Contents

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

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  
Gastrointestinal stromal tumors: a demographic, morphologic and immunohistochemical study  F Rauf, Y Bhurgri, S Pervez  
Analysis of Helicobacter pylori antimicrobial susceptibility and virulence genes in gastric mucosal biopsies in the United Arab Emirates  Mubarak S Alfaresi, Adeeel Islam Abdul Salam, Abida A Elkoush  
Gastrointestinal stromal tumors: a single institution experience of 50 cases  Senthil Rajappa, Krishna Mohan Muppavarapu, Shantveer Uppin, Raghunadharao Digumarti

Review
Celiac disease in India  Surender Kumar Yachha, Ujjal Poddar

Case Series
Hydatidiarrhea  Suyash Mohan, Ashish Verma, Sanjay Saran Baijal

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

Case Snippets
Endovascular management of hepatic hemorrhage and subcapsular hematoma in HELLP syndrome  Chandan Jyoti Das, Deep Narayan Srivastava, Jyotindu Debnath, Vijay Ramchandran, Sujoy Pal, Peush Sahni  
Visceral leishmaniasis: acute liver failure in an immunocompetent Asian-Indian adult  G Malatesha, Nishith K Singh, Vinay Gulati  
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

Letters
Colonoscopic and ileoscopie 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  
Delta hepatitis infection in northeast India  Biswa Jyoti Borkakoty, Dipankar Biswas, J agadish Mahanta  

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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 Pilpilidis, 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
Delta hepatitis infection in northeast India

Hepatitis D virus (HDV), a defective RNA passenger virus, infects more than 10 million of the 350 million chronically infected hepatitis B virus (HBV) carrier individuals worldwide.1 Though the incidence of delta hepatitis infection is decreasing around the world,1,2 the prevalence of HDV in many pockets of hyperendemic HBV infection is still unknown. Seroprevalence data of delta hepatitis in chronic hepatitis B virus infection from India are inadequate.

In August 2005, a study was conducted among an HBV-hyperendemic population comprising a primitive tribe of Upper Dibang valley, Arunachal Pradesh, to determine the prevalence of co-infection of HBV with delta virus. Serum samples from 93 HBsAg-positive apparently healthy subjects (EIA; EQUIPAR, Italy) were tested for anti-HDV and for hepatitis B core IgM (HBc IgM antibody, HDV ab, and HBc IgM EIA kits, EQUIPAR, Italy). Ethical clearance was obtained, and all participants gave informed and written consent.

Amongst the 93 HBsAg-positive individuals (median [range] age 17 [2-45] y), 33 (35.5%) tested positive for HDV Ab; 7 (7.5%) participants had low levels (5-20 IU/mL) of HBc IgM, suggesting chronic HBV infection. Prevalence of HDV infection was highest (17/32, 53.1%) in the 16-25 year age group. All subjects who had history of tattooing were older than 15 years; HDV infection was found in 14 of 20 (70%) of them, as compared to 19/73 (26%; p=0.0007, 95% CI 2.2-19.7) of non-tattooed subjects. In the above-15-years age group, 27/56 (48.2%) subjects were anti-HDV positive, as compared to 6/37 (16.2%; p=0.002, 95% CI 1.8-14.2) in the pediatric age group. Logistic regression showed that HDV prevalence was significantly associated only with age above 15 years age (p=0.001, 95% CI 2.3-22.58) and unmarried status (p=0.009, 95% CI 0.049-0.644).

It seems likely that superinfection rather than co-infection was the major pattern in our setting. Other studies from India among different categories of HBV-related liver disease and high-risk groups had shown a lower prevalence of HDV, from 3% to under 30%, with higher prevalence among patients with chronic liver disease and hepatocellular carcinoma.3,4,5

This study suggests that the impact of a high rate of dual infection (36%) might put liver-related morbidity and mortality at the forefront in this HBV-hyperendemic region of northeast India.

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Thrombophilic factors in Egyptian children with portal vein thrombosis

Several well-characterized hereditary thrombophilic conditions and C677T mutation in the methylenetetrahydrofolate reductase (MTHFR) gene have been implicated in the pathogenesis of vascular thrombosis. We assessed the prevalence of these conditions in 40 Egyptian children with portal vein thrombosis (PVT) (age range 1-15 [median 10.5] years); 27 were boys, and 20 age- and sex-matched controls (median [range] age 10.0 [2-15] years; 14 boys).

MTHFR C=T677 substitution was detected by polymerase chain reaction (PCR) amplification of a 198-bp DNA fragment and followed by Hinf I digestion, FVL mutation by PCR amplification and MnI1 restriction digestion analysis, and factor II 20210A allele by Hind III cleavage of 345-bp fragment amplified by PCR. Protein C, protein S and antithrombin III were measured using commercially-available functional assays (Diagnostica Stago, Parsippany, NJ, USA). Activated protein C resistance (APCR) was assessed using a commercial kit based on a modified APTT-based assay (Coatest APC resistance, DiaPharma, West Chester, Ohio, USA).

Our patients had no clinical, biochemical, or histologic evidence of liver involvement (biopsy was performed for 5 cases). Seven of our patients (17.5%) had multiple, co-existing hereditary thrombophilic conditions. The table shows the frequency of different thrombophilic factors, whether present alone or in combination, in cases and controls. FVL mutation was detected more often in patients than in controls, the relative risk of development of PVT using odds ratio was 6.0 [95% CI = 0.87-21.54; (p<0.05)]; all patients with FVL mutation had APCR. This mutation was the commonest hereditary thrombophilic condition in our cases, being present in 30%. The second most common hereditary thrombophilic condition was protein C deficiency (27.5%). Factor II mutation was found in 6 of our cases (15%).

Like in our study, Bombeli et al also found FVL mutation to be the commonest hereditary thrombophilic factor in patients with PVT, though other studies reported lower frequencies, 11.5%, 3%, and 1.6%. Amitrano et al found protein C deficiency in 26% of their patients with PVT, whereas Bombeli et al reported it in 7.1% of their patients with PVT. Since none of our patients had evidence of liver disease, protein C deficiency in them was unlikely to be related to reduced hepatic blood flow and porto-systemic shunting. Factor II mutation was found in 15% of our cases compared to none by Sharma et al. MTHFR C677T gene mutation was found in 4 (10%) of our cases (homozygous 2, heterozygous 2), a frequency comparable to that found in many healthy populations. In several studies, this mutation was not found to be a strong prothrombotic factor by itself; however, one study revealed a significant association between this mutation and the occurrence of PVT.

Multiple, co-existing hereditary thrombophilic conditions were present in 17.5% of our patients. Shah et al found multiple thrombophilic conditions in 53.8% of their patients with PVT.

In conclusion, hereditary thrombophilic conditions, particularly FVL mutation, were common in Egyptian patients with portal vein thrombosis.

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Table: Frequency of various thrombophilic conditions among children with PVT and controls

<table>
<thead>
<tr>
<th>Thrombophilia</th>
<th>Cases (n=40)</th>
<th>Controls (n=20)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Single factor abnormality</td>
<td>20 (50)</td>
<td>5 (25)</td>
</tr>
<tr>
<td>Protein C deficiency</td>
<td>8 (20)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>Protein S deficiency</td>
<td>0 (0)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>Antithrombin III deficiency</td>
<td>0 (0)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>Factor V mutation + APCR</td>
<td>7 (17.5)</td>
<td>1 (5)</td>
</tr>
<tr>
<td>Factor II mutation</td>
<td>3 (7.5)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>MTHFR C677T gene mutation</td>
<td>2 (5)</td>
<td>4 (20)</td>
</tr>
<tr>
<td>Combined factors abnormality</td>
<td>7 (17.5)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>Protein C deficiency + antithrombin III deficiency</td>
<td>1 (2.5)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>Protein C deficiency + Factor V mutation + APCR</td>
<td>1 (2.5)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>Protein C deficiency + Factor II mutation</td>
<td>1 (2.5)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>Factor V mutation + APCR + factor II mutation</td>
<td>2 (5)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>Factor V mutation + APCR + MTHFR C677T mutation</td>
<td>2 (5)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>No detectable abnormality</td>
<td>13 (32.5)</td>
<td>15 (75)</td>
</tr>
</tbody>
</table>

Values are as n (%)