Contents

Editorials
Measuring hepatic functional reserve using MEGX still a mirage!  
S K Sarin, Manoj Kumar 203
Gastrointestinal stromal tumor - paradigm for successful targeted therapy Susy Kurian 207

Original Articles
Impact of shorter duration of treatment on virological response rate in genotype 2 or 3 chronic hepatitis C virus infection Ioannis S Elefsiniotis, Konstantinos D Pantazis, Dimitrios Dimitroulopoulos, Sotirios Koutsounas, Antonios Moulakakis, Emmanuel Paraskevas 209
Gastrointestinal stromal tumors: a demographic, morphologic and immunohistochemical study F Rauf, Y Bhurgri, S Pervez 214
Analysis of Helicobacter pylori antimicrobial susceptibility and virulence genes in gastric mucosal biopsies in the United Arab Emirates Mubarak S Alfaresi, Adeel Islam Abdul salam, Abida A Elkoush 221
Gastrointestinal stromal tumors: a single institution experience of 50 cases Senthil Rajappa, Krishna Mohan Muppavarapu, Shantveer Uppin, Raghunadharao Digu marti 225

Review
Celiac disease in India Surender Kumar Yachha, Ujjal Poddar 230

Case Series
Hydatidiarrhea Suyash Mohan, Ashish Verma, Sanjaya Saran Baijal 238

Clinico-pathology conference
A treated case of follicular lymphoma presenting with fever and diarrhea Kim Vaiphei, Pankaj Malhotra, Nidhi Sharma, Anil Kumar Narasiyappah, Subhash Chander Varma 240

Case Snippets
Endovascular management of hepatic hemorrhage and subcapsular hematoma in HELLP syndrome Chandan Jyoti Das, Deep Narayan Srivastava, Jyotindu Deb nath, Vijay Ramchandran, Sujay Pal, Pesh Sahni 244
Visceral leishmaniasis: acute liver failure in an immunocompetent Asian-Indian adult G Malatesha, Nishith K Singh, Vinay Gulati 245
Endoscopic removal of chicken bone that caused gastric perforation and liver abscess R J Mukkada, A P Chettupuzha, V J Francis, P G Mathew, S P Chirayath, Abraham Koshy, Philip Augustine 246

Letters
Colonoscopic and ileoscopy biopsies increase yield of diagnosis in chronic large bowel diarrhea with normal colonoscopy S Khanna, R Talukdar, N Saikia, S Mazumdar, S Kulkarni, J C Vij, A Kumar 250
Delta hepatitis infection in northeast India Biswa Jyoti Borkakoty, Dipankar Biswas, J agadish Mahanta 251

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contd. on page iii ...

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## Contents (contd.)

Hepatitis C virus infection and risk behaviors among injection drug users of Nagaland
Hiranya Kumar Das, Biswa Jyoti Borkakoty, Jagadish Mahanta, Gojendra Kumar Medhi, Pradeep Kumar Chelleng 253

Endoscopic removal of giant colonic lipomas
Georgia Lazaraki, Dimitrios Tragiannidis, Anestis Tarpagos, Dimitrios Tzilves, Ioannis Pilipidis, Ioannis Katsos 255

Rectal bleeding due to leech bite in a young child
Vincent Ho, Peter Boyd 256

Cecal web causing neonatal intestinal obstruction
Sushil Budhiraja 256

### Images

Colonic leiomyoma with huge ulceration
Akihiko Takeda, Shinichi Ban, Akihiro Yasumoto, Keiko Ishikawa, Hiroyoshi Iseki, Hideki Takeuchi, Norio Takahashi, Isamu Koyama 213

Gastric cancer presenting with cutaneous metastasis
George Barreto, Shailesh Shrikhande, Parul Shukla 237

Gastroenterology Elsewhere 257

India Elsewhere 258

Announcements

Indian Journal of Gastroenterology J Mitra Memorial Award 206

New and Notices 216

Index to Advertisers 220

Instructions to Contributors 259
Fulminant liver failure due to visceral leishmaniasis is very rare, and has been described only in children. We report an immunocompetent adult man who developed acute liver failure due to leishmaniasis and dramatically recovered with amphotericin-B therapy.  

[Indian J Gastroenterol 2007;26:245-246]

Indian Kala-Azar (visceral leishmaniasis – VL) is a parasitic infection of reticulo-endothelial system caused most commonly by the hemoflagellate *Leishmania donovani*. Approximately, 100,000 new cases of this disease are seen in India every year. We report an unusual case of VL with acute liver failure in an immunocompetent adult man.

A 32-year old man, non-smoker, non-alcoholic, presented with altered sensorium. He had generalized weakness since four weeks, and nausea, diffuse abdominal pain and dark urine one week prior to admission. Past history was unremarkable for significant illness, drug or vaccine exposure, allergy, substance abuse or high-risk sexual behavior. The patient had visited the state of Bihar (high endemicity zone for VL), India within the last one month. On examination he was hypotensive, febrile, pale, icteric and in confusional state without localizing signs. Subconjunctival hemorrhages, splenomegaly and minimal hepatomegaly were noted. An initial toxic and metabolic panel, cerebrospinal fluid analysis and non-contrast computed tomography of the brain were normal. Empiric broad-spectrum antibiotic cover was instituted pending blood and urine culture results, which were eventually negative. EKG, chest X-ray, and ultrasound and doppler of the abdomen were non-contributory. Tests for enteric fever, leptospirosis, malaria, HIV, viral hepatitis (A, B, C and E), brucella, dengue, Wilson’s disease.

Table: Serial laboratory data from a patient with visceral leishmaniasis

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Day 1</th>
<th>Day 25</th>
<th>Reference Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Leukocyte count (×10^9/L)</td>
<td>12.1</td>
<td>3.9</td>
<td>4-11</td>
</tr>
<tr>
<td>Hematocrit (%)</td>
<td>12.0</td>
<td>46.4</td>
<td>41-50</td>
</tr>
<tr>
<td>Platelet count (× 10^9/L)</td>
<td>20</td>
<td>312</td>
<td>150-400</td>
</tr>
<tr>
<td>Erythrocyte sedimentation rate (mm in 1st hour)</td>
<td>80</td>
<td>18</td>
<td>0.2-20</td>
</tr>
<tr>
<td>Serum urea nitrogen (mg/dL)</td>
<td>67</td>
<td>29</td>
<td>6-22</td>
</tr>
<tr>
<td>Serum creatinine (mg/dL)</td>
<td>1.2</td>
<td>0.7</td>
<td>0.5-1.1</td>
</tr>
<tr>
<td>Total / conjugated bilirubin (mg/dL)</td>
<td>6.4/4.5</td>
<td>2.4/1.3</td>
<td>0.3-1.1</td>
</tr>
<tr>
<td>Total serum protein/albumin (mg/dL)</td>
<td>5.9/1.8</td>
<td>7.6/3.1</td>
<td>6.8-3</td>
</tr>
<tr>
<td>Serum ALP (IU/L)</td>
<td>187</td>
<td>98</td>
<td>20-90</td>
</tr>
<tr>
<td>AST (U/L)</td>
<td>2633</td>
<td>97</td>
<td>5-50</td>
</tr>
<tr>
<td>ALT (U/L)</td>
<td>531</td>
<td>37</td>
<td>5-50</td>
</tr>
<tr>
<td>IgG (g/L)</td>
<td>23</td>
<td>6.5</td>
<td>1.3-2.0</td>
</tr>
<tr>
<td>Prothrombin time (seconds)</td>
<td>54</td>
<td>14</td>
<td>11-14</td>
</tr>
<tr>
<td>APTT (sec)</td>
<td>30</td>
<td>25-35</td>
<td></td>
</tr>
</tbody>
</table>

**ALP=Alkaline phosphatase; ALT=alanine aminotransferase; AST=aspartate aminotransferase; APTT=Activated partial thromboplastin time**
and auto-immune hepatitis were inconclusive. CD4 cell counts were normal. Initial blood chemistry (Table) showed bi-cytopenia and reflected marked hepatocellular injury. Light microscopy of bone marrow aspirate revealed several amastigotes within marrow macrophages. Absent hemophagocytes, and normal serum triglycerides (160 mg/dL) and fibrinogen (180 mg/dL) levels ruled out hemophagocytic syndrome. After starting intravenous amphotericin-B (initiated in a gradually increasing dose from the 3rd day to maximum of 1 mg/Kg/day), the patient made a dramatic recovery over six weeks period. He had clinical and laboratory remission, and a repeat marrow aspirate (at 22 weeks) was negative for Leishmania donovani (LD) bodies.

Milder forms of liver involvement occur in 17% of cases with kala-azar, and are structurally and functionally reversible after treatment.\(^1\)\(^2\) Severe symptomatic forms like acute hepatocellular failure are rare and reported only in children. We did not find any previous case of acute liver failure due to leishmaniasis in an immunocompetent adult.\(^1\)\(^3\)\(^4\) Pathophysio logically, liver involvement in VL is typically self-limited and involves a mononuclear cell-dominated granulomatous inflammation mediated by cytokines, chemokines and reactive oxygen and nitrogen species.\(^5\) In our patient exposure to an endemic zone, cytopenia, splenomegaly, and altered albumin/globulin ratio were the vital clues which helped us diagnose the disease.

References

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