is useful for its diagnosis.

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Keywords: Collagenous colitis, Cryptogenic organizing pneumonia, Lansoprazole

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Conflict of Interest

Authors declare no conflict of interest.
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CASE REPORT

A two-year-old Sudanese male was presented in neurology clinical department with delayed growth and development, and seizures. Then referred to radiology department. Non-contrast magnetic resonance imaging scan of brain axial, sagittal and coronal. T1-weighted, T2-weighted, fluid attenuation inversion recovery (FLAIR) and diffusion weighted imaging (DWI) were done (Figure 1A–D). Images showed well defined intracranial cyst on left frontoparietal region connected with the ipsilateral lateral ventricle, associated with diffuse brain atrophic changes in term of dilatation of ventricular system, prominent cortical sulci and dilated extra-axial cerebrospinal fluid spaces.

The intracranial cyst margin not lined by a grey matter and is associated with a small amount of adjacent FLAIR hyper-intensities, no restricted diffusion noted in DWI. No soft tissue mass lesions. No intracerebral blood degradation products. Normal brainstem and cerebellum. Findings are impressive of congenital diffuse brain atrophic changes associated with significant left sided porencephalic cystic changes as described above.

DISCUSSION

Porencephaly is an extremely rare disorder involving encephalomalacia [1]. It has been classified as congenital or acquired. The congenital form is due to localize agenesis of the cortical mantle resulting in the formation a cavity or a lateral slit through which the lateral ventricle communicate with the convexity of the brain. The cavity is lined by ependyma and laterally by a thin pia-ependymal layer. The acquired type is secondary to any type of cerebral destructive process, ranging from trauma to infection. Sometimes called false porencephalic cyst [2].

Figure 1: Magnetic resonance imaging of brain. Axial images of porencephalic cyst on left frontoparietal region connected with the ipsilateral lateral ventricle (A) FLAIR image, (B) T2-weighted image, (C) Apparent diffusion coefficient image, and (D) Restricted diffusions image.
Patients with severe cases of porencephaly suffer epileptic seizures and developmental delays, whereas patients with a mild case of porencephaly display little to no seizures and healthy neurodevelopment. Infants with extensive defects show symptoms of the disorder shortly after birth [3].

Porencephalic diagnosis by magnetic resonance imaging, ultrasound and computed tomography scans. Magnetic resonance imaging scan of brain is the most sensitive and specific of the imaging techniques in children and adults. Because its sensitivity to distinguish porencephaly from open lipped schizencephaly, by absent of grey matter and associated with a small amount of adjacent FLAIR hyperintensity [4]. Porencephalic cyst should be differentiated from the neuroligial cyst, arachnoid cyst, interhemispheric cyst and holoprosencephaly. Neuroglial cyst is not communicating with the ventricles or subarachnoid space. Arachnoid cyst is extra axial in location and underlying grey-white matter is normal. Holoprosencephaly is due to normal neuronal separation, where fused thalami and monoventricles seen [5]. As of now, there is no definite cure for porencephaly. Research is still ongoing as to the causes of it and how to treat it. As of now, treatment is mainly supportive and consists: medications in the form of anticonvulsants are given to control the seizures. For infants with hydrocephalus due to porencephaly, use of a ventriculoperitoneal (VP) shunt is advised to remove excess fluid from the brain. The porencephaly patient can also undergo surgery for complete removal of the cyst from the cerebral hemisphere [6].

CONCLUSION

Magnetic resonance imaging scan of brain play important role to distinguish porencephalic cyst from other intracranial cyst.

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Keywords: Porencephalic cyst, Magnetic resonance imaging, Schizencephlic cyst

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Common peroneal nerve palsy caused by an initially misdiagnosed extraneural and intraneural benign ganglion cyst of the peroneal nerve in a 11-year-old child: A rare but severe condition

Ingo Schmidt

To the editor,

An 11-year-old obese boy presented with a right common peroneal nerve (CPN) palsy. At first presentation in our hospital, the patient reported progressive palsy starting only with painless weakness for dorsiflexion of his right foot two years ago. There was no history of any trauma. Within these two years, the patient was explored by his family doctor, a pediatrics, and a neurologist. The diagnostic management included magnetic resonance imaging (MRI) of the cerebrum and overall spine, electromyography, electroencephalography, analyses of blood and cerebrospinal fluid samples. No causes could be found by the treating physicians that declared his CPN palsy. So the diagnosis of a peroneal nerve mononeuropathy with unclear genesis was made, and the patient was treated by oral medication of glucocorticoids and vitamin $B_12$. On first clinical examination in our hospital, a painless foot drop was present, and the sensibility at the peripheral peroneal nerve area was completely lost. The strength of dorsiflexion of foot was completely lost according grade 0 in Medical Research Council scale (0–5), and electromyography revealed a severe axonal lesion in the absence of motor activities. Magnetic resonance imaging (MRI) scans of his right knee revealed a subcutaneous, well demarcated and lobulated cyst adjacent to the proximal tibiofibular joint which was clinically not palpable through the thick layer of subcutaneous fat of the obese patient (Figure 1A–B). According to these findings, the diagnosis of a proximal tibiofibular joint cyst was made by the radiologist. Based on the presented peripheral neurological symptoms, surgical revision was detected by us. The lesion was surgically exposed through a large longitudinal incision starting distally over the lateral aspect of fibula, passing the popliteal fossa with a vertical incision, and extending up longitudinally on the dorsal aspect of the thigh. Intraoperatively, a cyst originating from the proximal tibiofibular joint could not be found, but there was an extraneural and intraneural ganglion cyst with size of $7 \times 2.5$ cm involving the CPN, and both its superficial sensory nerve (SSN) and deep motor nerve (DMN) distally (Figure 1C). After careful dissection and removal of the tumor in a monobloc manner, a division of the CPN distally was seen, but macroscopically there were no structural damages (i.e., neurotmesis) both of the CPN and its DMN/SSN (Figure 1D). Histological examination confirmed the diagnosis of a benign ganglion cyst. The wound healing was uneventful. One year after surgery, a functional recovery could not be observed, so a tendon transfer procedure to restore dorsiflexion of his foot was recommended by us, but declined by the parents of the patient at this time.

The CPN originates from the sciatic nerve, passes posteriorly and laterally to the biceps femoris muscle in the popliteal fossa, crosses the head of the fibula, and descends to lower leg after dividing in its SSN and DMN. Most often, CPN palsy occurs at the fibular neck, where the nerve is superficial and vulnerable to direct injury, overstretching, and entrapment. The entrapment of CPN potentially leading to palsy (i.e., foot drop) caused by extraneural tumors around the proximal tibiofibular joint is known from literature [1].

Nerve entrapment caused by ganglion cysts is still widely known from the upper extremity. Ganglion cysts around the proximal tibiofibular joint leading to CPN palsy, first described in 1921 by Sultan [2], is much less common, it has a peak incidence in the fourth decade of life, and it may occur both extraneural and intraneural [3–5]. Our patient’s case suggested that ganglion cysts of the peroneal nerve in MRI can mimicking proximal tibiofibular joint cysts. Its prevalence with 0.76% is
also very rare that was found in a cross-sectional study including 654 knee MRI scans in patients with the most common clinical diagnosis of meniscal tears (42.8%), the mean age of these patients was 43.4 years (range 42–54 years), and the cysts ranged in size from 1.0–2.8 cm [6]. In literature, some case reports have been described CPN palsy caused by a ganglion cysts of the peroneal nerve or a cyst originating from the proximal tibiofibular joint in children [7–12].

With our patient, it must be noted critically that the ganglion cyst was initially misdiagnosed over a period of two years, and so a functional recovery could not be achieved, despite surgical removal of the cyst and neurolysis of the CPN. It must be suggested that the prolonged time in diagnostic management (initially demyelinating entrapment with weakness only) led to a severe and irreparable axonotmesis of the peroneal nerve. When a CPN palsy is present, surgical nerve decompression with neurolysis should be done as early as possible. The prognosis of functional recovery in patients with CPN palsy depends on the severity of neurological deficit and time of surgical neurolysis. In general, the prognosis for a demyelinating lesion is much more favorable than for an axonal loss lesion [13]. The outcome is usually favorable if surgery is done within four months after first presentation of neurological deficits, and less favorable in patients who have neurological symptoms for longer than one year [1, 14]. For irreparable CPN palsy, a tendon transfer procedure utilizing the tibialis posterior, first described in 1933 by Ober [15], with or without a combined transfer of the flexor digitorum longus is the method of choice for functional recovery of patients (Figure 2A–C) [16, 17].
Keywords: Ganglion cyst peroneal nerve, Common peroneal nerve palsy


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