Hydatid cyst of the neck

Sir,

Only a few cases of hydatid cyst occurring in the neck area have been reported – the biggest series being 9 cases reported by Touhami et al.1 This prompted us to report a further case.

A 25 year old male presented with a swelling on the left side of the lower neck, gradually progressive in size for one year. The swelling was 10 x 8 cm, situated partially under the left lower part of the sternocleidomastoid and was soft in consistency. Oral and ear, nose and throat examination were normal. Fine needle aspiration was carried out which revealed clear fluid and the cytology report was inconclusive. The cyst was excised and the histopathology confirmed it to be echinococcal disease. During follow-up the patient was evaluated for any other site of hydatid, but radiology of chest and ultrasonography of liver, spleen and kidney were normal.

Hydatid cyst develops most commonly in liver (60%), lungs (20%) and rarely in brain, eye, heart, bone or other internal organs.2

The disease presents as a slow growing benign tumour with pressure symptoms according to its site of occurrence. Immunoelctrophoresis is a highly specific test for diagnosis. In the neck area it is very difficult to diagnose hydatid cyst. Even fine needle aspiration cytology is inconclusive unless one submits it for microscopic examination with a high suspicion index. In endemic areas, one should keep in mind the possibility of hydatid cyst when presented with a cystic lump in the neck.

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References

Chilaiditi’s syndrome presenting as unexplained tender hepatomegaly

Sir,

Hepatodiaphragmatic interposition of intestine (HDI; Chilaiditi’s syndrome) is a condition when part of the intestine is interposed between the liver and the right dome of the diaphragm. It is a rare condition, the incidence varying between 0.006% to 0.2%, depending on the age and sex of the patients.3 Recently, we have come across an unusual case of HDI mimicking amoebic liver abscess.

A 46 year old labourer was admitted with moderate to severe, throbbing, non-radiating pain in the right upper quadrant of the abdomen of 8 days duration, low grade, intermittent fever and anorexia. Physical examination revealed a firm, smooth and tender liver palpable 15 cm below the subcostal arch in the right midclavicular plane. The percussion note over the liver was tympanitic in character which merged with the note over the chest. The patient was diagnosed as having an amoebic liver abscess, and was treated with metronidazole.

Complete haemogram and liver function tests were normal. The routine stool examination for Entamoeba histolytica was negative. The plain X-ray of the abdomen taken in the erect position revealed gas under the right diaphragm with a horizontal fluid level. Ultrasonography and contrast enhanced computed tomography confirmed the colonic interposition between the liver and the right dome of the diaphragm. The liver was normal and was pushed downward and medially by the colon. During his hospital stay, he remained afebrile, although the tender hepatomegaly persisted.

HDI is generally described as an asymptomatic and clinically silent syndrome. However, it is of interest to radiologists since it has to be differentiated from the various causes of ‘air under the diaphragm’.2 This condition may occur transiently (sliding type), however, adhesions may cause persistence of the interposition.3 The interposed organ is hepatic flexure of the colon in most cases but small intestine and omentum may also be present. Multiple factors, discussed at length in an earlier report,4 are implicated in the causation of interposition. This condition may not be entirely clinically irrelevant, as symptoms such as diffuse abdominal pain, nausea, vomiting, flatulence, constipation, shortness of breath and pain in the substernal area may occur. Various authors have emphasized that abdominal tenderness and displacement of the liver to the left side of the abdomen may be observed. However, a moderately large palpable liver of 15.0 cm with tenderness over the right hypochondrium, as observed in the present report, has not been previously described. Since Chilaiditi’s syndrome is relatively frequent in the rural population and those consuming a vegetarian diet, for example, the Mediterranean region5 and Siberia,6 it should theoretically be more prevalent in the Indian subcontinent, though no such epidemiological study has been reported from this area. Hence, it is rational to suspect HDI, when unexplained hepatomegaly (with or without tenderness) is encountered.

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References


Diagnosis of hepatic hydrothorax in the absence of ascites by intraperitoneal injection of 99m-Tc-Fluor colloid

Sir,

Hepatic hydrothorax affects approximately 5% of patients with cirrhosis of the liver. Although usually associated with ascites, some patients show no clinically detectable intraperitoneal fluid, and a few cases with negative abdominal ultrasonographic examination have also been reported. The pathogenesis of hepatic hydrothorax has been controversial. Some studies have demonstrated a transdiaphragmatic flow of ascitic fluid to the pleural space through communications between peritoneum and pleura, which have also been observed at necropsy. We report a cirrhotic patient who developed hepatic hydrothorax. The administration of colloid Fluor-Sn-Tc99m into the peritoneal cavity demonstrated a peritoneal–pleural communication when there was no ultrasonographic detectable ascites.

A 66 year-old male with alcoholic cirrhosis and previous ascites and right pleural effusion was admitted in September 1989 with a massive right pleural effusion, but no clinically detectable ascites. The serum albumin level was 34 g/l and the pleural fluid examination indicated a transudate. Abdominal ultrasonography showed no evidence of ascites. A total of 370 MBq of Fluor-Sn-Tc99m was injected into the peritoneal cavity. Thorax and abdomen were scanned and images obtained at 15 min and 5 h showed a continuous and progressive flow of the radiopharmaceutical into the pleural cavity. A rectilinear hyperactivity over the right hemidiaphragm, suggesting the existence of a communication could also be observed. A chemical pleurodesis with tetracycline gave only a transitory benefit. Two weeks after this the effusion recurred and the patient died 3 months later. The necropsy showed mild ascites and a large pleural effusion. However, a detailed examination of the right hemidiaphragm failed to evidence any defect.

This case report demonstrates the natural evolution of hepatic hydrothorax in several respects. First, the patient developed a massive pleural effusion with a progressive reduction in the amount of peritoneal fluid which could not eventually be demonstrated by ultrasonography as reported previously. Thus absence of ascites cannot exclude the cirrhotic aetiology of a pleural effusion. Second, immediately after each thoracentesis, we noticed a rapid reaccumulation of pleural effusion. This can be explained by transdiaphragmatic flow of fluid, due to the negative intrapleural pressure.

Thoraco-abdominal scintigraphy with intraperitoneal administration of Tc99m colloid has been useful in the diagnosis of hepatic hydrothorax even in the absence of clinical ascites. The case reported is the first to our knowledge in which radionuclide imaging has been performed with ultrasonographic proven absence of ascites.

With respect to the treatment of hepatic hydrothorax the combination of chemical pleurodesis and peritoneal shunt should probably be used as conservative measures usually fail.

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