Management included face-mask continuous positive airway pressure (CPAP) with a good response. At 24 hours, however, she became pale and inactive and blood culture was positive for group B haemolytic streptococcus. Rapid improvement followed penicillin treatment, and CPAP was discontinued after 72 hours. Progress was good until day 10 when she again became pale and inactive with increasing respiratory distress. Clinical signs and chest radiography suggested right pleural effusion, but aspiration failed to confirm the presence of fluid. On day 12 apnoea necessitated mechanical ventilation. Chest signs were unchanged but the abdominal findings were remarkable. The liver was not palpable in the right hypochondrium, which felt strikingly "empty." It was, however, easily felt more medially as a mass extending to the level of the umbilicus. A well-defined edge passed beneath both costal margins at the lateral border of the rectus muscle. Chest radiography (see figure) confirmed the medial displacement of the liver and showed radiolucent areas in the right lower thorax. At the time loculated pyopneumothorax was considered the likely diagnosis. On a Bennett ventilator and antibiotics improvement was steady, with resolution of the radiolucent areas on chest radiography and the abdominal signs. But on day 18 she collapsed again. The physical sign was again noted of an empty right hypochondrium with the liver palpable only in the epigastrium. Radiography showed a loop of bowel, lateral to the liver, extending in the right hemithorax up to the level of the third dorsal vertebra. She failed to respond to increased ventilator pressures and 100%-inspired oxygen and died three hours later.

Chest radiograph on day 12 showing radiolucent areas on right with mediastinal and hepatic shift to left.

Necropsy showed that there was herniation of a loop of small bowel and the right lobe of the liver through a large central defect in the right diaphragm. A well-marked fibrous constriction was present around the right lobe of the liver. No obvious ascites, pleural effusion, hernial sac, or lung hypoplasia were found. There was extensive bronchopneumonia in the left lung.

Case 2

A 3660 g boy delivered by forceps for fetal distress at term had respiratory difficulty from birth. The liver was not palpable in the right hypochondrium which felt empty, but in the midline it extended down to the level of the umbilicus, with its edge passing beneath the right costal margin in the mid-clavicular line. Chest radiography showed a RDH. The baby died shortly after transfer to the regional centre, where emergency thoracotomy showed small-bowel and right liver-lobe herniation through a large central diaphragmatic defect.

Discussion

The diagnosis of RDH is often difficult. The first case particularly illustrates many of the lesser-known but reported features. Chest radiography may initially be normal.¹ The radiological changes may not only mimic inflammatory chest disease and pleural effusion but can also be intermittent.² The liver at first prevents herniation through

a large right-sided defect until changes occur in the relative intra-abdominal and thoracic pressures.³

It is in the context of these diagnostic difficulties that we report the unusual clinical sign present in both these infants. The finding of a liver extending unusually far below the costal margin in the midline, yet impalpable laterally, was in each case subsequently proved to be due to partial herniation of the right lobe of the liver through the diaphragmatic defect.

We thank Dr R L'E Orme and Dr L Haas for permission to report these cases.

¹ Canino, C W, *et al*, *Radiology*, 1964, **82**, 249.

² Kirchner, S G, *et al*, *Radiology*, 1975, **115**, 155.

³ Hurdiss, L W, Hooshand, Taybi, and Johnson, L M, *The Journal of Pediatrics*, 1976, **88**, 990.

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Small-intestinal obstruction from a Richter's hernia at the site of insertion of a laparoscope

Laparoscopy has become a standard gynaecological technique that is also widely practised by gastroenterologists in Europe, America, and Japan. Only recently in Britain has the procedure been recognised as a valuable technique for replacing or augmenting laparotomy, particularly in managing jaundice¹ and liver disease.² Complications are uncommon, and the Royal College of Obstetricians and Gynaecologists is conducting a national survey into their incidence among its members. I report a case of small-intestinal obstruction as a result of a laparoscopy.

Case history

A 61-year-old Indian woman, who came to Britain 12 years ago, presented to Dr P J Toghill with hepatosplenomegaly and iron deficiency anaemia. Her platelet count and prothrombin time were normal. Since she spoke almost no English and was unable to co-operate enough to allow percutaneous liver biopsy to be undertaken with safety, she was referred for laparoscopic liver biopsy under general anaesthetic, and this was performed on 28 June 1977. After induction of a 4-litre carbon dioxide pneumoperitoneum, an infraumbilical stab incision was used for inserting a 12 mm trocar and an Eder laparoscope.

A grossly enlarged spleen was seen, and the liver appeared normal. Biopsy of the right lobe was carried out under laparoscopic control. She made an uneventful recovery. The liver histology was normal. Six days after laparoscopy she started to vomit copiously. Oral intake was reduced, and she received intravenous fluids but continued to vomit. There was no abdominal pain or tenderness. On the 12th post-laparoscopy day, supine and erect abdominal radiographs showed the features of high small intestinal obstruction.

At laparotomy on the 13th post-laparoscopy day, the upper jejunum, duodenum, and stomach were grossly dilated. The mid-jejunum was tethered to the anterior abdominal wall at the site of the laparoscopy stab incision by a Richter's type hernia, which resulted in kinking of the bowel causing obstruction. Since the jejunum would not reduce easily, the laparoscopic stab incision was re-opened and the hernia reduced. The bowel wall was viable. The spleen was noted to be enlarged and surrounded by many adhesions. The hernial defect was repaired and the incision closed. She made an uneventful recovery from the operation. The cause for her splenomegaly and anaemia remain obscure, however.

Comment

Schiff and Naftolin³ reported two patients with small bowel

incarceration at the site of laparoscope insertion that resulted in intestinal obstruction, and claimed that this was the first report of this complication of laparoscopy. Both patients eventually needed small bowel resection, 14 and 21 days after laparoscopy. These two patients, and the one described above, were initially well after laparoscopy and then started to vomit on the third and the sixth days after laparoscopy. Thompson and Wheelless⁴ reported that burns or traumatic injury typically present between the third and seventh post-laparoscopic day and then need urgent surgical treatment. They recorded 11 bowel injuries in a series of 3600 laparoscopic sterilisations. Thus, obstructive symptoms occurring a week or more after laparoscopy may be due to bowel incarceration at the site of laparoscope insertion.

Herniation may occur at the site of laparoscopic insertion, and Bishop and Halpin⁵ reported protrusion of omentum through an infraumbilical laparoscopy incision. This occurred on the third post-laparoscopy day after a coughing bout.

These three reports of herniation and one of protrusion at the site of laparoscope insertion were all associated with either 11 or 12 mm-diameter instruments. These rare complications might be eliminated if smaller diameter instruments, which are available, were used.

¹ Cuschieri, A, *Annals of the Royal College of Surgeons of England*, 1975, **57**, 33.

² Balfour, T W, *Lancet*, 1976, **1**, 612.

³ Schiff, I, and Naftolin, F, *Obstetrics and Gynecology*, 1974, **43**, 674.

⁴ Thompson, B H, and Wheelless, C R, *Obstetrics and Gynecology*, 1973, **41**, 669.

⁵ Bishop, H L, and Halpin, T F, *American Journal of Obstetrics and Gynecology*, 1973, **116**, 585.

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Hepatic-cell adenoma presenting with intraperitoneal haemorrhage in the puerperium

An association between hepatic-cell adenomas and the contraceptive pill was first suggested in 1973 by Baum *et al.*¹ and since then over 100 cases have been recorded.²⁻⁵ We describe a case of fatal intraperitoneal haemorrhage due to rupture of a hepatic-cell adenoma that presented five days after caesarean section and 13 months after stopping oral contraceptives.

Case report

A 26-year-old woman presented at 15 weeks in her fourth pregnancy. After her third pregnancy she started lynestrenol and ethinyloestradiol (Minilyn) and continued for three and a half years. Her pregnancy started three months after stopping oral contraceptives and progressed normally until 38 weeks, when moderate pre-eclampsia developed and labour was induced. Fetal distress necessitated a caesarean section, and a healthy 3180 g girl was delivered through a lower segment incision. The puerperium was satisfactory until the fifth day, when the patient suddenly complained of weakness and epigastric pain. The patient rapidly became shocked.

Laparotomy disclosed massive intraperitoneal haemorrhage caused by a ruptured ill-defined yellow tumour that affected both lobes of the liver. The tumour was biopsied, and the bleeding site was packed with surgical and the capsule sutured. Histological examination showed multiple fragments of a hepatic-cell adenoma with considerable disruption of the tissue by haemorrhage. Typically, the adenoma was devoid of portal triads and consisted of hepatocytes in an almost normal trabecular arrangement with moderately numerous binucleated and multinucleated cells (see fig). There were no fibrous tissue septa. A vascular component was prominent, and there were numerous thick-walled blood vessels but no blood-filled cysts. In the sinusoids extramedullary haemopoiesis was present. The patient never regained consciousness, and despite intensive treatment and assisted respiration died seven days later.

Hepatic-cell adenoma showing a trabecular arrangement with binucleated and multinucleated hepatocytes. There is no fibrosis and portal triads are absent. (H and E $\times 77$.)

At necropsy the liver weighed 2600 g and was enlarged by a circumscribed yellow-tan tumour that had replaced most of the left lobe and extended across the hilum into the right lobe. It measured 10 $\times$ 10 $\times$ 5 cm and had ruptured through the posterior inferior surface of the liver. There was no spread of tumour beyond the liver. The histology was similar to that of the biopsy specimen and confirmed a hepatic-cell adenoma.

Comment

Hepatic-cell adenomas are usually reported in association with oral contraceptive use and appear related to the drug exposure time. Regression of the tumour probably occurs on withdrawal of medication, and diagnosis after ceasing oral contraceptives is uncommon.²

Presentation of a hepatic-cell adenoma with rupture in the puerperium 13 months after stopping oral contraceptives is an unusual sequence of events, but Fechner,⁴ in an extensive review, noted two patients with similar histories. An analysis of the first 100 cases in a registry of liver tumours⁵ started in 1973 found three hepatic-cell adenomas presenting in the puerperium, but with no history of oral contraceptive hormones. The evidence for a relationship between oral contraceptive hormones and hepatic-cell adenomas is now reasonably well established,²⁻⁴ but the association with pregnancy has been noted only in a few cases, and this is not evidence of a causal link.

Possibly, however, pregnancy could influence the outcome of an adenoma already present in the liver. By stimulating active cellular growth pregnancy may predispose to haemorrhage and rupture a tumour that might otherwise have remained symptomless and regressed once oral contraceptive hormones were stopped.

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¹ Baum, J K, *et al*, *Lancet*, 1973, **2**, 926.

² Sherlock, S, *Gut*, 1975, **16**, 753.

³ Edmondson, H A, Henderson, B, and Benton, Barbara, *New England Journal of Medicine*, 1976, **294**, 470.

⁴ Fechner, R E, *Human Pathology*, 1977, **8**, 255.

⁵ Christopherson, W M, and Mays, E T, *Journal of the National Cancer Institute*, 1977, **58**, 167.

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