**Letters**

**Single-port transumbilical laparoscopic-assisted appendectomy**

Laparoscopic appendectomy (LA) is generally performed using three ports, although two-port LA technique has been described for acute appendicitis by completing appendectomy extracorporeally through one of the ports.\(^1\)\(^2\)\(^3\) We evaluated the safety and feasibility of single-port transumbilical laparoscopic-assisted appendectomy (TULAA) for uncomplicated appendicitis.

Single-port TULAA was done in eleven patients aged 12 to 56 years (mean 34) with uncomplicated appendicitis. Patients with appendicular mass and overt appendicular abscesses were excluded. A 10-mm operating telescope with a 5-mm telescope and 5-mm working channel was introduced through a single 10-mm umbilical port. The appendix was visualized, and grasped with a non-traumatic grasper and evaluated for mobility. The appendix was pulled out from the umbilical port after deflation of pneumoperitoneum and appendectomy carried out in the conventional way. The average operative time was 20 minutes (range 15-25). Mean postoperative stay was 1.5 days (range 1-2); six patients were discharged on the same day. Only one patient required narcotic analgesics postoperatively for pain control. There were no major or minor postoperative complications. All patients were satisfied with the cosmetic results.

Single-port TULAA has been described in literature.\(^4\)\(^5\)\(^6\) We performed single-port TULAA by an extracorporeal method through the umbilical port. This technique is feasible only when the cecum is intraperitoneal and appendix is mobile. If the appendix is retrocecal or adhesions are present, or if the cecum is immobile, then this technique may be converted to two-three-port technique. The procedure is also not suitable for very obese patients or for complicated appendicitis. If the diagnosis of appendicitis is doubtful, an additional port may be required to screen the small bowel and pelvis.

Single-port TULAA is simple and safe, as it is performed under direct vision extracorporeally. It is virtually scarless and an effective way for the management of uncomplicated appendicitis in selected patients.

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**Serum hepatocyte growth factor in autoimmune and hepatitis B-associated liver diseases**

Hepatocyte growth factor (HGF) is produced by various cells types and is the most potent complete mitogen for the liver. Serum levels of HGF are elevated in acute liver disease, cirrhosis and hepatocellular carcinoma (HCC). HGF has been suggested as a better indicator than alfa-fetoprotein (AFP) for the carcinogenic state of the liver.\(^1\)\(^2\) We studied serum HGF levels in hepatitis B virus and autoimmune associated liver diseases. The study was approved by the ethics committee of Hacettepe University Hospital.

The study groups included 13 healthy volunteers, 12 HBsAg carriers, 19 patients with chronic hepatitis B, and 28 patients with definite diagnosis of autoimmune liver disease (ALD) (11 autoimmune hepatitis, 12 primary biliary cirrhosis, 5 primary sclerosing cholangitis). Patients with decompensated cirrhosis and HCC were excluded. Data on healthy controls was published earlier.\(^3\) HGF was measured in serum samples stored at -20°C by using *Biosource* ELISA (International Immunoassay kit, California, USA). *Immulite* ELISA (2000 AFP, Los Angeles, USA) was used for determining serum AFP levels.

Kruskal-Wallis and \(\chi^2\) test were done for statistical analysis by using SPSS 10.0 program. \(p<0.05\) was considered significant.

The mean (SD) age for the whole study group

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was 40.7 (13.5) years; healthy volunteers (33.1 [7.3] y) were younger than those with ALD (47.3 [14.7] y; p=0.011). The other groups were similar in age. Ten of 13 healthy volunteers, 25 of 28 ALD patients, 6 of 12 HBsAg carriers, and 3/12 with chronic hepatitis B were women. The other autoimmune diseases associated with ALD were Type 1 DM (2 patients), vitiligo (2), ulcerative colitis (2), Hashimoto thyroiditis (1), atrophic gastritis (1), scleroderma (1) and Sjogren syndrome (1).

All subjects had serum AFP levels less than 20 IU/mL; the mean AFP level for the whole group was 2.77 (1.61) IU/mL. There was no difference between groups for AFP levels. Serum HGF levels ranged from 0.54-4.53 ng/mL (mean 1.19 [0.72]), and were similar in liver diseases groups (ALD 1.31 [0.84], chronic hepatitis B 1.31 [0.90] ng/mL) and others (healthy volunteers 0.88 [0.17], HBsAg carriers 0.7 [0.19] ng/mL).

This study did not show any difference in serum HGF levels between patients with ALD and HBV-associated chronic hepatitis, which have respectively low and high risk for HCC development. HGF is known to be the most potent trigger of liver regeneration and its serum levels increase in acute and chronic liver diseases and HCC. The matrix injury during acute liver injury releases plasminogen, which converts pro-HGF activator (pro-HGFA) to active HGF. HGFA cleaves pro-HGF to activate and stimulate hepatocyte regeneration. Unlike acute liver disease, the high serum HGF levels in cirrhosis are not due to increased secretion but due to abnormal interaction between hepatocytes and matrix resulting in decreased HGF elimination. HGF is synthesized by either cancer cells or cells of the immune system infiltrating the tumor in HCC; high serum level of HGF is a well known diagnostic and prognostic marker for HCC.1,2,7

Studies with larger patient groups are needed in order to determine the role of HGF as a marker for carcinogenetic state in cirrhosis of different etiologies.

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Point prevalence of hepatitis B in mother-child dyads in a stratified random sample in an urban resettlement community in Delhi

There have been no community studies, employing epidemiologically valid sampling techniques, looking at prevalence of hepatitis B in India1. We did this study to see how many mothers and children were HBsAg positive, and to estimate the number of children who can be protected by selective immunization of HBsAg positive mothers.2

A cluster of approximately 20,000 households with a population of about 100,000 persons in a resettlement colony in East Delhi was selected. 600 households were chosen by stratified random method. All 302 households with children-under-5 years were approached. Written informed consent was obtained. Mother and their children were considered as dyads for analysis. Blood was tested using HEPACARD HBsAg spot test. (Hepaocard; J Mitra, New Delhi: Test sensitivity 99%, specificity 100%) Every positive sample was tested a second time for confirmation. The study had the approval of the hospital research committee.

156 households with 242 children consented to participate and provided blood samples; 17 samples
were lost so 148 mothers and 231 children were tested. The group that participated in the study was similar to the non-participants in economic and religious groupings.

Five mothers were HBsAg positive (point prevalence 3.38% [proportion 0.034; 95% CI 0.015-0.077]).2 3 children were positive (point prevalence 1.30% [proportion 0.013; CI: 0.004-0.037]). The 5 mothers who tested positive had 9 children, of whom 3 were positive. All these 3 were children of the 5 HBsAg-positive mothers.

Three of 9 children of HBsAg-positive mothers presumably got infection vertically. The anti-HBs status of children was not tested; so it is not clear how many others acquired the infection and cleared it subsequently.

A study by Nayak et al.,3 like our study, suggested that 33% of chronic carriers in India get the infection from their mothers. Immunization at birth targeted at babies of HBsAg-positive mothers will reduce this 33% of infection and also horizontal spread from this group. Another study also looking at mother-child dyads but not utilizing random sampling techniques, recruiting 400 children and their mothers, found that 2.25% of children below five were HBsAg positive. The authors concluded that vertical transmission is responsible for the majority of chronic carriers.

Cost constraints in India stand in the way of universal immunization at birth. Other alternatives are universal immunization starting at 6 weeks or selective immunization at birth to babies of HBsAg-positive mothers.5,6 Immunization at 6 weeks will not protect against vertical transmission. Also, coverage with immunization is unlikely to be complete; those who are not immunized are at risk from the pool of children who get the infection vertically.

A systematic review of world literature found that no study has demonstrated that the carrier rate can come down with immunization starting after 6 weeks.7 Further community-based studies are needed before a final answer is available.

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Differences in frequency of colonic polyps in different ethnic groups in the United Kingdom

Colorectal cancer rates vary considerably geographically, with age-standardized, incidence rates per 100 000 men for colon and rectum combined at 49.3 in Japan, 44.4 in North America, 42.9 in western Europe, and 3.1 in India.1,2 Studies on migrant populations suggest that environmental influences affect the epidemiology, and incidence rates of cancer in immigrants and their descendants demonstrate a relatively rapid convergence of risk to that of the host country.3 Few studies have examined the ethnic variation in polyp frequency, and none within the UK.

We performed a retrospective colonoscopy database audit in our unit, which is a large teaching hospital based in South West London. The local population comprises a wide cultural and racial mix and we investigated the ethnic differences between the frequency of colonic polyps in the local Indian sub-continent Asians and all other ethnic groups.

Colonoscopy records for the period 1992 to 2004 were retrieved and patients of Indian subcontinent Asian origin were identified by surname and forename. This
method has previously been shown to have acceptable sensitivity and specificity in the identification of Asian ethnic origin. A previous study in our institution demonstrated that this method matched well with the ethnicity recorded on the hospital database.

Analysis was limited to the first colonoscopy and all those undergoing surveillance colonoscopy were excluded; this gave an initial population of 8307 patients. Of these, we compared patients with colonic polyps as compared to other findings. Patients in whom the indication for colonoscopy was a family history of colorectal cancer were excluded so as not to include any genetic confounding factors (33 Indian subcontinent Asians and 857 other ethnic groups), giving a study population of 7417.

Indian subcontinent Asians were significantly younger than other ethnic groups (mean age 54.7 v 69.8 years, p<0.0001). Fewer Indian-subcontinent Asians were colonoscoped for a positive family history than other ethnic groups (4.6% v 11.3%, p<0.0001). Fewer subcontinent Asians of both sexes had polyps than other ethnic groups (21.0% vs 28.1%, respectively; p<0.0001), this effect was more marked in younger subjects (Table).

This large, endoscopy-based study is the first to describe ethnic differences in the frequency of colonic polyps. We demonstrated that fewer Indian-subcontinent Asians had polyps compared to other ethnic groups, and this effect is greater in younger subjects.

Colorectal cancer is uncommon in India, and it would therefore be expected that fewer Indian-subcontinent Asians would have polyps. Further support comes from the observation that fewer Indian-subcontinent Asians underwent colonoscopy due to a family history of colorectal carcinoma.

Further studies are warranted to confirm our findings; to investigate whether the incidence of polyps and colorectal cancer in this group of subjects increases with time; to further analyze polyp status for site and histological type and to investigate possible protective factors in the Indian sub-continent Asian diet and lifestyle.

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### Autoimmune hemolytic anemia and erythroid hypoplasia associated with hepatitis E

Hemolysis has been reported in association with hepatitis A and B, and rarely with hepatitis E. We report a 25-year-old lady who developed autoimmune hemolytic anemia in association with hepatitis E, which responded spontaneously.

A 25-year-old lady presented with nausea, vomiting, decreased appetite, fever, and jaundice of 20 days' duration. Examination revealed jaundice and hepatomegaly (2 cm below costal margin). Hemogram and peripheral smear examination were normal. Serum bilirubin level was 10 mg/dL (direct 6), AST 745 IU/L, ALT 538 IU/L, alkaline phosphatase normal. Antibodies to hepatitis E (IgM) were positive and markers for hepatitis A, B, C, parvovirus and cytomegalovirus were negative. With supportive therapy she improved, but 14 days later developed marked pallor and increasing jaundice. Repeat hemoglobin was 4.2 g/dL; WBC and platelet counts were normal. Peripheral smear showed spherocytes and marked polychromasia. Serum bilirubin was 26.6 mg/dL (direct 10), with AST 106 IU/L, ALT 63 IU/L. Plasma hemoglobin level was raised and urine hemosiderin was positive. Coomb's test (direct and indirect) was positive. Reticulocyte count was 1.4%; bone marrow biopsy revealed marked paucity of erythroid precursors. Tests for G6PD deficiency, lupus anticoagulant (LAC), anti-nuclear antibodies, double-
stranded DNA, and anticardiolipin antibodies were negative. Steroid was not started for the autoimmune hemolytic anemia because of acute hepatitis E, and hemoglobin remained steady around 5 g/dL without blood transfusion. Two weeks later, hemoglobin was 11 g/dL, reticulocyte count was 9.1% and serum bilirubin level was 1.6 mg/dL, (direct: 0.9). At the time of discharge, Coomb’s test was negative. On follow up at 3 months, test for IgM anti-HEV was negative.

Hemolysis in association with hepatitis A has been recorded previously, either alone or in association with other viruses and concomitant G6PD deficiency. Steroids were used in one patient, who subsequently died of renal failure. Hepatitis E was associated with severe hemolysis and renal failure in five patients with G6PD deficiency. However autoimmune hemolytic anemia has not been reported with hepatitis E. Our patient did not have any other predisposing factors for hemolysis. Steroid therapy has been used in such cases with gratifying results without evidence of reactivation of viral infection. Our patient improved without steroid treatment.

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Sugar application in reduction of incarcerated prolapsed rectum

Incarceration, strangulation, ulceration and hemorrhage may occur rarely in rectal prolapse. We report two patients with incarcerated rectal prolapse in whom we applied table sugar to help in reduction.

A 36-year-old woman noticed a mass protruding per anum during defecation, after which she could not pass flatus or defecate for two days. There was a 15-cm irreducible complete rectal prolapse. Digital replacement of the rectal prolapse was unsuccessful even under general anesthesia. We then applied approximately 25 g of ordinary sugar granules on the mucosal surface of the prolapsed rectum. Three minutes later the edema decreased dramatically and the mass became manually reducible. On digital rectal examination the tone of the sphincter was loose. We performed Thierch procedure by using prolene mesh.

A 34-year-old pregnant woman was admitted with uterine contractions at 40 weeks of gestation. She had severe rectal pain and could not pass flatus or defecate. A 10-cm irreducible rectal prolapse was seen. Since we were unable to reduce this manually, approximately 20 g of sugar granules was applied to the mass. The edema dissolved in five minutes and the mass was reduced manually. The patient delivered by caesarean section.

Reduction of incarceration can be difficult because of severe edema and ischemia. Local anesthesia, ice application, injection of dilute adrenaline or hyaluronidase is effective in reducing edema. If these techniques are unsuccessful reduction under general anesthesia has been tried. If the prolapsed segment remains irreducible, necrosis of the rectum may occur, and an emergency resection must be performed.

Myer and Rothenberger reported that application of sugar was a known procedure among veterinarians in the treatment of prolapse in cats, dogs and horses. It facilitated the reduction of incarcerated colostomy, ileostomy and rectal prolapses. In addition, the authors occasionally used this method in prolapsed, non-thrombosed, edematous internal hemorrhoids. We applied this technique successfully in
two patients with incarcerated rectal prolapse.

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Arterioportal fistula presenting as ascites

We report a 74-year-old lady who presented with progressive abdominal distension since two months. There was no history of jaundice, GI bleed, abdominal pain, fever, pedal edema, or breathlessness. She had been involved in a road traffic accident 15 years earlier. General examination was unremarkable except for mild pallor. Abdominal examination revealed splenomegaly, moderate ascites and a bruit over the right hypochondrium. Laboratory investigations were normal except for mild anemia. Diagnostic paracentesis of ascitic fluid showed low-protein, high-SAAG ascites, suggestive of portal hypertension. CECT scan revealed ascites and a normal liver, with early enhancement of portal vein in the arterial phase, suggestive of a left lobe hepatic artery-portal vein fistula. Upper GI endoscopy revealed small isolated gastric varices and mild portal gastropathy. Diagnostic laparoscopy was normal and liver biopsy done did not reveal evidence of cirrhosis. She was initially managed with diuretics and paracentesis of ascitic fluid; however the ascites was refractory to treatment.

She underwent celiac and superior mesenteric angiography, which confirmed the fistula (Fig). Following gel foam embolization of the fistula, her ascites resolved completely and she could be discharged without diuretics. She had recurrence of ascites at 1 month. Repeat angiography showed partial persistence of fistula. Ascites resolved completely on repeating the embolization. She had no further recurrence of ascites 1 year later.

Arterioportal fistulas (APF) rarely present as ascites, the usual presentation being with gastrointestinal variceal bleeding. Their inflow is usually from the hepatic artery (65%), splenic artery (11%) or superior mesenteric artery (10%). APF may be classified into type 1 (small peripheral asymptomatic fistulas with minimal physiological insult), type 2 (larger central fistulas causing physiological insult), and type 3 (congenital fistulas). APF may produce no symptom or may present with complications of portal hypertension, intestinal ischemia, or heart failure. Most cases are due to chronic liver disease, hepatic trauma, or in association with malignancy. The only clue to the likely cause in this case was a remote history of trauma. Early filling of the portal vein on arterial phase and the presence of wedge-shaped, transient peripheral areas of enhancement during arterial phase are typical findings on CT. Arteriography is the gold standard in diagnosis. Arterial embolization is very effective; steel coils, alcohol, gel foam particles, isobutyl cyanoacrylate, angioplasty, balloon tamponade, and detachable balloons have been used. Hepatic artery ligation or fistula resection, segmentectomy or lobectomy are other treatment options.

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Gastrointestinal mucormycosis in a neonate: survival without antifungal therapy

Gastrointestinal mucormycosis (GIM) is an extremely rare and usually lethal disease entity. Only six survivors have been reported in the neonatal age group.1-4 We report a 16-day-old male neonate with colonic perforation due to mucormycosis who survived without antifungal treatment.

A full-term male neonate with birth weight of 3.2 Kg required exchange blood transfusion for hyperbilirubinemia. He developed abdominal distension and obstipation on 14th day of life. X-ray abdomen revealed pneumoperitoneum. On exploratory laparotomy, there was 1.5 cm x 1.5 cm perforation of sigmoid colon with fecal peritonitis. Rest of the gut was healthy looking without any morphological evidence of necrotizing enterocolitis (NEC). Resection of perforated colon and proximal colostomy was done.

Post operative course was uneventful and patient was discharged on 8th postoperative day. Blood culture and intra operative peritoneal fluid samples were sterile on culture. Subsequently biopsy report revealed broad, aseptate, branching hyphae suggestive of mucormycosis infiltrating the gut wall and blood vessels (Fig). On follow up, after 3 weeks of discharge he was thriving well. Colostomy closure was done at 5 months of age and patient was doing well at 1 year follow up.

In contrast to adults, where stomach is most commonly involved organ, neonatal GIM predominantly involves colon, followed by stomach, ileum, rarely esophagus and appendix.3 Most patients present as necrotizing enterocolitis (NEC).4 GIM can be differentiated from NEC by the absence of pneumatosis intestinalis, wide spread small vessel thrombosis and poor response to usual chemotherapy given for NEC.5

GIM is associated with high mortality. In a literature review only six neonatal survivors had been reported till date.1-4 Aggressive surgical debridement of devitalized tissue augmented by I.V amphotericin B after confirmation of diagnosis has been suggested as the main stay of treatment.4 Surprisingly on reviewing the management of the survivors, we noticed that three of seven neonates (including present one) survived without amphotericin B therapy.2,4 All three survivors had localized gastrointestinal pathology without systemic manifestations of mucormycosis. We think that the patient’s immunological status and virulence of organisms may play an important role in determining localization of disease and survival of the patient.

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