Letters to the Editor

ACUTE ACALCULOUS CHOLECYSTITIS

Dear Editor,

This refers to the case report "Acute Acalculous cholecystitis" by Lt Col DJ Singh recently published in MJAFI [1].

Clinical and ultrasonographic evidence of gall bladder involvement has also been reported in acute viral hepatitis (AVH) [2]. Involvement of gall bladder wall (GBW) by the virus has been postulated as the most plausible explanation for the GBW thickening in AVH [3].

The other possible mechanisms suggested are hypoalbuminemia, lymphatic obstruction, reduced bile flow, and involvement of GBW in the immuno-inflammatory process occurring in adjacent liver tissue. We had studied GBW thickness in 118 consecutive patients of AVH and compared this with their clinical and biochemical profile during 1st to 4th week of illness [2]. Significant thickening of GBW was observed in 77% of patients. A statistically significant correlation was seen with non-A, Non-B hepatitis (NANB). Within NANB group, comprising 83 patients, GBW thickening was again significantly more pronounced ($P = < .001$) in those 20 patients who had presented with right upper quadrant pain, fever and a positive ultrasonographic Murphy's sign, a presentation like acute cholecystitis - similar to cases No. 2 and 3 of the authors [1].

These observations warrant a careful evaluation of all patients of acute acalculous cholecystitis (AAC) for pres-

ence of AVH since surgery in AVH can prove hazardous. Rightly so, the author's patients were managed conservatively and improved. He has not mentioned the levels of liver enzymes (SGOT, SGPT) in his report. These are usually raised in AAC associated with AVH. A study of viral markers for hepatitis B, hepatitis C, and hepatitis E virus (HBV, HCV and HEV respectively) may also help in establishing the diagnosis.

The question which still remains unanswered is that which of the currently known Non-A, Non-B viruses (HCV, HEV hepatitis G virus or hepatitis H virus) are likely to predominantly attack gall bladder producing an acute cholecystitis like picture? Our own unpublished experience so far suggests that it is at least not the HCV. It could possibly be HEV or another non-A, non-B virus.

Lt Col R DOGRA

**Department of Gastroenterology,
Command Hospital (EC), Calcutta - 700 027.**

REFERENCES

1. Singh DJ. Acute Acalculous cholecystitis. MJAFI 1995; 51 : 56-7.
2. Sharma MP, Dogra R, Tandon BN. Significance of gall bladder wall thickening in Acute Viral Hepatitis. An ultrasound study APICOM 1992; 356/018 (HEP).
3. Maudigal D, Jones MHW, Joseph AEA. Gall bladder wall abnormalities in acute infectious hepatitis. A prospective study. Dig Dis Sci 1984; 29 : 257-60.

Author's reply

The comments asked for are as follows :

- (a) SGOT, SGPT levels in respect of cases 2 and 3 reported in the article under reference, were monitored, and were normal. Only serum bilirubin and alkaline phosphatase levels were raised.
- (b) Only facilities for HBsAg were available, which were carried out, and were negative in both cases.
- (c) On these grounds, the possibility of acute viral hepatitis (AVH) was ruled out.

The aim of the paper was to present an appraisal of the conservative management of acute acalculous cholecystitis (AAC). This experience, is contrary to the generally accepted view that AAC must be managed energetically on an emergency or semi-emergency footing. Quite

obviously, these cases of AAC do not include those associated with AVH where surgical intervention would be disastrous. The cases reported were basically of a surgical nature, monitored closely with a view to avoid surgical intervention in an acute inflammatory state, but keeping the possibility at the back of the mind.

In my limited experience (now spanning 13 cases of AAC), a conservative line of management has a definite role to play in the management of AAC. The outcome was very satisfactory in all the cases.

Lt Col DJ SINGH

**Classified Spl (Surgery)
Military Hospital Jodhpur**