Ultrasonography in malarial hepatitis

Hepatocellular jaundice occurring in patients with falciparum malaria is known as malarial hepatitis. The spectrum of hepatocellular dysfunction ranges from conjugated hyperbilirubinemia with or without elevation in serum transaminase levels, to fulminant hepatic failure. Clinically, it is sometimes difficult to differentiate malarial hepatitis from other causes of hepatocellular jaundice like viral hepatitis and hepatitis associated with other bacterial and viral infections.

Ultrasonography in acute viral hepatitis commonly shows hepatomegaly with low echogenicity and thick gall bladder wall. We studied the ultrasonography findings in patients who had malaria with jaundice.

The study was conducted on patients with Plasmodium falciparum infection with jaundice and serum bilirubin >10 mg/dL admitted in this tertiary care hospital during the epidemic of September - October 2001. The diagnosis was confirmed by demonstrating asexual form of the parasite in peripheral blood smear. Other conditions were ruled out by clinical examination and relevant tests including blood culture, test for antibody against leptospirosis, IgM anti HAV, HBsAg, IgM anti HBC and IgM anti HEV. Ultrasonographic examination of the liver and gall bladder was done after patients fasted for 12 hours, with a B-scan gray-scale analog unit, taking care to adjust the gain control appropriately, using transducer of 3.5 to 5 MHz. Measurement was made in a plane where the beam was perpendicular to the gall bladder wall.

Of 33 eligible patients only 29 (21 men) could be studied. Their mean (SD) age was 37.7 (14.5) years, total bilirubin level 19.5 (8.5) mg/dL, conjugated bilirubin 13.0 (4.4), AST 557.5 (264.8) IU/L, ALT 676.7 (303.6) IU/L, and prothrombin time 27.9 (14.2).

Ultrasonography showed hepatomegaly in 24, splenomegaly in 23, hepatosplenomegaly in 20 patients. Liver echogenicity was decreased in 7 patients, and gall bladder wall thickness increased in 5 patients. One patient had multiple gallstones: the wall thickness in this patient was attributed to cholelithiasis.

These observations suggest that inflammatory changes similar to those seen in viral hepatitis occur in patients with malarial hepatitis. Murphy et al observed that malarial hepatitis was an important contributory factor for jaundice in patients with malaria. Other workers have reported histological evidence of hepatocellular damage in such patients. Liver tissue obtained postmortem in four of our patients revealed swollen hepatocytes, presence of hemozoin deposits, portal infiltrates by mononuclear cells, congestion, Kupffer cell hyperplasia, centrilobular necrosis, bile stasis and fatty change.

Thus, the presence of hepatomegaly with low echogenicity and thick gall bladder wall in patients with Plasmodium falciparum malaria and jaundice suggest diffuse hepatocyte dysfunction.

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References

Heavy metals in 'herbal' medicines

We read with interest the report on lead poisoning by Sood et al. We would like to congratulate the authors for highlighting the importance of good history-taking in arriving at a diagnosis, especially regarding intake of herbal medicines. At the adverse drug reaction monitoring cell in our department, we have also come across 9 patients in the year 2002 who reported with features suggestive of toxicity with heavy metals, with a history of taking herbal medicines. Analysis of the herbal medications (by inductively coupled plasma-atomic emission spectroscopy) that were being taken by the patients showed the presence of lead in two samples, mercury in one, and arsenic in one sample.

A case we would like to highlight is that of a woman who presented with thrombocytopenia. She had a history of repeated abortions for which she was taking an Ayurvedic medicine garbhapat ras. This formulation contains nagbhasma (lead), vangbhasma (tin), lobahasma (iron), hingula (mercuric sulfide) and three plants Cinamomum zeylanica, Felterta cardamomum and Cinamomum tamal. Plasma of the patient showed 11.9 µg/dL arsenic, which is above the permissible limits (0.17 to 5), 17.9 µg/dL lead (permissible levels 10 to 25) and mercury below 5 µg/dL (i.e., below permissible limit. The tablet itself contained lead (10-400 ppm), arsenic (7100 ppm) and mercury (34000 ppm). We then tested five different marketed preparations of Garbhapat ras and found arsenic in four of the five preparations, ranging from 1000 to 7100 ppm. The point to be noted is that arsenic is not an ingredient of this medication.

There are several issues that emerge from these cases. Firstly, heavy metals may be present in these 'Ayurvedic' or herbal preparations either because they are supposed to be there, as in herbomineral preparations (bhasmas), or because herbal medicines, that are supposed to contain only plants, have metals in them as impurities (e.g., pesticide residues). The problem with the first category of medicines is that the process of preparation of bhasmas is a complex procedure. The method of preparation described in Ayurveda for each of these bhasmas is unique in terms of raw material requirements and processing methodology, including critical parameters like temperature, duration required for process, and number of cycles of heating (shodhan). If this process is not followed meticulously, there is a possibility of formation of toxic compounds.

It is estimated that about 80% of the Indian population consume Ayurvedic medicines, often self-prescribed. There is also a general misconception that Ayurvedic drugs are safe compared to allopathic drugs; they are therefore preferred by patients for liver and pancreatic diseases, irritable bowel syndrome, chronic constipation, and many more GI disturbances. Hence, it

References

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