Correspondence

Sulindac hepatotoxicity

Sir,—We were interested in the well documented report by Dr Whittaker et al concerning two patients with hepatotoxicity due to sulindac (Gut 1982; 23: 875). We report here a patient whom we believe suffered from this, but in whom a liver biopsy showed histological features differing from those reported by those authors.

A 45 year old Asian woman presented with a six day history of jaundice, abdominal pain, and fever. Her drug history was of six years therapy with dihydrocodeine for back pain, and three months' treatment with sulindac (Clinoral). Four days after the onset of jaundice her family practitioner treated her with co-trimoxazole. On admission she had tender hepatomegaly and her spleen was just palpable. Serum bilirubin was 200 μmol/l, aspartate amino transferase (AST) 246 IU/l (N<40), gamma-glutamyl transpeptidase 1140 IU/l (N<35), and serum alkaline phosphatase 595 IU/l (N<133). Viral hepatitis markers were negative. After exclusion of extrahepatic obstruction by ultrasound examination, in view of the persisting symptoms, liver biopsy was performed 10 days after admission, which was also 10 days after withdrawal of drug therapy. At this time bilirubin was 78 μmol/l, AST 440 IU/l, and alkaline phosphatase 1040.

The histological features (Figure) were as follows: the vascular pattern was retained but there was mild hyperplasia of liver plates. There was focal hepatocyte necrosis, with focal inflammatory infiltrates throughout the parenchyma, and eosinophils present among the infiltrating cells. The Kupffer cells were not notably reactive. Portal tracts were unremarkable but with a predominantly mononuclear cell infiltrate. There was no evidence of bile duct proliferation or obstruction, and ceroid pigment was not apparent.

Twelve weeks after presentation, however, the function test returned to normal and the patient was well and has remained so subsequently.

The clinical features of our patient are similar to those of Whittaker et al, and of other cases of sulindac hepatitis, but are obviously not specific. The histological features, with eosinophils present in the inflammatory infiltrate, and little reaction among the Kupffer cells, are suggestive of a drug reaction. The ceroid pigment noted in both Whittaker's cases was not found. There may therefore be a variety of histological abnormalities associated with a reaction to sulindac.

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Books


This short review has conventional chapters on duodenal ulcer and its complications, gastric ulcer, special aspects, surgical treatment, Zollinger Ellison syndrome, and acute stress ulceration. In this book duodenal ulcer is said to have pain occurring at 11 am, 4 pm, 10 pm, and 1 am, which is usually relieved within a matter of minutes by food or antacids; assessment of the gastric secretory capacity is said to be a useful procedure even though the appendix, Protocol for gastric analysis, alleges there is no pathognomonic range of acid production for any disease state with the possible exception of the Zollinger Ellison syndrome. Radiography is said to be the most practical and useful tool in diagnosing duodenal ulcer.
In treatment, alcohol, caffeine, and cocoa are excluded during the acute phase. Drug treatment begins with an antacid regimen of 80 mEq hourly for one week, one and three hours after meals and at bedtime for six weeks, and one hour after meals and at bedtime for two months. If anticholinergic drugs are used, the start is tincture of belladonna, 10 drops in water four times a day before meals and at bedtime. Although the imprint is September 1983 the USA drug lag means that treatment used in Europe for years such as ranitidine, pirenzepine, trimipramine, and colloidal bismuth, are referred to merely as undergoing clinical trials. Carbenoxolone received 11 lines, but spelled as carbenoxalome. Most chapters have case histories of individual patients and barium meal radiographs.

The Contemporary patient management series claims to be dedicated to the specialist as in depth guidelines for effective management, thorough background information, and current preferences for an advanced perspective. This specialist considers this monograph does not meet these objectives.

J H BARON

Viral hepatitis: laboratory and clinical science
Edited by F Deinhardt and J Deinhardt. (Pp. 585; illustrated; SwFr.238.) New York: Marcel Dekker, 1983.

This book is intended to fulfill the needs of students, clinicians, and laboratory investigators for an overview of the various aspects of human viral hepatitis from the history of the disease to the virology, from laboratory diagnosis to epidemiology, from clinical description and management of disease, with control measures, with a review of the background to our present knowledge and speculation on the developments to be expected. How far has the book succeeded in fulfilling these wide and laudable aims? The 34 contributors are certainly all distinguished and chosen from all over the world, Europe being unusually well represented for a multi-author offering. The number of authors and their differing backgrounds makes for a certain patchiness and for inevitable overlap. In some ways, however, this is an advantage, allowing the discerning reader to obtain different points of view on a particular aspect of viral hepatitis.

Among the many excellent chapters, the article by Arie Zuckerman on the history of hepatitis is particularly scholarly and comprehensive. The article by Bianchi and Gudat on the immuno-pathology of viral hepatitis is very well done and with beautiful illustrations. Clinical syndromes and pathology are very full and well covered. Treatment is a little flimsy but perhaps this reflects the paucity of effective measures anyway. References are profuse but it is a pity that they are not uniform in presentation and the titles of those quoted are not always given. This limits the value of the book as a reference source. It is inevitable, also, that although the book is published in mid-1983 references do not extend beyond 1982, and there are indeed very few for that year. Hepatitis B and its relation to hepatocellular carcinoma is a rapidly growing field, and this chapter includes 150 references (no titles!).

I could, however, find only five from 1982, and three of these came from a monograph and not from the original literature.

This is an extremely important source book for those interested in all aspects of viral hepatitis in man. It should be included in all medical libraries, and the hepatologist should have it on his bookshelf.

SHEILA SHERLOCK

News

BSG Research Award 1983
A three page summary of personal research work is invited by the Awards Committee who will recommend to Council the recipient of the Award for 1983. A bibliography may also be submitted if desired. The Award consists of a medal and £100 prize. Entrants must be 40 years or less (on 31 December 1983) but need not be a member of the BSG. All (or a substantial part) of the work must have been performed in the UK or Eire. The recipient will be required to deliver a 40 minute lecture at the Plenary Session of the Spring meeting in 1984. Applications (six copies) should be made to: The Honorary Secretary, BSG The Rayne Institute, 5 University Street, London WC1E 6JJ. Closing date December 1983.

Second British Congress of Lasers in Medicine and Surgery
This meeting will be held on 13 December 1983 at the Barbican Centre, London. Further details from Mr J A S Carruth, Consultant Surgeon, Royal South Hants Hospital, Graham Road, Southampton SO9 4PE.