two adult patients with angiomyolipoma.[2–4] Gastrointestinal PEComa shows a marked preponderance in women and one-third of the cases occur in the paediatric age group.[1] These tumours may be associated with neuroblastoma and tuberous sclerosis.[2,5]

PEComa seems to have a similar biological behaviour to GIST.[6] The negative reactivity for CD117 and CD34 went against the diagnosis of epithelioid GIST and KIT-negative GIST. Endocrine tumours are usually positive for NSE and synaptophysin. Cytokeratin was negative, excluding the diagnosis of carcinoma. Germ cell tumour was ruled out by the negative reaction for AFP and PLAP. Malignant melanoma and granular cell tumour could not be considered because S100 was negative. Smooth muscle tumour with epithelioid morphology was considered as a differential diagnosis due to the positivity of few cells for desmin and SMA. However, there were no spindled foci with perinuclear vacuoles or cigar-shaped nuclei. In our patient, the morphology of the tumour showed abundant granular eosinophilic to focally clear cytoplasm, delicate sinusoidal network and presence of cytoplasmic glycogen; these were in favour of PEComa.[7] The immunohistochemical reaction with diffuse strong positivity for HMB-45 antibody, and focal positive staining for SMA, highlighted the myomelanocytic nature of this tumour.[5,7]

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Should gut malrotation be suspected in adolescents and young adults presenting with failure to thrive?

Introduction

Failure to thrive in young patients is usually attributed to chronic infections such as tuberculosis, human immunodeficiency virus (HIV) or to malabsorption syndromes. Gut malrotation (GM) as a cause of failure to thrive is usually a far-fetched suspicion. It is a congenital anomaly referring to either lack of or incomplete rotation of the foetal intestines around the axis of the superior mesenteric artery (SMA) during foetal development. Approximately 90% of patients with GM are diagnosed within the first year of life; of which 80% are diagnosed within the first month of life.[1]

Case report

Two young patients presented with failure to thrive and non-specific abdominal symptoms. Subsequently, they were diagnosed as cases of GM, which was responsible for their clinical condition.

The first case was a 13-year-old boy who presented with a history of poor appetite and failure to thrive since he was 5 years old. He was considerably malnourished and his weight was only 12 kg. Abdomen showed visible left to right peristalsis

in the epigastrium. Ultrasound of the abdomen revealed dilated duodenum with reverse peristalsis. Upper gastrointestinal (GI) endoscopy showed dilated first and second part of the duodenum. The child was resuscitated aggressively. Visible left to right peristalsis and bilious vomiting raised the possibility of an obstructive aetiology. Exploratory laparotomy was performed. It was found that there was partial malrotation of the midgut with dilated incomplete C loop of the duodenum and absent ligament of Treitz. The SMA was posterior to the duodenum, the bands of Ladd were overlying the anterior surface of the duodenum and the mesentery was short and narrow. The bands were divided, mesentery widened and the caecum was replaced and fixed in the right iliac fossa. Postoperative recovery was uneventful. The child started feeding well and gained weight. At 8 months of follow-up, he had gained up to 12 kg.

The second case was a 22-year-old woman who presented with a history of weight loss, vague abdominal pain and distension of 8 years duration. She had undergone surgery for congenital diaphragmatic hernia (CDH) soon after birth. Abdominal examination revealed visible left to right peristalsis. Barium meal showed dilated duodenum and small bowel with markedly delayed emptying. In view of uncertain diagnosis, she was taken up for exploratory laparotomy and was found to have GM with caecum and appendix in the left hypochondrium and there was no duodenojejunal (DJ) flexure. Adhesions were released, mesentery widened and appendectomy was done. The postoperative recovery was good and after 6 months, the patient has gained 17 kg of weight.

Discussion

Congenital bands extending from the right lateral abdominal wall, across the duodenum to the caecum are called the bands of Ladd. These may cause external compression to the duodenum and may be the cause of upper abdominal pain, bloating, loss of appetite and subsequently malnutrition and failure to thrive. In our first case, there was no rotation of the proximal segment, leading to absence of C loop of the duodenum and ligament of Treitz and also the SMA lying posterior to the duodenum. The second case was also of partial GM and it was associated with CDH. The most common congenital anomaly associated with CDH is intestinal malrotation and it is present in almost all cases of CDH.[2]

Presentation of GM after childhood is rare and, as seen in the reported cases, is that of failure to thrive, unexplained weight loss and vague abdominal symptoms.[3] GM is usually not considered as an aetiology because the symptoms are vague and non-specific. This may lead to delay in diagnosis and treatment. For these reasons, it is crucial that all surgeons should have a high index of suspicion for such a late presentation of GM in malnourished patients with upper GI symptoms; and have firm knowledge of intestinal embryology and its anatomical variations.

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Intracranial fungal infections in chronic liver disease: a report of two cases

Introduction

We report two patients of advanced cirrhosis who developed intracranial fungal infections, each of them having variable outcome with the treatment administered.

Case report

Case 1:

A 40-year-old man with alcoholic liver disease (Child C cirrhosis; Child–Pugh score 12) presented with complaints of fever for one month and altered sensorium for a week. He was in grade