Laparoscopy, histology and tissue culture are the most useful diagnostic methods in patients with ascites. These methods are uneconomic, expensive and need experienced specialists. CA-125 is a tumor marker that is often elevated in patients with ovarian cancer and peritoneal malignancies. On the other hand, in patients with tuberculous peritonitis, there is an increase in the serum level of CA-125.

CA-125 levels were assessed in serum and ascitic fluid of 53 consecutive patients with ascites of different etiologies. The study was conducted at hospitals affiliated to the Ahwaz University of Medical Science between 2000 and 2002. Patients were divided into four groups: a) tuberculosis peritonitis, all with laparoscopy and confirmatory pathology (n=9); b) malignancy, prior to therapy (n=6) (one lymphoma, 2 ovary cancer, 2 adenocarcinoma of stomach, one colon cancer); c) cirrhosis (n=33) of various etiologies with no evidence of hepatoma or other malignancy; and d) nephrotic syndrome (n=5) of various etiologies. The control group consisted of 29 patients (19 males) without ascites, malignancy, cirrhosis or proteinuria.

One blood sample and one sample of ascitic fluid from each patient were tested for CA-125 (ELISA, CAN-Antigen, Sweden; normal level up to 35 U/mL). In patients with tuberculosis peritonitis serum test for CA-125 was repeated after completion of treatment. Data were analyzed using SPSS for Windows (version 10.0.1).

In patients with tuberculosis peritonitis, CA-125 was high in all serum samples and 8 of 9 samples of ascitic fluid (Table). Serum CA-125 level was normal after anti-tubercular treatment. In cirrhotic patients, serum and ascitic fluid level of CA-125 was higher than normal in a majority of cases. In patients with malignancy, CA-125 level was increased in all 6 serum samples and 4 of 6 ascitic samples. The serum and ascitic fluid level of CA-125 was normal in both control and nephrotic syndrome groups, except for one case in the latter group (Table). The rise in CA-125 level in serum and ascitic fluid of patients with cirrhosis, tuberculosis, and malignancy was significant in comparison to the control group and patients with ascites related to nephrotic syndrome (p=0.001).

In a previous study on patients with tuberculous peritonitis, the average serum level was 316 u/mL. Levels decreased after anti-tubercular treatment. The dialysis fluid of patients undergoing peritoneal dialysis also showed high level of CA-125. CA-125 level in patients undergoing abdominal surgery is higher than in patients undergoing other surgeries. The level in serum and ascitic fluid of patients with Meig’s syndrome was also reported to be high and recovered after treatment.

We believe that the peritoneum is a probable source of CA-125 production; any factor that stimulates production of ascitic fluid can lead to increase in this tumor marker.

The near-normal CA-125 level in nephrotic syndrome could be because of urinary excretion of this glycoprotein. CA-125 can be used for evaluation of response to anti-tubercular treatment.

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References


Table: Level of CA-125 (u/mL) in serum and ascitic fluid of patients with ascites

<table>
<thead>
<tr>
<th>Groups</th>
<th>No.</th>
<th>Sample</th>
<th>Median (range)</th>
<th>No. above control cut-off</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cirrhosis</td>
<td>33</td>
<td>Serum</td>
<td>437 (9-1054)</td>
<td>28</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Ascitic</td>
<td>380 (9-1500)</td>
<td>31</td>
</tr>
<tr>
<td>Malignancy</td>
<td>6</td>
<td>Serum</td>
<td>202 (10-436)</td>
<td>6</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Ascitic</td>
<td>411 (55-1015)</td>
<td>4</td>
</tr>
<tr>
<td>Nephrotic syndrome</td>
<td>5</td>
<td>Serum</td>
<td>16 (9-27)</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Ascitic</td>
<td>11 (8-74)</td>
<td>1</td>
</tr>
<tr>
<td>Tuberculosis peritonitis</td>
<td>9</td>
<td>Serum</td>
<td>438 (42-1054)</td>
<td>9</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Ascitic</td>
<td>6101 (178-1200)</td>
<td>8</td>
</tr>
<tr>
<td>Control</td>
<td>29</td>
<td>Serum</td>
<td>8 (3-35)</td>
<td>-</td>
</tr>
</tbody>
</table>


**Prevalence of celiac disease in first-degree siblings of celiac disease patients**

The prevalence of celiac disease (CD) in India is not known as large epidemiological studies are not available, but a study from northern India suggests prevalence similar to Western countries. Estimates of risk to siblings have ranged from <5% to >20%. 2,3

Twenty-four patients with CD with age <15 years attending our outpatient department between April 2005 and December 2005 were included in the study. The diagnosis of CD was made by the modified European Society of Paediatric Gastroenterology and Nutrition (ESPGAN) criteria and positive serology for IgA anti-tissue transglutaminase (tTG).

We included patients who had one or more siblings, all of whom were willing to participate in the study. All siblings were screened by IgA tTG antibody estimation ELISA (Autoimmune Diagnostic Assay, Germany) and those found positive were subjected to clinical, anthropometric and biochemical evaluations. Duodenal biopsy was performed in siblings who were positive for serology and gave consent for the same. Institutional ethical committee clearance was also obtained.

The mean age of the index cases was 8.0 (SD 3.2) years (15 males). Sixty first-degree siblings were screened for IgA tTG antibody, of which 14 (23%) were positive. Duodenal biopsy was done in 9 consenting subjects and all showed histological changes characteristic of CD (Marsh grade III).

The mean age of serologically-positive siblings was 10.1 (5.7) years (7 males). Nine of the 14 siblings were asymptomatic; 5 had pallor, one had diarrhea and 3 had growth retardation. Three index cases had more than one sibling who were positive for IgA tTG antibody.

In a large study from the US, the prevalence of CD was 4.5% in first-degree relatives and 2.5% in second-degree relatives. 4 In a Finnish study of 380 patients with CD and 281 with dermatitis herpetiformis, the prevalence of CD in first-degree siblings was 7%. 5 In another study the prevalence of CD in first-degree relatives was 8.3%. 6 The exact prevalence of CD in the general population and in first-degree siblings of CD in India is not known. We found that 14 (16%) of 60 siblings were positive for serology. Biopsy was characteristic of CD in all 9 patients in whom it was done. Our data suggest that the prevalence of CD in first-degree siblings in India is similar to that in the West. Larger studies are required to confirm these findings.

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**References**


**Concordance between endoscopic and histological gastroesophageal reflux disease**

The gold standard for diagnosis of erosive Gastroesophageal reflux disease (GERD) is upper gastrointestinal endoscopy while there is presently no gold standard for the diagnosis of non-erosive GERD (NERD). 1 Though 24-hour esophageal pH monitoring can confirm the diagnosis of GERD, it is not widely available. Patients without obvious esophageal erosions are treated with a two-week course of proton pump inhibitor and their symptom response evaluated (PPI test). Histology does not appear to play a significant role in the diagnosis of GERD. 2,3
Eighty-one patients (median age 49 years, range 13-80; 42 male) who had reflux symptoms and were referred for upper GI endoscopy were recruited into the study. History of treatment with acid-suppressive drugs was noted and consent taken for esophageal biopsy. At endoscopy esophageal mucosa was assessed by trained endoscopists, and mucosal breaks classified using the LA classification. Biopsies taken at 3 cm above the gastroesophageal junction were evaluated by a single pathologist, blind to the clinical findings. A histological diagnosis of GERD was made when there was coexistence of basal cell hyperplasia greater than 15% of mucosal thickness and papillary height greater than 50% of mucosal thickness. Alcian blue/periodic acid Schiff (AB-PAS) stain was used to delineate the basal layer of the squamous epithelium (which is glycogen-depleted) and to demonstrate the presence of intestinal metaplasia in Barrett’s esophagus.

The predominant symptom was retrosternal pain (66 subjects), followed by indigestion (54). Classical reflux symptoms of heartburn and regurgitation were elicited in 43.2% (35) and 38.3% (31) of subjects, respectively. Regurgitation was the only symptom that correlated significantly with endoscopy-positive GERD (Fisher’s exact test, p=0.006).

Thirty-six of the 81 subjects (44.4%) had erosive GERD with a majority having mild grades, i.e., LA grade A – 25 subjects, grade B – 7 subjects, grade C – 1 subject and grade D – 3 subjects. The esophageal mucosa was endoscopically normal in 39 (48.1%) subjects. Other findings included Barrett’s esophagus (1 patient), white patches on esophageal mucosa (2), benign stricture (1), irregular Z line (1) and esophageal web (1). Hiatus hernia was present in 4 cases.

Histological GERD was diagnosed in 33.3% (27/81) of subjects. Less than half (15/36) the number of patients with erosive esophagitis showed histological evidence of GERD. In LA grade A, 9 of 25 subjects had histological GERD; grade B – 3 of 7 subjects; grade C – 1 of 1 subject and grade D – 2 of 3 subjects. In contrast, 12/45 subjects (26.7%) with non-erosive esophageal mucosa had evidence of histological GERD. Sixteen of 44 patients with classical reflux symptoms had evidence of histological GERD. The present study reports a 4.9% (4/81) prevalence of Barrett’s esophagus by histology, which is slightly higher compared to other studies. One of the 2 patients who had white patches on endoscopy had Candida infection. Two of the four cases with hiatus hernia had histological evidence of GERD.

There was no significant difference in the presence of histological GERD between patients who had previous treatment with acid-suppressive agents and those who did not (Fisher’s exact test, p=0.06).

One of the major findings in this study is the poor concordance between erosive GERD and histological evidence of GERD, throughout all the grades of the LA classification (k value of 0.04 to 0.07). When all LA grades were grouped together as ‘endoscopic GERD’ and normal endoscopy was classified as ‘no GERD’, the agreement between endoscopy and histology remained poor (k value 0.16). The concordance between endoscopy and histology was poor (k value 0.025) even taking into consideration only patients with classical symptoms of GERD.

In conclusion, most patients in the present study suffered from NERD or mild erosive esophagitis. The macroscopic appearance of esophageal erosions did not correlate with classical histological features of reflux esophagitis. Conversely, a normal-looking esophagus was not proof of histologically normal mucosa.

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References


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Persistence of anti-HBs titers after two different doses of Genevac B, a recombinant hepatitis B vaccine, in healthy adolescents

We compared the immunogenicity, safety and persistence of anti-HBs titers after two different doses of a new recombinant hepatitis B vaccine, Genevac B (Serum Institute of India), in healthy adolescents.

School children studying at the Corporation Higher Secondary School, Adyar, Chennai were recruited after obtaining written informed consent from their parents. Healthy adolescents aged 11-19 years of either sex; tested negative for HBsAg, anti-HBs, anti-HBc IgM; and with no history of previous vaccination against hepatitis B were included in the trial. The exclusion criteria were: active moderate or severe illness; previously diagnosed hepatitis B positivity; hepatomegaly and/or splenomegaly; uncontrolled coagulopathy; known immunological deficiency including HIV infection; treatment with immunosuppressors including corticosteroids; chronic illness like epilepsy; evidence of skin diseases or infection at any site.

Two hundred healthy adolescents were recruited and randomly allocated as 100 subjects each to receive 1 mL doses (20 µg) and 0.5 mL doses (10 µg) of Genevac B vaccine. The vaccine was administered in the deltoid region at 0, 1 and 6 months. Following administration of each dose, the subjects were observed for adverse events. Blood samples collected one month after each dose were assayed for quantitative levels of anti-HBs (Monolisa Anti-HBs [BIORAD] 3.0; Sanafi Pasteur). Seroconversion and seroprotection rates were defined as anti-HBs titer <10 mIU/mL and >10 mIU/mL levels, respectively.

On completion of the seventh month and after one year, 94 children receiving the 20 µg dose and 96 receiving the 10 µg dose followed up. The seroconversion rates on completion of the vaccination were 100% in both groups (Table). The anti-HBs levels achieved (GMT) were 2628.98 mIU/mL and 1372.80 mIU/mL, respectively (p>0.05). The GMT dropped at one year after vaccination to 2261.7 mIU/mL and 1038.5 mIU/mL, respectively. Pain at the site of injection and fever were the only symptoms, seen in equal numbers in both the groups.

We conclude that Genevac B boosts good immune response even with 10 µg dose in healthy adolescents. Vaccination with 10 µg of this lost-cost vaccine can provide an effective, economical alternative to the 20 µg dose normally recommended for healthy adolescents.

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Department of Microbiology, Dr A L M Post Graduate Institute of Basic Medical Sciences, University of Madras, Taramani, Tamil Nadu 600 113

Table: Seroconversion and seroprotection rates after hepatitis B vaccination

<table>
<thead>
<tr>
<th>One month after</th>
<th>One year after</th>
</tr>
</thead>
<tbody>
<tr>
<td>1st dose 2nd dose 3rd dose</td>
<td>3rd dose</td>
</tr>
<tr>
<td>20 µg</td>
<td>20 µg</td>
</tr>
<tr>
<td>Seroconversion</td>
<td>46.8% 94.6% 100% 100%</td>
</tr>
<tr>
<td>Seroprotection</td>
<td>19.1% 86.1% 100% 100%</td>
</tr>
<tr>
<td>Anti-HBs GMT (mIU/mL)</td>
<td>Anti-HBs GMT (mIU/mL)</td>
</tr>
<tr>
<td>10 µg</td>
<td>10 µg</td>
</tr>
<tr>
<td>14.47 94.30</td>
<td>26.28 2261.7</td>
</tr>
<tr>
<td>37.5% 92.7% 100% 100%</td>
<td></td>
</tr>
<tr>
<td>10.4% 70.8% 100% 100%</td>
<td></td>
</tr>
<tr>
<td>13.8 48.3</td>
<td>1372.8 1038.5</td>
</tr>
</tbody>
</table>

Reference

Acknowledgement: We thank M/s Serum Institute of India, Pune, for financial assistance to conduct this study
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Randomized, comparative study of cefotaxime 2 versus 4 grams in spontaneous bacterial peritonitis

The bacterial load in spontaneous bacterial peritonitis (SBP) is low when compared with other infections. The MIC values of commonly used antibiotics are lower than those for other infections.1 The elimination of cefotaxime and its active metabolites decreases in cirrhotic patients, due to functional renal failure.2 We conducted a randomized, prospective trial, from June 2003 to July 2004 comparing two dose schedules of cefotaxime. Thirty-six episodes of SBP were diagnosed on hospital admission in 36 cirrhotic patients. SBP was diagnosed on the basis of ascitic fluid polymorphonuclear leukocytes (PMN) count over 250/mm³ in the absence of features suggesting secondary peritonitis. Patients taking antibiotics within
2 weeks prior to hospital admission and those with history of β-lactam antibiotic hypersensitivity were not included. Patients were randomized at admission to receive 2 grams (n=19) or 4 grams (n=17) cefotaxime per day. Infection was considered resolved when clinical signs of infection had disappeared, peripheral white blood cell count had normalized, ascitic fluid PMN count had decreased to <250/mm³, and ascitic fluid cultures became negative if positive at admission. In patients with hepatorenal syndrome (HRS), cefotaxime dosages were adjusted according to renal functions.

All patients gave informed consent to participate in this study.

Continuous data are presented as mean (standard deviation) unless otherwise specified. Comparison of continuous variables between groups was performed by the independent t test and chi-square test was used for comparison of categorical variables.

The two groups were matched in all characteristics except international normalized ratio (INR) and ascitic fluid culture positivity (Table). Causative bacteria were isolated in ascitic fluid of 12 cases, including E. coli in six.

There was no significant difference between the groups in relation to duration of antibiotic treatment, rate of decrease in ascites PMN count, infection resolution, development of HRS, encephalopathy, and mortality (Table). Nine patients died in hospital. The causes of death were gastrointestinal bleeding (3 cases), HRS (3), sepsis (1), terminal liver failure (1), hemopneumothorax (1), and hypernatremia (1). Death of one case was attributed to both gastrointestinal bleeding and HRS. There was no adverse event related to the antibiotic administration.

The results of this study suggest that cefotaxime in a dosage of 1 g per 12 hours is as effective as the recommended dosages for the treatment of SBP. The mortality rate in our study is similar to that in other studies.

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References

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Is laparoscopic appendectomy contraindicated in mucocele of appendix?

The article by Rangarajan et al is perhaps the largest case series on laparoscopic appendectomy for mucocele of appendix. We would like to raise some issues regarding this report. The available literature does not favor laparoscopic appendectomy for mucocele, which is considered a benign tumor of the appendix. Gonzalez et al have mentioned laparoscopic appendectomy to be contraindicated in mucocele and Butcher et al have reported that laparoscopic appendectomy may be associated with inadequate resection of appendiceal tumors. The evidence provided by Rangarajan et al seems to contradict such skepticism.

The experience of the operating surgeon be-

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Table: Baseline characteristics and complications in the two treatment groups

<table>
<thead>
<tr>
<th></th>
<th>Group I (n=19)</th>
<th>Group II (n=17)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>50.6 (12.0)</td>
<td>46.5 (17.9)</td>
<td>ns</td>
</tr>
<tr>
<td>Sex (female/male)</td>
<td>11/8</td>
<td>5/12</td>
<td>ns</td>
</tr>
<tr>
<td>CTP score</td>
<td>10.8 (2.2)</td>
<td>12.1 (2.3)</td>
<td>ns</td>
</tr>
<tr>
<td>MELD score</td>
<td>11.9 (10.2)</td>
<td>18.6 (12.3)</td>
<td>ns</td>
</tr>
<tr>
<td>International normalized ratio</td>
<td>1.7 (0.5)</td>
<td>2.3 (0.8)</td>
<td>0.01</td>
</tr>
<tr>
<td>Ascites culture positivity</td>
<td>3 (15.8%)</td>
<td>9 (52.9%)</td>
<td>0.018</td>
</tr>
<tr>
<td>Duration of antibiotic (days)</td>
<td>5.5 (1.6)</td>
<td>5.6 (2.4)</td>
<td>ns</td>
</tr>
<tr>
<td>Infection resolution</td>
<td>17 (89.5%)</td>
<td>14 (82.4%)</td>
<td>ns</td>
</tr>
<tr>
<td>HRS</td>
<td>5 (26.3%)</td>
<td>6 (35.3%)</td>
<td>ns</td>
</tr>
<tr>
<td>Deaths</td>
<td>3 (15.8%)</td>
<td>6 (35.3%)</td>
<td>ns</td>
</tr>
</tbody>
</table>

ns: non significant. HRS: Hepatorenal syndrome, PMN: polymorph nuclear leukocytes.
Beyond the learning curve is very important when dealing with fragile tissue like mucocele of the appendix, due to the risk of rupture and subsequent pseudomyxoma peritonei. This article fails to highlight this prime fact.

Secondly, the authors define mucocele as “aseptic dilatation of appendix secondary to obstruction”. To the best of our knowledge, appendiceal diseases are not considered aseptic due to exposure to intestinal flora; we would like to know the basis for this definition.

The authors note that one patient was excluded from the series due to rupture of appendix after diagnostic laparoscopy, leading to open appendectomy. Is diagnostic laparoscopy a routine for every suspected appendicitis in the author’s unit? If the diagnostic laparoscopy had not complicated this case, would the patient get a laparoscopic appendectomy? If the answer to either of these is yes, this patient should have been included in the study.

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References

Reply from the authors

There are many reports that favor laparoscopic excision for mucocele, and mostly case reports that state that laparoscopy is contraindicated in mucocele. If wound protector or non-permeable endobag is used to deliver the specimen, recurrence can be avoided. The resection was adequate in all our cases. Currently, laparoscopic esophagogastrectomies, gastrectomies and colectomies are being done routinely by several centers, including ours. These procedures carry an even bigger risk of tumor seeding and recurrence, but with proper precautions, this can be avoided.

Our article defined mucocele as “a collection of mucus within the appendiceal lumen secondary to obstruction. Initially, it is sterile and secondary infection may set in if treatment is delayed”.

We agree that the experience of the surgeon is vital in this procedure. Dr. Palanivelu has performed all the surgeries in this series and has vast experience with laparoscopic surgery.

The last few lines in ‘Discussion’ highlight the importance of handling the specimen properly. It should be clarified that a mucocele handled by the surgeon’s hand during an open procedure can also rupture. Bucher et al admit that the outcome and prognosis of patients treated for appendiceal neoplasms are comparable after laparoscopic and open appendectomy; still, they don’t recommend laparoscopy, and we don’t see why.

Finally, our article states that “one patient underwent diagnostic laparoscopy followed by laparotomy as he had pseudomyxoma peritonei secondary to rupture of mucocele of the appendix”. The pseudomyxoma peritonei was discovered on diagnostic laparoscopy and not caused by it.

M Rangarajan

References

Pitfalls in the diagnosis of insulinoma

Jyotsna and colleagues confirm what is reported in literature, that the pre-operative diagnosis of insulinoma is still a challenge for clinicians. An additional problem, which the authors did not refer to, is the possible cystic nature of insulinomas. Although rare, such lesions can cause scant symptoms and weak laboratory evidence of hypoglycemia and hyperinsulinemia, and so may remain undetected for a long time. Because of this, the risk of malignant degeneration of cystic insulinomas might be higher than that of its solid counterpart. Imaging modalities may not be able to distinguish such a tumor from other neoplastic or non-neoplastic cysts of the pancreas.
Letters

References


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Reply from the authors

Of the 31 patients with insulinoma that we studied, none had a cystic tumor.

Viveka P Jyotsna

CT colonography versus conventional colonoscopy

I agree with Kalra et al¹ that colonoscopy is an invasive procedure with risk of perforation and hemorrhage, which varies from 0.34%-2.5%.²

The obvious advantage of colonoscopy, however, is that it can be used as a therapeutic modality as well. With false negative and false positive findings on CT colonography (CTC), its use even as a primary investigation modality is controversial. The duration for CTC and colonoscopy is more or less the same. In experienced hands, complete colonoscopy takes less than 20 minutes, with completion rate of over 94%.³

I therefore believe that CTC should be used mainly in patients with failed colonoscopy. Its role in oclusive colonic growth is controversial as the insufflation can lead to perforation. Also, there is the possibility that bowel preparation would be inadequate in an obstructing lesion, leading to high failure rates for CTC.

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References


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Reply from the authors

We agree with Dr Shah that colonoscopy has the advantage that polypectomy/biopsy can be done at the time of the procedure.

Currently the clinical indications for CTC are incomplete colonoscopic examination and colonic evaluation proximal to an obstructing neoplasm. Cecal intubation rates increase with increasing experience of the endoscopist.¹ There is no controversy regarding the role of CTC in oclusive colon growths. The overall risk of perforation with CTC is only 0.059%,² with no additional risk in patients with oclusive growths. Bowel preparation with polyethylene glycol may lead to retention of fluid in such patients. The difficulty is overcome by scanning in both supine and prone positions as it displaces the retained fluid in the colon. CTC without bowel preparation, by using fecal tagging with iodine or barium, may also be resorted to in these cases.

Another indication for CTC is evaluation of patients who are clinically unfit to undergo colonoscopy. Its utility in colon cancer screening is under evaluation. The results of trials have been varied, with the highest by-patient sensitivity of 94% for adenomas larger than 1 cm reported in the Pickhardt trial.³

N Kalra, S Suri, D K Bhasin, S K Sinha, N Saravanan, T Kour, K Vaiphei, J D Wig

References


Ciprofloxacin-induced cholestatic jaundice

We report a 26-year-old man with jaundice, anorexia and weakness of 10 days’ duration. There was no fever or
pain in abdomen. He was taking ciprofloxacin 500 mg twice daily for acute diarrheal illness. Jaundice was noticed 5 days after starting ciprofloxacin. These symptoms were preceded by a rash over the trunk that subsided with oral anti-histaminics. On examination, the patient was afebrile, icteric, and had scratch marks over the trunk, with few pigmented spots. Per abdominal examination showed non-tender mild hepatomegaly. There were no signs of acute liver failure.

Etiologic work-up showed negative viral markers (HBsAg, IgM anti-HAV, IgM anti-HEV, total anti-HBc). G6PD and serum ceruloplasmin levels were normal; KF ring was absent. His autoimmune profile (ANA, ASMA, p-ANCA, LKM antibodies) was negative. Ultrasonography showed moderate hepatomegaly; the biliary tree was normal. Liver biopsy in the second week of illness revealed bile stasis with pericholangiolar inflammatory infiltration with lymphocytes, polymorphs and occasional eosinophils. There were focal necrotic changes in the parenchyma. Serum bilirubin level gradually increased up to 43 mg/dL and then decreased over a period of 6 months. The pattern of liver injury was predominantly hepatocellular type in the early phase of illness (AST/ALT 1055/1700 IU/L at onset) but became predominantly cholestatic in the later phase.

The patient was treated with anti-histaminic (cetrizine) and ursodeoxycholic acid (1200 mg/d). Prednisolone (1 mg/Kg/d) was added in the cholestatic phase. While on tapering doses of steroids, he developed herpes zoster ophthalmicus of the right eye, from which he recovered uneventfully.

Ciprofloxacin has been listed as a potential hepatotoxic agent. Review of case reports reveals that the injury is usually idiosyncratic and is associated with other allergic manifestations like skin rash. The pattern varies from asymptomatic rise in transaminases to acute liver failure. Cholestatic liver injury occurs more commonly. Most patients recover uneventfully. Liver biopsy helps in the diagnosis if typical histological features (eosinophilic infiltration, lobular inflammation, duct destruction) are seen. It also helps in excluding drug-induced vanishing bile duct syndrome, which carries poor long-term prognosis. A few reports have suggested a role for steroids in treating drug-induced liver disease.

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