TRANSIENT ISCHAEMIC ATTACK AFTER SPINAL ANAESTHESIA

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SUMMARY
A case of transient ischaemic attack lasting 6 h occurred after spinal anaesthesia with bupivacaine. The level of sensory block was satisfactory and there was no significant hypotension. We discuss the possible cause of this previously undescribed complication.

KEY WORDS

Spinal anaesthesia is associated frequently with side effects such as hypotension, low back pain and headache. Neurological complications described include subdural haematoma [1], cerebral uncus herniation [2], intracranial haemorrhage [3], paresis of cranial nerves [4-6], pneumocephalus [7], visual disturbance [8, 9] and sensory and motor deficit at and below the level of dural puncture [10]. Convulsions and temporary hemiparesis have been described after spinal anaesthesia in a child with Moyamoya disease [11]. Unexplained hemiplegia and retrobulbar neuritis has also been reported in a healthy patient after hydrocele repair under spinal anaesthesia [12]. We describe a case of transient ischaemic attack characterized by loss of consciousness and temporary hemiparesis after induction of spinal anaesthesia in a healthy 58-yr-old male. There was no associated hypotension.

CASE REPORT
A 58-yr-old male presented with benign prostatic hypertrophy for transurethral prostatic resection. Pre-anaesthetic assessment revealed a history of hypertension, controlled well with atenolol. He was a non-smoker and had no other relevant past medical history. On examination he weighed 85 kg, cardiovascular system was normal with arterial pressure of 120/80 mm Hg and there were no carotid bruits. ECG, haemoglobin and serum concentrations of urea and electrolytes were normal. Premedication comprised papaveretum 20 mg and hyoscine 0.4 mg i.m. 1 h before operation.

On arrival of the patient in the anaesthetic room, a vein was cannulated and ECG and non-invasive arterial pressure monitoring attached. Systolic arterial pressure was 160 mm Hg. An i.v. preload of Hartmann's solution 750 ml was commenced and midazolam 4 mg given i.v., resulting in satisfactory sedation. Initial attempts at spinal anaesthesia were unsuccessful at L4–5 because of inability to find the interspace. Dural puncture at L3–4 with a 26-gauge spinal needle was successful at the first attempt and plain 0.5 % bupivacaine 2.75 ml was injected. Arterial pressure was measured at 3-min intervals and the level of sensory block was T10 bilaterally 25 min later. Systolic arterial pressure did not decrease to less than 110 mm Hg at any time and the patient remained alert and co-operative. He was transferred to the operating theatre and connected to ECG, non-invasive arterial pressure and pulse oximetry monitoring (Datex cardiocap).

As the procedure was about to commence, the patient became dysarthric then lost consciousness and became apnoeic. His lungs were ventilated with 100 % oxygen by face mask; arterial pressure was 120 mm Hg systolic. Ephedrine 10 mg was given i.v. as it was thought that this reading might be incorrect, and subsequent arterial pressure was found to be 140/90 mm Hg. Initially, a diagnosis
of high spinal block was considered, but the patient responded to pain originating above the level of T10. The period of apnoea lasted only about 5 min and resolved spontaneously. The patient’s conscious state improved also, such that he was able to maintain his own airway. Arterial pressure and pulse oximetry saturation measurements remained normal. The lack of a temporal relationship between the administration of any drug and the sudden onset of symptoms made a pharmacological effect unlikely, but flumazenil (100-µg boluses to a total of 400 µg) and naloxone (50-µg boluses to a total of 700 µg) were given i.v. to antagonize any benzodiazepine or opioid effect, without improvement. The patient remained semiconscious, with signs of a left hemiparesis. Surgery was cancelled and the patient transferred to the intensive care unit. Arterial blood-gas tensions, serum concentrations of urea and electrolytes, cardiac enzymes, blood glucose concentrations and 12-lead ECG were normal. An urgent computed tomographic (CT) brain scan was performed and showed no focal abnormality or evidence of intracranial bleed. Contrast medium enhancement was not used as it was felt that this would add little additional information and the patient’s symptoms were resolving. Over the following 6 h the patient made a full recovery and had no recollection of the event. A diagnosis of transient ischaemic attack was confirmed by the spontaneous recovery within 24 h and normal CT scan. No significant change in heart rate or arterial pressure occurred, but we accept that, with intermittent non-invasive pressure monitoring there may have been periods of pressures less than those recorded. The onset and duration of symptoms correlated well with that of the spinal anaesthesia and it is unlikely that this attack was a purely coincidental event. We feel, however, that there are similarities between our patient and the case described of a child with Moyamoya disease [11]. In that patient, convulsions and hemiparesis lasting 10 days occurred after spinal anaesthesia. In both patients, symptoms of cerebral ischaemia occurred despite cardiovascular stability and there was good recovery.

Moyamoya disease is a syndrome with which there is a compromised cerebral circulation caused by bilateral stenosis and obstruction of major cerebral vessels and development of a vascular network at the base of the brain [14, 15]. This results in symptoms of cerebral ischaemia, usually in the form of transient ischaemic attacks. Without cerebral angiography, we have no conclusive evidence, but propose that our patient also had a compromised cerebral circulation caused by accelerated atheroma secondary to hypertension. He had a strong family history of cerebrovascular disease and, although he had no previous history of transient ischaemic attacks, blood flow to an area with critical cerebral perfusion could have decreased to less than adequate levels. This could occur without a large change in arterial pressure, because changes in cardiac output may have been more important. Even if arterial pressure is maintained by compensatory vasoconstriction above the level of a spinal block, cerebral blood flow may be reduced [16, 17].

In conclusion, we have observed a previously unreported complication following spinal anaesthesia. We suggest that this was a brainstem transient ischaemic attack consequent upon the
adverse haemodynamic effects of spinal anaesthesia in a susceptible individual.

REFERENCES