Neurogenic T waves after subdural haematoma

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INTRODUCTION

Electrocardiogram (ECG) abnormalities are well known to occur in the setting of cerebral diseases, especially after intracranial haemorrhage and ischaemic stroke. The incidence after subdural haematoma is not clearly defined although there have been occasional case reports (Ohtsuka et al, 2000). This article describes a case of extensive ischaemic-appearing neurogenic ECG changes in a patient who presented with a fall.

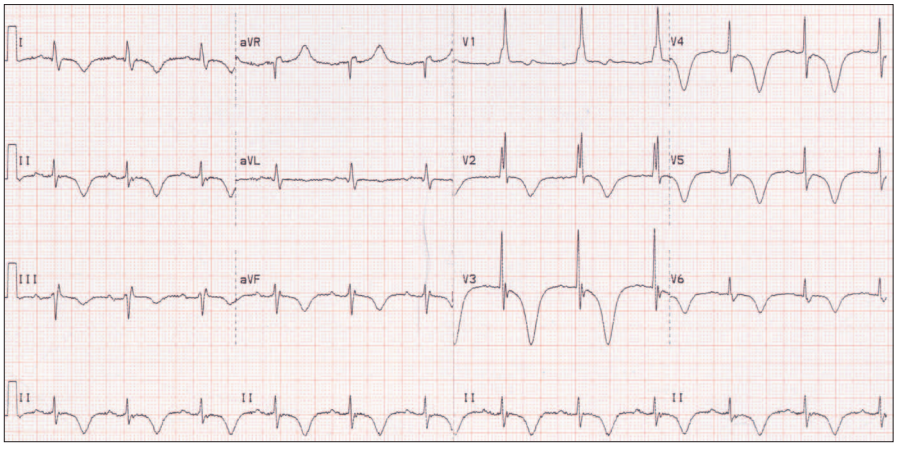
CASE REPORT

A 63-year-old woman, living alone, was admitted following a fall. She had not lost consciousness and did not have a fit. Her only complaint was of dull headache. On arrival she was haemodynamically stable, did not have any external injury marks and there were no focal neurological findings. An electrocardiogram (ECG) revealed bifascicular block with widespread, deep, symmetrical T-wave inversions consistent with subendocardial ischaemia (*Figure 1*). The patient denied any chest pain, palpitations or cardiac symptoms. She had received a coronary artery bypass graft for triple vessel coronary disease 2 years before and was symptom free since. She did have bifascicular block in her previous ECGs, but the T wave changes were new developments.

Her creatine kinase and cardiac troponin I, performed in proper time frame, were not elevated and she did not have any electrolyte abnormalities. Sequential ECGs continued to show similar T changes. In view of the history of fall she was not commenced on usual antiplatelet and antithrombotic therapy. An urgent computed tomography scan of the head was arranged which revealed a right parietal subdural haematoma about 1.1 cm in maximum thickness with no midline shift. Management was conservative on the advice of the neurosurgeon. An exercise thallium scan failed to show any significant perfusion defects and echocardiogram was normal.

She made good progress and uneventful recovery. At outpatient clinic review 4 weeks later, all T-wave changes had resolved (*Figure 2*). It was concluded that ischaemic-appearing ECG changes were neurogenic, secondary to her head injury with subdural haematoma.

Figure 1. Electrocardiogram on admission.



DISCUSSION

There is a frequent association between cerebral lesions and electrocardiographic changes. The precise incidence of ECG abnormalities after intracranial event is not known even though an incidence as high as 80–90% has been reported after subarachnoid haemorrhage (Davis et al, 1993). The most common changes described are ST segment deviation, QT prolongation, increased T wave amplitude and abnormal U waves (Rudehill et al, 1987). Occasionally ECG changes can be a manifestation of preexisting cardiac disease, which is common in this population and which becomes clinically apparent during the physiological stress of an intracranial event. It is often difficult to discern whether the ECG changes are the result of the stroke or of underlying preexisting cardiac disease.

It is believed that altered sympathetic tone and catecholamine levels (mainly a markedly increased sympathetic and parasympathetic output) are partly responsible for these ECG changes (Sato et al, 1999). There have been case reports of patients with intracranial haemorrhage and ECG changes consistent with myocardial infarction and normal coronary anatomy (Kitching and Bernstein, 1994).

This patient had manifested asymptomatic cardiac ischaemia and associated ECG changes. This may have been as a result of the stress of head injury with subdural haematoma exacerbating her underlying intrinsic cardiac disease, or alternatively the ECG changes may have been simply secondary to subdural haematoma. Elevated levels of troponin I are highly specific and as sensitive as MB fraction of creatine kinase (CK) for the detection of myocardial injury. This patient did not have elevated troponin I or CK levels, suggesting that she probably did have transient cardiac dysfunction rather than myocardial injury.

This case report illustrates how ECG abnormalities in a patient with head injury can cause difficulties for attending physicians. However, it is important to realize that these ECG abnormalities do not appear to represent an adverse cardiovascular outcome (Zaroff et al, 1999).

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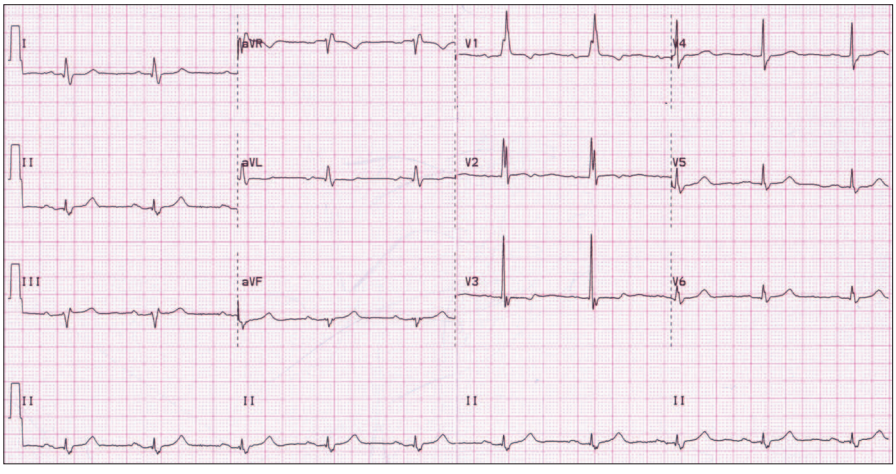

Figure 2. Electrocardiogram 4 weeks after discharge.

CONCLUSION

A diagnosis of primary cardiac disease based on ECG changes in the setting of suspected head injury can result in inappropriate and sometimes dangerous therapy (e.g. antiplatelet, anticoagulation, thrombolysis). A thorough history and clinical examination is therefore mandatory to avoid the catastrophic consequences of administering antiplatelet or thrombolytic therapy. HM

Davis TP, Alexander J, Lesch M (1993) Electrocardiographic changes associated with cerebrovascular disease: A clinical review. *Prog Cardiovasc Dis* **36:** 245–60

Kitching AD, Bernstein M (1994) Primary intracranial hemorrhage presenting as acute myocardial infarction: a contraindication to thrombolytic therapy. *CMAJ* **150:** 519–22

Ohtsuka T, Hamada M, Kodama K et al (2000) Neurogenic stunned myocardium. *Circulation* **101:** 2122–4

Rudehill A, Olsson GL, Sundqvist K et al (1987) ECG abnormalities in patients with subarachnoid hemorrhage and intracranial tumors. *J Neurol Neurosurg Psychiatry* **50:** 1375–81

Sato K, Masuda T, Izumi T (1999) Subarachnoid hemorrhage and myocardial damage clinical and experimental studies. *Jpn Heart J* **40:** 683–701

Zaroff JG, Rordorf GA, Newell JB et al (1999) Cardiac outcome in patients with subarachnoid hemorrhage and electrocardiac abnormalities. *Neurosurgery* **44:** 34–9

IMAGES IN MEDICINE

Biliary ascariasis

Ramin Ghadimi, Mehdi Mohamadnejad, Seyed Amir Mirbagheri

A 28-year-old woman was admitted to the hospital with fever and right upper quadrant pain of 2 weeks' duration. On laboratory evaluation she had leukocytosis, elevated serum alkaline phosphatase, and mildly elevated serum bilirubin level. Blood culture was negative. Right upper quadrant ultrasonography was unremarkable.

On endoscopic retrograde cholangiography the bile duct was dilated and contained multiple longitudinal filling defects that extended into the intrahepatic bile ducts (*Figure 1*). Biliary sphincterotomy was undertaken and eight ascaris worms were retrieved with a basket. Treatment was initiated with intravenous ampicillin and gentamycin as well as piperazine with mebendazole. One week later, repeated endoscopic retrograde cholangiography showed only one linear filling defect in the common and left hepatic duct (*Figure 2*). That worm was also removed with basketing. Her general condition quickly improved. She is in good health after 17 years of follow up. HM

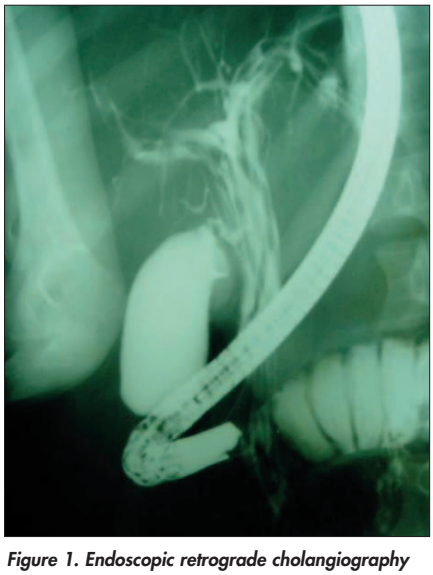

Figure 1. Endoscopic retrograde cholangiography showing dilated common bile duct that contained multiple longitudinal filling defects that extended into intrahepatic bile ducts.

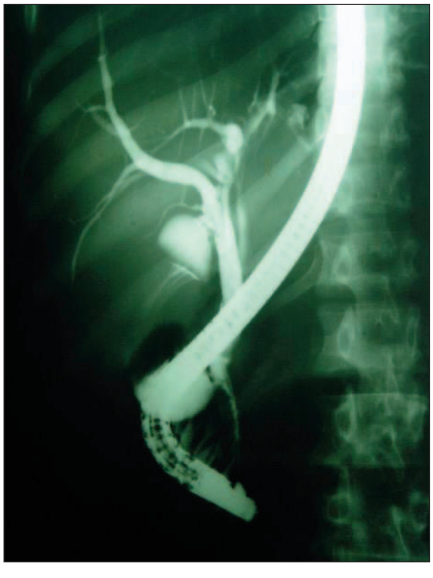

Figure 2. Repeated endoscopic retrograde cholangiography shows one linear filling defect in the common and left hepatic duct.

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