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 68 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.

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

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abnormal electrocardiographic findings associated with subarachnoid haemorrhage. The importance of excluding intracerebral pathology when presented with a patient with no obvious cardiac cause for an abnormal electrocardiogram should be re-emphasized.

References

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