Clinical Reports

Subarachnoid haemorrhage: a cause of left bundle branch block?

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Summary: We describe what we believe to be the first reported case of documented *de novo* left bundle branch block in association with acute subarachnoid haemorrhage.

Introduction

Many electrocardiographic changes have been described in association with subarachnoid haemorrhage. The phenomenon was first reported in 1947.¹ The development of *de novo* left bundle branch block has not previously been documented with subarachnoid haemorrhage.

Case report

A 77 year old man attended the Accident and Emergency Department with an episode of pre-syncope. He reported dizziness, nausea and mild neck discomfort. There was no history of palpitations, chest pain or headache and before this he had been feeling completely well. He was a non-insulin-dependent diabetic controlled on diet, with no hypertension or previous cardiac history.

On examination the patient was pale, sweaty and was retching. He was apyrexial and normotensive, with a regular pulse of 88 beats per minute. He had no neurological signs, papilloedema or neck stiffness. Initial investigations, including a full blood count, urea and electrolytes and liver function tests were all normal, as was a chest X-ray and an electrocardiogram (Figure 1).

He was admitted for observation, continuous electrocardiographic monitoring and cardiac enzyme measurements. He was treated with bed rest, oral analgesia (paracetamol 1 g and dihydrocodeine 20 mg in combination, four times daily) and an intravenous anti-emetic (metoclopramide

10 mg three times daily). Serial cardiac enzymes were subsequently documented to be normal and he had no arrhythmias on monitoring.

On the day following admission, he developed a severe headache and profuse vomiting. There was now marked meningism, with neck stiffness and exaggerated reflexes. The electrocardiogram now showed left bundle branch block, with an unchanged axis, in sinus rhythm at a rate of 60 beats per minute (Figure 2). His blood pressure was now 180/100 mmHg. An urgent computerised tomographic brain scan showed no abnormalities before and after intravenous contrast. At lumbar puncture the cerebrospinal fluid was xanthochromic, with slightly increased white cells, grossly raised red cells and high protein. Culture revealed no bacterial growth.

Figure 1 Electrocardiogram on admission, with no atrio-ventricular delay. QRS duration 100 milliseconds, sinus rhythm 88/minute, axis -20° .

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Figure 2 Electrocardiogram 20 hours after admission, showing left bundle branch block. QRS duration 200 milliseconds, sinus rhythm 68/minute, axis -20° .

A diagnosis of subarachnoid haemorrhage was made and conservative management was chosen. Anti-hypertensive treatment with a calcium channel blocker (nifedipine slow release 10 mg twice daily) was initiated following the lumbar puncture. The patient made an uneventful recovery, anti-hypertensive treatment was stopped 2 days before discharge and the patient left the hospital a total of 10 days after admission. At out-patient follow-up 6 weeks later he was well, normotensive on no current treatment and his electrocardiogram indicated persistence of the left bundle branch block pattern.

Discussion

It is well known that subarachnoid haemorrhage produces cardiac and pulmonary abnormalities.² Cardiac arrhythmias have been documented in greater than 90% of cases of subarachnoid haemorrhage.³ Morphological abnormalities of the electrocardiogram have also been widely described, with changes occurring in at least 50% of cases.⁴ The findings on electrocardiography commonly exhibit 'ischaemic' features, such as ST-segment or T-wave changes, or U-wave abnormalities, but also include abnormal P waves, or changes in the PR or QT intervals.⁴ The cause of these electrical changes in subarachnoid haemorrhage is not fully understood, but it would appear to have a multi-factorial aetiology.

Intracranial events have been shown to result in both histologically visible myocardial necrosis and subendocardial haemorrhage,^{5,6} features that sug-

gest structural cardiac damage as a cause for the electrocardiographic changes. Thallium myocardial scintigraphy has also suggested that myocardial perfusion is impaired in a proportion of cases with abnormal electrocardiograms after subarachnoid haemorrhage,⁷ thus implying that there is an ischaemic element to the electrocardiographic changes. However, a positive thallium scan was not related to any specific electrocardiographic feature. It is also important to note that none of these patients had had ischaemic heart disease excluded prior to the subarachnoid haemorrhage.

Animal work has shown that intracranial injection of blood gives rise to electrocardiographic changes, mainly T-wave changes, in a proportion of cases, but not in all.⁸ In patients who have electrocardiographic abnormalities compared to those without, it has been demonstrated that there is increased excretion of catecholamine metabolites.⁹ The increased catecholamines may be caused by hypothalamic damage in subarachnoid haemorrhage.⁹ Some of the electrocardiographic changes relating to the effects of the catecholamines are abolished by treatment with β -blocker¹⁰ but in clinical trials this treatment has not been beneficial. Other workers suggest that only sinus tachycardia and T-wave inversion are associated with raised catecholamines.¹¹ It is implied that at least some of the recognized electrocardiographic changes are secondary to changes in catecholamine levels.

In two previous articles, bundle branch block has been associated with subarachnoid haemorrhage but, in both of these reports no premorbid electrocardiograms were quoted, and therefore it is not clear whether the bundle branch block was a new development.^{12,13} In contrast, the patient that we have reported clearly had a normal electrocardiogram on admission with mild non-specific symptoms. The left bundle branch block occurred when the symptoms became more classical of a subarachnoid haemorrhage. The clinical signs supported this diagnosis, which was ultimately confirmed by the lumbar puncture findings.

Left bundle branch block occurs as a result of delayed conduction within the left bundle of His. It usually indicates the presence of significant cardiac disease and is a strong predictor of cardiac mortality.¹⁴ It is unlikely that a coincidental cardiac event occurred in this patient to cause the left bundle branch block because there were no symptoms to suggest myocardial ischaemia and a normal cardiac enzyme series was recorded. Development of left bundle branch block can also be rate-related¹⁴ but this was not true in this patient. It is concluded that the left bundle branch block was related to the subarachnoid haemorrhage.

In summary we would like to add the development of left bundle branch block to the list of

abnormal electrocardiographic findings associated with subarachnoid haemorrhage. The importance of excluding intracerebral pathology when present-

ed with a patient with no obvious cardiac cause for an abnormal electrocardiogram should be re-emphasized.

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