CASE REPORT

Multiple territory watershed infarcts following spinal anaesthesia

Tarig Mohammed Abkur, Mohamed Bakri Mohamed, Catherine Peters

Department of Internal Medicine, University Hospital Limerick, Limerick, Ireland

Correspondence to
Dr Tarig Mohammed Abkur, tarig1982@hotmail.com

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SUMMARY

Hypotension is one of the most common complications of spinal anaesthesia (SA). The intraoperative drop in the blood pressure is clearly associated with increased morbidity and mortality. In order to increase the awareness of this complication, we report a case of devastating stroke following an SA in a previously well woman with risk factors for vascular diseases. The patient developed multiple territory watershed infarcts and she eventually died from aspiration pneumonia 14 days after hospitalisation. The case highlights the risk of hypotension complicating SA.

BACKGROUND

It highlights the risk of intraoperative hypotension following spinal anaesthesia (SA) and how can doctors deal with it.

CASE PRESENTATION

A 77-year-old woman developed acute confusion and left upper limb weakness following an elective left knee replacement. The operation itself was performed under an SA, was uneventful except for one episode of hypotension intraoperatively, when her blood pressure dropped to 90/55 and remained low for about 30 min. Postoperatively, she was noted to be confused and unable to move her left upper limb.

The medical team on call was consulted for further evaluation and management. Assessment revealed a previously independent woman with a background history of hypertension, osteoarthritis and active smoking. Medications on admission included indapamide, paracetamol and naproxen. Initially her Glasgow Coma Scale (GCS) was 14/15 but deteriorated rapidly to 9/15. She had no fever, her blood pressure was 106/60 and the heart rate was 96 bpm sinus rhythm. Her left upper limb was hypertonic and plantars were up going bilaterally.

INVESTIGATIONS

The laboratory results are shown below in table 1. An ECG was sinus rhythm at 90 bpm. The chest X-ray was normal.

An urgent CT of the brain was obtained, which was reported as normal (figure 1).

An urgent MRI of the brain was performed and it showed a watershed (WS) infarction in the territories of the anterior cerebral artery and the middle cerebral artery (MCA). It also showed an extensive involvement of the occipital cortex bilaterally as well as minimal areas of infarction in the right cerebellar hemisphere close to the tentorium. There was no evidence of intracranial haemorrhage or midline shift (figure 2).

Stroke work-up included Holter monitor, transthoracic echocardiogram with bubble study and carotid Doppler ultrasound. There was no evidence of irregular heart rhythm at the 24 h Holter monitor. Carotid Doppler ultrasound showed mixed density plaque in the right internal carotid artery (ICA), which extends over 1 cm causing mild spectral broadening in keeping with a less than 20% stenosis. There was atheroma in the left internal carotid without haemodynamic significance. Transthoracic echocardiogram revealed mild concentric left ventricular hypertrophy with no evidence of patent foramen ovale.

TREATMENT

An acute ischaemic stroke was diagnosed and the patient was admitted to the high dependency unit and kept fasting because of a reduced GCS. Aspirin 300 mg was started rectally. The following day her GCS remained unchanged and the blinking reflex to visual threat was lost. During her hospital stay she regained consciousness and was able to answer questions by no or yes. Further assessment revealed bilateral blindness.

OUTCOME AND FOLLOW-UP

Her general condition continued to deteriorate and she died 14 days postadmission from aspiration pneumonia.

DISCUSSION

An ischaemic event occurring on the border zones between the regions of two main arteries in the brain is known as a WS stroke. They account for up to 10% of the total cases of ischaemic stroke.[1] WS infarcts carry their name because they affect the distal areas of the brain. The terminology arose from German literature, which employed the irrigation system analogy. Scholars from Germany compared the blood flow in brain's distal arterial territories to the last field on a farm receiving the lowest supply of water. Both areas showed increased susceptibility to any decline in flow.[2] In the medical field, the term WS stands for the regions that receive dual blood supply from the branches of two arteries.

Despite the fact that the descriptions of the pathological and imaging characteristics of WS infarcts are clear, there is still a debate on its pathogenesis. WS regions are believed to be vulnerable because they are distally located. Reduced perfusion pressure and recurring hypotensive episodes in the

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Table 1 Laboratory investigations

WCC	14.6	Creatinine	59 μmol/L
Hb	10.9 g/dL	Corrected calcium	2.46 mmol/L
Platelets	153	Bilirubin	27 μmol/L
Coagulation profile	Normal	GGT	220 IU/L
Na	139 mmol/L	Alanine transaminase	153 IU/L
K	3.5 mmol/L	Alkaline phosphatase	61 IU/L
Urea	7.4 mmol/L	Glucose	7.3 mmol/L

Hb, haemoglobin; GGT, gamma-glutamyl transferase; WCC, white cell count.

setting of severe ICA disease are considered to precipitate WS infarcts.

Periodic syncope at the onset of a WS stroke as well as the distinctive fluctuating or progressive hand weakness and the occasional trembling of the upper limb are consistent with and typically regarded as markers of haemodynamic failure. The analysis is backed by research demonstrating that WS infarcts that are distal to ICA disease are more likely to occur with incompetent circle of Willis. On the contrary, ICA disease embolism has a predilection to affect the stem and the large MCA branches, leading to the development of deep striatocapsular and or cortical 'wedge-shaped' infarcts.

Clinical signs and symptoms indicate that WS infarcts tend to be haemodynamic in nature, because iatrogenic or postural drops in the blood pressure precipitate these events. Other clues to the pathogenesis include the frequently observed loss of consciousness at onset with half of the patients experiencing increased haematocrit. Heart disease is also common, particularly bradyarrhythmias. In addition, the majority of patients experience occlusion or severe impediment of the ipsilateral and the contralateral ICA.[3] Nonetheless, embolism may be attributed to particular cases and in many cases; both hypoperfusion and embolism may have a role to play.[4]

WS infarcts clinical presentation is heterogeneous and is dependent on the location of the ischaemic changes. Systemic hypotension may present with bilateral signs and symptoms while unilateral severe carotid stenosis may present with unilateral signs and symptoms. It is rare to come across the typical

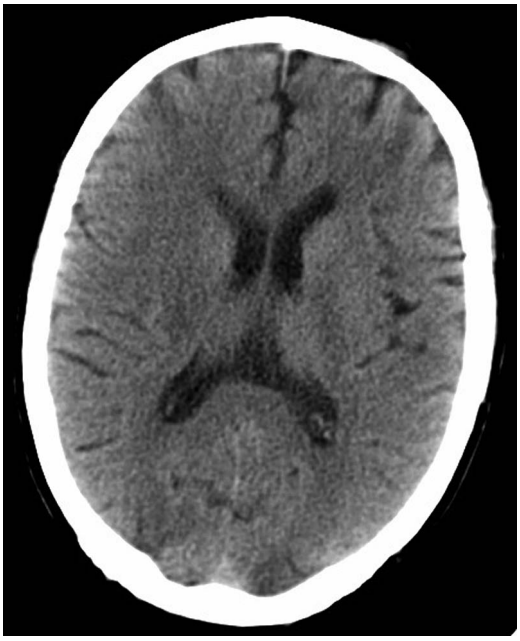

Figure 1 CT of the brain showing no acute changes.

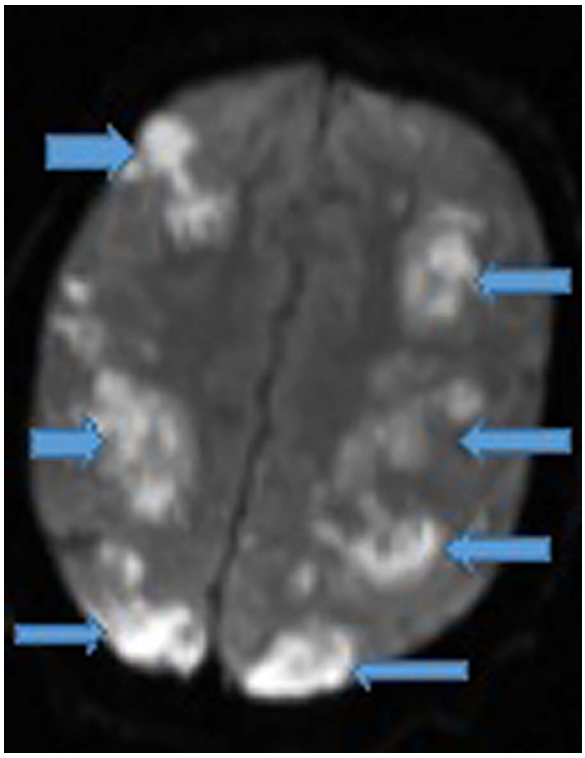

Figure 2 Axial diffusion-weighted MRI showing multiple territory watershed infarcts bilaterally (arrows).

view of the bilateral deep anterior WS stroke (the man in barrel), which is characterised by proximal weakness of the upper and lower limbs. Balint's syndrome (asimultagnosia, optic ataxia and ocular apraxia) is typically attributed to posterior infarction. Infratentorially, the WS infarcts are not well explored.[5] Haemodynamic failure which occurs after a prolonged hypotension or cardiac arrest may lead to cortical blindness from bilateral WS infarction between the territories of posterior cerebral and carotid arteries.[6 7]

Patients with external border zone infarcts have a more favourable prognosis compared with patients with internal border zone infarcts. However, the severity of the clinical presentation and the score on the National Institutes of Health Stroke Scale (NIHSS) at the time of initial assessment might not vary significantly between the two groups of patients. It is postulated that external border zone infarcts have a more benign course due to the development of collateral supply via dural and leptomeningeal anastomoses. However, when external border zone infarcts and internal border zone infarcts occur concurrently, there are higher chances of haemodynamic impairment and the outcome is expected to be poor.

WS infarcts can be subdivided according to the location into anterior and posterior. The anterior WS infarct occurs in the region between the anterior and middle cerebral arteries, while the posterior WS infarcts take place between the middle and the posterior cerebral arteries. Most of the WS infarcts occur in the anterior circulation; however they may also occur in the cerebellum, thalamus and brain stem.

The size of border zone infarcts present in the cerebellum is usually less than 2 cm. They usually occur at the border zones of the anterior inferior cerebellar artery, superior cerebellar artery, posterior inferior cerebellar artery as well as their branches. Usually, those lesions of the small border zone tend to coexist with large territorial lesions.[8]

Localisation of ischaemic areas can be achieved using MRI and CT scans. Diffusion-weighted MRI is useful in differentiating between acute stroke and chronic event or old stroke in patients having border zone infarcts. It has greater sensitivity compared with standard MRI techniques, and is superior in localising border zone infarcts with respect to vessels and this may be of help for their categorisation as either cortical or internal. Perfusion MRI or CT scan has been performed in patients with a border zone infarct detected at routine CT, MRI or diffusion-weighted MRI. There may be differing results on perfusion attributed to the mechanism of causation of border zone infarction.

Nevertheless, the way border zone infarcts appear with the anatomic imaging techniques does not always correlate with the aetiology.

The results of perfusion imaging may be vital clinically for the identification of both chronic and acute strokes. Embolism, hypotension or stenosis of a proximal vessel, which are the causes of border zone infarct, may also be suspected from the results of perfusion imaging. Identification of specific certain perfusion patterns is important in the management of patients. Normal perfusion (transient deficit), localised perfusion deficit and extensive perfusion deficit are the three perfusion patterns that have been identified.[9]

We have postulated that the stroke in our patient was precipitated by the drop in the blood pressure which complicated the use of SA. SA is a form of regional anaesthesia, which is increasingly used as an alternative to general anaesthesia in surgery. It has the ability of providing good surgical conditions for infra-umbilical operations. There is routine performance of caesarean sections under spinal block as well as total replacement of the hip and total knee replacement.[10]

Injection of local anaesthetic solution into the subarachnoid space impedes impulse conduction along all nerves it comes in contact with. There are three different nerve classes, which include sensory, motor and autonomic. The motor nerves transmit information for muscles to stimulate contraction and when these nerves are blocked it causes paralysis of muscles. Sensory nerves convey sensations like pain and touch to the spinal cord, then to the brain. The autonomic nerves regulate the calibre of the blood vessels, heart rate, contraction of the gut and other activities that are not controlled with the conscious mind. The benefits of SA include avoiding compromise of the airway and a reduction in blood loss. There is also less risk of postoperative deep vein thromboses compared with general anaesthesia. Finally, there are minimal expenses attributed to SA.

The occurrence of hypotension due to SA ranges between 15% and 56.5% in some studies.[11 12] Hypotension results from a decline in systemic vascular resistance from the sympathetic blockage via vasodilation and the spread of central blood volume to the splanchnic beds and lower extremities.

A threefold increase in the risk of developing a significant blood pressure drop was observed in patients with chronic alcohol consumption. The sympathetic nervous system can be affected by alcohol-related neuropathy, and this results in orthostatic dysregulation.[13]

Patients requiring immediate surgical intervention have higher chances of experiencing hypotension. The body conditions of such patients are usually unstable. There may be inappropriate assessment of the pre-existing conditions particularly in diseases of the cardiovascular system. Regarding patients with known hypertension, there is a twofold risk for a significant decline in blood pressure.[14] It was also observed that there is an increased risk of developing hypotension among obese patients. Large doses of local anaesthesia and immediate supine positioning can also lead to hypotension.

The spinal block sometimes extends to the levels supplying cardiac sympathetic fibres and can result in bradycardia. The patient may also be prone to cardiac arrest. Conditions such as meningitis, cauda equine syndrome, headache of postlumbar puncture and transient neurological syndrome are among the other possible side effects. Respiratory depression may develop because of respiratory centre hypoperfusion occurring after hypotension. Hypothermia may be facilitated by vasodilation. SA is not appropriate for operations lasting more than 2 h. Early observation is important since progression of blockage can be prevented. In the event of hypotension, treatment must be fast and may incorporate a head down posture, oxygen therapy, intravenous hydration as well as vasopressors.[15–17]

Haemodynamic dysfunction is responsible for internal WS infarcts in the majority of the cases, particularly the rosary-shaped infarcts in the centrum semiovale. Although severe haemodynamic compromise appears to be responsible for combined cortical and internal WS infarcts, embolism from artery to artery may hold a crucial role in cortical WS infarcts that are isolated.

In view of an increased prevalence of microembolic signals observed by ultrasound in the setting of symptomatic carotid artery disease, a recent hypothesis assumed that embolism and hypoperfusion may play a synergistic role, through which small embolic material susceptible to deposit in the distal region of arterioles could cause cortical microinfarcts during persistence of hypoperfusion. Future research combining brain perfusion imaging, diffusion-weighted imaging as well as microembolic detection by ultrasound should aid in clarifying these outstanding questions.

Learning points

- One of the common complications of spinal anaesthesia is hypotension.
- Sensory block height, age, body mass index (BMI), chronic alcohol consumption, history of hypertension and urgency of surgical operations have been discovered to be independent risk factors leading to the occurrence of intra-operative hypotension after a spinal block.
- Regulating sensory block height, closely monitoring trends in blood pressure and heart rate as well as responding rapidly and critically to these shifts are the relevant strategies for disaster avoidance.

Contributors TMA was the major contributor in studying the case, writing the manuscript and was involved in the medical care of the patient. MBM assisted with literature review. CP edited the report and she was the medical consultant responsible for the patient management.

Competing interests None.

Patient consent Obtained.

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