LETTER TO THE EDITOR

BILIARY ASCARIASIS AND EXTRAHEPATIC CHOLANGIOCARCINOMA

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Dear Sir

We were intrigued after reading the abovenamed paper[1], and feel that several points need clarification. The term 'extrahepatic cholangiocarcinoma' in the title implies cancer of the extrahepatic bile duct only, not the gall bladder. The pathological behaviour and natural history of these two cancers have little in common and should be considered separate entities.

The findings on ultrasound in the first case suggest dilatation, not only of the intrahepatic ducts, but the common bile duct (CBD) as well, but surprisingly there was no mention of the gall bladder. However, at surgery, the CBD below the gall bladder (GB) mass was found to be normal, because only adjacent common hepatic duct (CHD) was invaded by the tumour. The ultrasound findings in this situation are quite characteristic and similar to those seen in hilar cholangiocarcinoma and Mirizzi syndrome[2], and direct cholangiography is necessary to confirm the diagnosis[2].

Over 75 percent of carcinoma GB occur in elderly patients with cholethiasis. Since this tumour was discovered in an acalculous gall bladder, the presence of an anomalous pancreaticobiliary junction (APBJ) should have been suspected, because several authors have found this anomaly in patients with gall bladder tumours[3,4]. The surgical procedure in cases with APBJ must then include excision of the supraduodenal CBD, closure of the lower end and a hepaticojejunostomy to ensure separation of the biliary and pancreatic systems to prevent further mucosal change[4].

The T-tube radiograph of the second patient (no-T-tube is visible) following a palliative cholecystojejunostomy shows a non-distended GB and no contrast in the jejunum. The left ductal system and CHD appear grossly dilated while the right ducts are normal, and is not due to obstruction caused by the tumour. It is also very unlikely that the peripherally located *Ascaris is* responsible for this unusual ductal dilatation. This type of dilatation is suggestive of choledochal cyst affecting the left and common ducts only, the features of a type IVa cyst. Some form of cholangiography may perhaps have documented an anomalous junction in this patient also, which is present in the majority of cases of choledochal cysts.[5]

The *Ascaris* is perhaps a coincidental finding and is known to cause cholangitis[6,7] but it would be mere speculation to consider it carcinogenic. Because Ascariasis is endemic in rural Malaysia, cases of cholangitis are fairly common, but then should not cholangiocarcinoma be encountered more often? The authors' contention that chronic cholangitis may progress to cholangiocarcinoma is not based on fact, because 2 large series reporting cholangitis and hepatolithiasis encountered only one malignancy in 557 patients[8,9].

We believe that the *Ascaris* worm caused cholangitis in both patients during its biliary sojourn and paid the price. Based on the facts presented by the authors, we feel that consideration was not given to an APBJ without cystic dilatation in the first case and a type IVa choledochal cyst in the second case, and both presented with malignancy, a well known complication and with little likelihood of being caused by the *Ascarid* misadventure.

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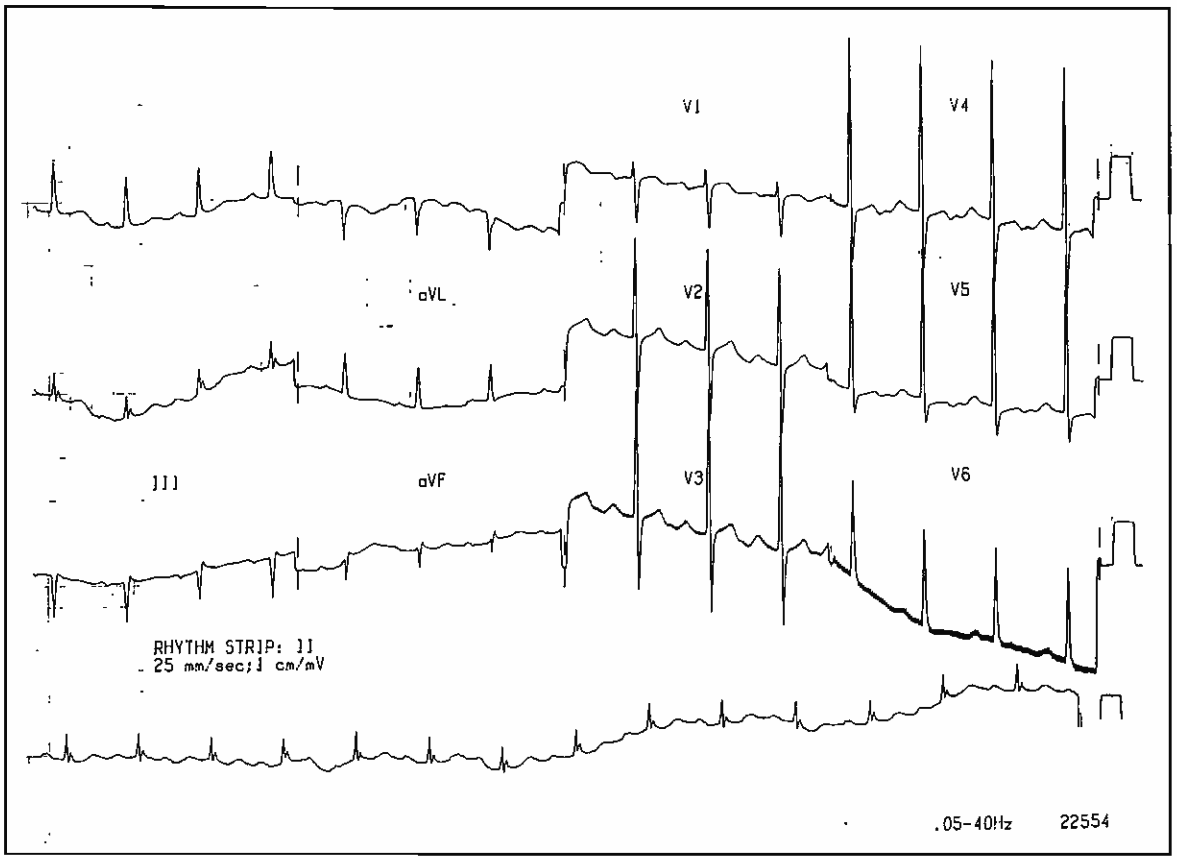
Editorial Comments: The above letter has been sent to the authors for comment, but to date no reply has been received.

Fig 2 – 12-lead electrocardiogram done three months prior to electrocardiogram in Fig 1



ANSWER TO ELECTROCARDIOGRAPHIC CASE

Diagnosis: Cerebral autonomic T wave abnormalities from a right acute subdural haematoma

DISCUSSION

The differential diagnosis in this case are myocardial ischaemia/non-Q infarction and cerebral autonomic abnormalities.

Fig 1 shows the following abnormalities:

- Asymmetrical T wave inversion in V1-6
- ST depression in V3-6
- Tall R waves in V2-5
- Left ventricular hypertrophy
- Isolated Q in III
- 1st degree heart block

Fig 2, the ECG done three months earlier, showed left ventricular hypertrophy with probable old inferior infarct. However, the T waves were normal in V2-6.

The CT scan of the head for this admission showed an acute right subdural haematoma extending from the right frontal bone to the right parietal bone with adjacent cerebral oedema, some compression of the right lateral ventricle and shift of midline to the left suggesting a contracoup injury. There was an infarct in the left lentiform nucleus. The cardiac enzymes done for this admission were normal. Craniotomy and evacuation of haematoma was done on the same day. Intraoperatively, the vital signs remained stable and there was no change in the ECG. Hence, the most probable cause of the inverted T waves was unlikely to be of cardiac origin but rather due to the right acute subdural haematoma. She was finally discharged three weeks later, alive and able to talk, although with slurred speech.

This case is reported to make one aware of the ECG changes in cerebral disorders. It is often a dilemma to determine whether the ECG changes are cardiac or cerebral in origin. It is especially important in a patient who has to undergo anaesthesia for an evacuation of the haematoma because delay in the evacuation may occur if the ECG is mistaken to be due to myocardial ischaemia[1].

Cropp and Manning in 1960[2] advocated that without a good history or clinical findings of heart disease, an ECG showing changes suggestive of recent infarction or acute myocardial ischaemia should be interpreted as indicative of intracranial rather than of heart disease. In the study, the ECG changes were most suggestive of anterior myocardial ischaemia or infarction. The typical pattern was one with flat or negative T waves in leads I, aVL and V4-6, along with ischaemic RS-T segment changes. The ECG changes in this study were seen in subarachnoid haemorrhage.

Hugenholtz in 1961[3] reported marked prolongation of Q-T interval and extremely wide, deeply inverted T waves and prominent U waves in cerebral disorders. In his article, he quoted Burch and associates in 1954 that "some of the widest and largest T waves seen in clinical ECG were recorded in cerebral disorders, especially cerebral haemorrhage."

Ashby and Chadha reported in 1968[4] that a large number of intracerebral conditions including subarachnoid haemorrhage, intracerebral haemorrhage, cerebral thrombosis, subdural haematoma and cerebral tumour can be associated with abnormal ECGs.

Hunt et al in 1969[5] reported that the presence or absence of pathological Q waves is the most useful point in

differentiation from myocardial infarction. Serum enzyme levels ie creatine phosphokinase and aspartate transaminase are elevated in both subarachnoid haemorrhage and myocardial infarction, but consideration of the time pattern of enzyme elevation may be helpful in differentiation. In subarachnoid haemorrhage, the enzymes rise more slowly and may persist. In contrast, in myocardial infarction, the levels peak within 6-12 hours and return to normal in 3-6 days.

Though, typically, T waves become inverted after cerebrovascular accidents, Runge and Bousvaros in 1970 reported that several authors (Byer et al, 1947; Burch et al, 1954; Kreus et al, 1967) have mentioned the appearance of large upright T waves[6].

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