rerouting the tube through the nose. While rerouting , we retrieve the nasally placed Ryle's tube from the posterior pharynx into oral cavity by using a tongue depressor and Magill forceps or by manipulating it with fingers. Both these procedures cause discomfort to patients and may traumatize the posterior pharyngeal wall.

In principle our technique avoids the passage of the nasally placed RT deep into the esophagus. This is achieved by instructing the patient not to swallow and by applying gentle external pressure on the cricoid cartilage. The anterior part of the cricoid cartilage forms an important landmark in front of the neck; it lies opposite the sixth cervical vertebra, and demarcates the junctions of pharynx with esophagus and larynx with trachea.[1] Thus given its position above the pharyngoesophageal junction, external pressure on the cricoid cartilage compresses the upper esophageal sphincter against the vertebral column thus preventing the passage of RT deep into the esophagus. Further, by instructing the patient not to swallow and advancement of the nasally placed RT simultaneously causes redirection of the RT into the oral cavity with or without loop formation (**Figures 1 & 2**). Once RT comes out through the oral cavity its tip is traced and attached to the NJT, NGT or NBD catheter already in place. Thereafter the nasally placed RT is pulled through the anterior nares which redirects the orally placed tube back through the nose.

This technique was prospectively applied on 30 patients, and was successful in 28 patients. One patient was extremely uncooperative, in whom the RT came over dorsum of tongue and the other patient needed transoral manipulations with fingers. None of the patients experienced aspiration or transtracheal passage of the tube. The advantages of this technique include, 1) less discomfort to the patient, 2) lower risk of posterior pharyngeal trauma, 3) avoids accidental bite injury to endoscopists, particularly with uncooperative patients, and 4) decreases cost and burden of sterilizing instruments.

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An unusual case of epidermoid-splenic cyst

Introduction

Splenic cysts are uncommon entities with an incidence of 0.5-2.0%.[1]Acomprehensive differential diagnosis of cystic lesions of the spleen includes parasitic echinococcal cyst, congenital cyst, intersplenic pancreatic pseudocyst, pseudocystarising from splenic trauma,necrotising infection, pyogenic splenic abscess, metastatic diseases and cystic lymphangioma or hemangioma.[2]

Case report

A 35-year-old male presented with complaints of abdominal discomfort and pain left hypochondriumradiating to ipsilateralshoulder. There was no history of trauma and weight loss. Physical examination revealed a large palpable ,nonmobile, firm to hardswellingin the left hypochondrium. Routine investigations were normal. An ultrasonogram (USG) revealeda well defined cystic mass.On CT scan a 14 × 12 cm cyst was localized to the spleen which had displaced the splenic parenchyma andthe possibility of a hydatid cyst was entertained. Exploratory laparotomy with splenectomy was performed. Peroperatively a large cyst sized 14 × 12cm was seen. The splenectomyspecimen was received in our pathology department,weighing 550gm and measuring 15 × 13 × 4cm. Grossly, awell encapsulated cystic area along with normal splenic tissue was identified. On cut section, the cyst had a thick wall and contained brown colored fluid. The interior surface was trabeculated (**Figure 1**). Microscopic examination revealed a thick fibrous wall lined by squamous epithelium (**Figure 2**). Focal areas of calcification were also present. Immunohistochemistry was positive for cytokeratin (CK) (**Figure 3**) and negative for calretinin. The final diagnosis of a

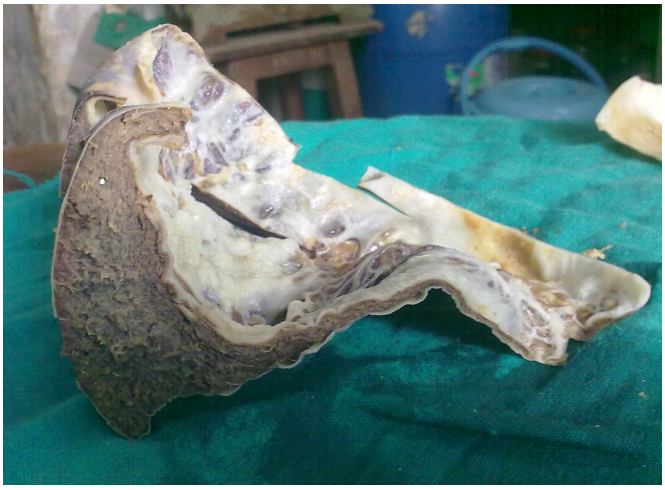

Figure 1: Gross specimen showing anunilocular cyst with attached spleen.

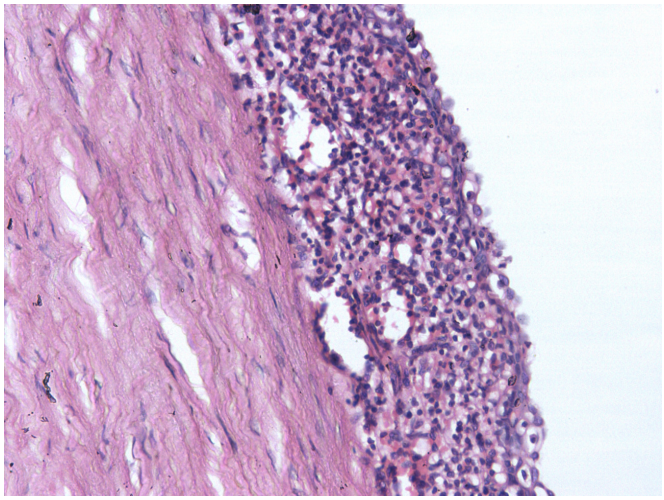

Figure 2: Cyst lining of the primary epidermoidsplenic cyst at 200× magnification.

primary epidermoid cyst of spleen was made.

Discussion

Fowler in 1953 reviewed 265 cases from global literature and proposed the first pathological classification for splenic cysts, dividing them into parasitic (75%) and non-parasitic cysts (25%)[3]. Non-parasitic cysts are further classified as primary or secondary cysts. Secondary cysts are associated with splenic trauma or hemorrhage. No epithelial lining is present in these and the cyst wall is composed of dense fibrous tissue with 25% of cases showing areas of calcification. A primary or true cyst on the other hand has a thick fibrous wallwhich is at least partially surfaced by amesothelialand/or a squamous epithelial layer.[4]Depending on the type of lining they are classified as epidermoid, dermoid and mesothelial cysts.[5]

Epidermoidcyst of spleen is a rare primary cyst. These are

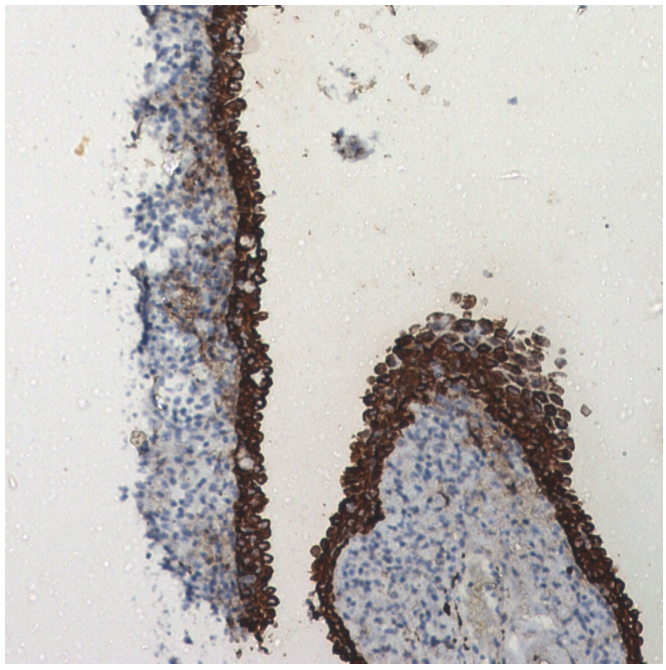

Figure 3: Immunohistochemistry displaying the cytokeratin positive lining of the primary epidermoid splenic cyst.

common in thesecond and third decades of life, but can occur at any age. Male predominance has been suggested. An abdominal mass is the most common presentation accompaniedby abdominalpain or gastrointestinal disturbances.[6] Onultrasonography, epidermoid cysts appear as well defined thin walled anechoic lesions. On CT scan, they manifest as rounded, well demarcated non-enhancing water attenuation lesions. Sonographic and CT scan findings are not specificand various cysts may present with similar appearance.Complications of cysts include rupture, infection and intracystic hemorrhage. Spontaneous rupture of a splenic cyst can cause massive haemoperitoneum or peritonitis. If the cyst is small (<5cm) no treatment is needed. If it is bigger than 5cm, is located at the hilum or is causing complicationsthen surgery is the gold standard line of management. Different modalities include splenectomy, partialsplenectomy, cystectomy, partial cystectomy or splenic decapsulation.[4] Microscopicallythe presence of an epithelial lining confirms the cyst as a primary splenic cyst. The final diagnosis can only be made at histopathology and confirmed by immunohistochemistry.

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