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Intravenous Dexamethasone as an Adjuvant to Stellate Ganglion Block for Refractory Cerebral Vasospasm After Aneurysmal Subarachnoid Haemorrhage: A Case Report

Francisco Vaz Pereira [1], Joana Rodrigues [1], José Miguel Guerreiro [1]

1. Anaesthesiology, Unidade Local de Saúde de São José, Lisbon, PRT

Corresponding author: Francisco Vaz Pereira, vazpereira.francisco@gmail.com

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Abstract

Refractory cerebral vasospasm after aneurysmal subarachnoid haemorrhage (aSAH) is associated with delayed cerebral ischaemia (DCI) and remains difficult to manage when systemic and endovascular therapies fail. Stellate ganglion block (SGB) is a bedside option that may attenuate the sympathetic component of vasospasm, but its effect is limited by the short duration of local anaesthetics. We describe the case of a 60-year-old woman with a modified Fisher Grade IV, Hunt and Hess Grade 3 aSAH from an anterior communicating artery aneurysm, treated with stent-assisted coiling, who developed symptomatic vasospasm refractory to systemic milrinone and intra-arterial therapy. A first ultrasound-guided SGB with ropivacaine was followed by clinical and sonographic improvement and Horner's syndrome lasting less than 24 hours. A second block, with intravenous dexamethasone 8 mg, was followed by a longer-lasting Horner's syndrome (approximately 43 hours), consistent with sustained sympathetic blockade, and by sustained clinical improvement that allowed milrinone to be weaned. No haemorrhagic complications occurred under dual antiplatelet therapy. At six months, her modified Rankin Scale score was one. Because intravenous dexamethasone has anti-inflammatory effects independent of the block, and because concurrent therapies and the natural course of vasospasm may have contributed, the observed response cannot be attributed to prolonged sympathetic blockade alone. This single observation does not establish efficacy; it generates the hypothesis that intravenous dexamethasone may prolong SGB while adding an independent anti-inflammatory effect, which warrants evaluation in controlled studies.

Categories: Neurosurgery, Anesthesiology
Keywords: aneurysmal subarachnoid haemorrhage, cerebral vasospasm, delayed cerebral ischaemia, dexamethasone, regional anaesthesia, stellate ganglion block, sympathetic block

Introduction

Aneurysmal subarachnoid haemorrhage (aSAH) is a severe subtype of stroke, with a worldwide incidence of approximately six to nine per 100,000 person-years [1,2]. Despite early aneurysm occlusion, nearly 30% of survivors develop delayed cerebral ischaemia (DCI), a major cause of long-term disability and mortality [3]. Angiographic vasospasm and DCI are related but distinct entities: vasospasm describes arterial narrowing on imaging and occurs in up to 70% of patients, whereas DCI is a clinical and radiological diagnosis of ischaemia that may occur without vasospasm and also involves mechanisms such as microvascular thrombosis and cortical spreading depolarisation [3]. Vasospasm, nevertheless, remains significantly associated with territory-specific ischaemia and poor neurological outcome [3].

Standard management focuses on the prevention of DCI with oral nimodipine and maintenance of euvolaemia and normotension [2]. When medical therapy fails, endovascular rescue therapies, such as intra-arterial vasodilator infusion or balloon angioplasty, are used. These therapies are invasive, carry procedural risks such as vessel dissection or rupture, and the effect of intra-arterial drugs is often transient, which may require repeated interventions and repeated transport of unstable patients to the angiography suite [4].

The cerebral vasculature receives dense sympathetic innervation from the cervical sympathetic chain, and the sympathetic surge that follows aSAH may contribute to both large-vessel narrowing and microcirculatory dysfunction [5]. Stellate ganglion block (SGB) interrupts this sympathetic outflow to the ipsilateral cerebral vasculature, which may reduce vascular tone, particularly in distal arterial beds, and thereby improve cerebral blood flow at the bedside, with less systemic haemodynamic instability than is often seen with intra-arterial therapy [5,6]. The clinical evidence for SGB in this setting, however, remains limited to small and heterogeneous studies [6].

A practical limitation of SGB is the short duration of local anaesthetics, which may allow early recurrence of vasospasm as the block wears off. In regional anaesthesia, intravenous dexamethasone at doses of 8 to 10 mg prolongs the duration of sensory block and analgesia after peripheral nerve blocks [7]. This evidence derives exclusively from somatic nerve blocks; whether dexamethasone prolongs autonomic sympathetic blockade

has not been established, and any such effect remains hypothetical. In aSAH, dexamethasone also has an additional and independent action, as glucocorticoids exert anti-inflammatory effects that may mitigate the early brain injury and neuroinflammation triggered by subarachnoid blood [8].

We describe a case of refractory symptomatic vasospasm in which two SGBs were followed by clinical and sonographic improvement, with a longer-lasting response after intravenous dexamethasone was added to the second block. To our knowledge, the use of intravenous dexamethasone as an adjuvant intended to prolong SGB in refractory cerebral vasospasm has not been previously reported. This report does not establish the efficacy of this combination; its purpose is to describe the observation and to discuss why the contributions of sympathetic blockade, dexamethasone and concurrent therapies cannot be separated.

Case Presentation

On Day 1, a 60-year-old woman (165 cm, 70 kg; body mass index 25.7 kg/m^2) with a history of arterial hypertension, dyslipidaemia and spondyloarthropathy treated with methotrexate presented to the emergency department with severe headache and hypertension. The presentation was attributed to a hypertensive crisis, and she was discharged on antihypertensive and analgesic therapy without imaging. On D6, she returned with neurological deterioration (Glasgow Coma Scale (GCS) [9] score of 13, E3V4M6) and oculocephalic deviation to the left. Computed tomography revealed diffuse thick subarachnoid haemorrhage with intraventricular haemorrhage, modified Fisher [10] Grade IV and Hunt and Hess [11] Grade 3 (Figure *1A*). Digital subtraction angiography identified a wide-necked anterior communicating artery aneurysm, which was treated with stent-assisted coiling (Figures *1B*, *1C*), after which dual antiplatelet therapy with aspirin and ticagrelor was started. Day 1 was considered the day of the haemorrhage. Mild bilateral vasospasm of the internal carotid artery termini, more pronounced on the left, was noted during the procedure and treated with intra-arterial verapamil (5 mg on the right, 10 mg on the left). She was admitted to the intensive care unit, and nimodipine, normotension, euvolaemia and a systemic milrinone protocol (0.75 mcg/kg/min) were started, with clinical recovery: the headache resolved and the GCS improved to 15.

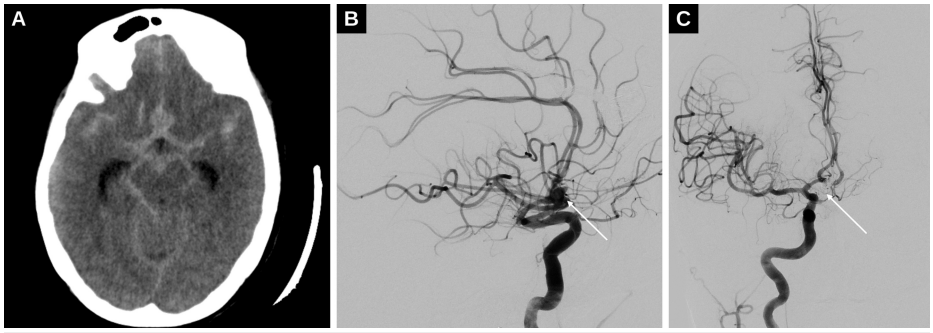


FIGURE 1: Initial neuroimaging and endovascular treatment of the ruptured aneurysm.

(A) Non-contrast head computed tomography on D6 showing diffuse thick subarachnoid haemorrhage with intraventricular haemorrhage (modified Fisher grade IV). (B) Digital subtraction angiography, lateral projection of the right internal carotid artery injection, before embolization, showing a wide-necked anterior communicating artery aneurysm (arrow). (C) Digital subtraction angiography, anteroposterior projection of the right internal carotid artery injection, after stent-assisted coiling, showing the coil mass (arrow).

On Day 11, transcranial Doppler (TCD) showed moderate-to-severe vasospasm of the right anterior cerebral artery (ACA). Despite escalation of systemic milrinone and intra-arterial milrinone on Day 11, digital subtraction angiography on the morning of Day 13 showed persistent severe vasospasm of the right A1 segment and moderate vasospasm of the distal ACA branches and of both middle cerebral arteries (MCA), and intra-arterial milrinone was repeated. By then, the patient had developed headache and mild disorientation (GCS 14), interpreted as symptomatic vasospasm. Alternative causes of neurological deterioration were excluded by repeat head computed tomography, serum sodium measurement, which was normal, and a laboratory work-up without abnormalities that could account for the deterioration.

Because vasospasm remained refractory despite systemic milrinone at 1 mcg/kg/min, a first right SGB was performed in the afternoon of Day 13, after the morning intra-arterial treatment. Both blocks were performed without sedation, under real-time ultrasound guidance with colour Doppler to identify and avoid vascular structures along the needle path (Figure *2*), and under dual antiplatelet therapy; the bleeding risk was weighed against the expected benefit in refractory symptomatic vasospasm. The first block used 10 mL of 0.75% ropivacaine. An ipsilateral Horner's syndrome appeared, and the headache and disorientation resolved (GCS 15). Mean flow velocity fell by 42% in the right ACA and by 11% in the right MCA, and the left MCA velocity fell below the vasospasm threshold (Table *1*). The response was short-lived: by the morning of Day 14, the Horner's syndrome had resolved, right ACA velocity had increased again, and milrinone was

increased to 1.5 mcg/kg/min (Table 1).

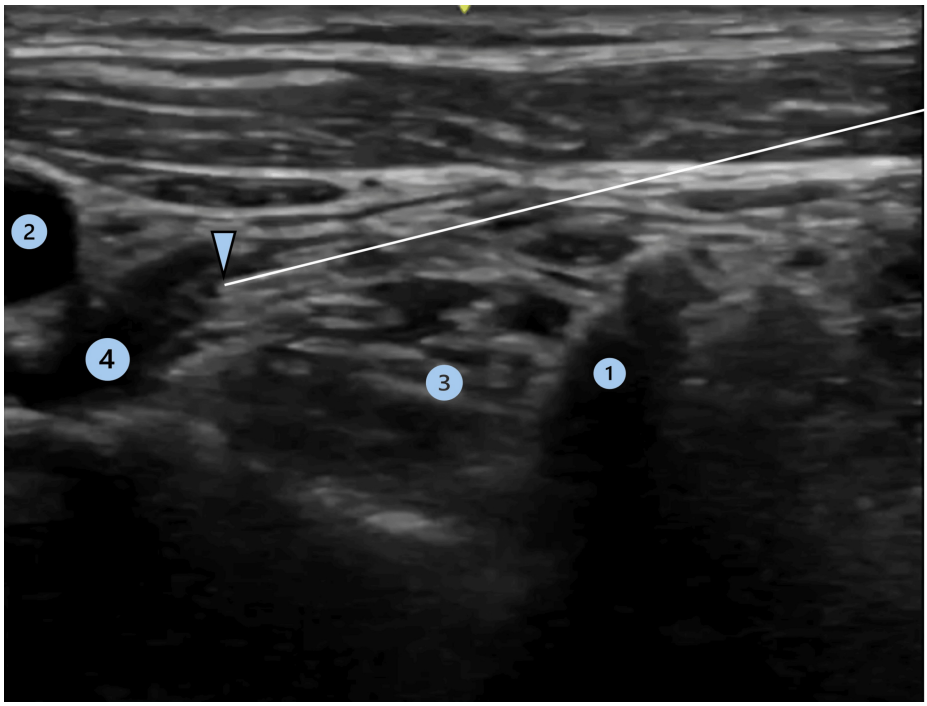


FIGURE 2: Ultrasound-guided stellate ganglion block technique

The block was performed with the patient supine and the neck slightly extended, using a high-frequency linear transducer and colour Doppler to identify vascular structures. The anterior tubercle of the sixth cervical vertebra (Chassaignac tubercle, (1) was the primary landmark. Under real-time visualisation of the carotid artery (2) and longus colli muscle (3), a 50-mm needle was advanced in-plane, laterally to medially, through the prevertebral fascia; the needle tip is indicated by the arrow. Local anaesthetic (4) was deposited over the longus colli muscle, deep to the prevertebral fascia, with spread around the cervical sympathetic trunk. The internal jugular vein is not visible because it was compressed by transducer pressure. The first block used 10 mL and the second 7 mL of 0.75% ropivacaine; intravenous dexamethasone 8 mg was given with the second block. Sympathetic blockade was confirmed clinically by an ipsilateral Horner's syndrome.

Day/event	ACA R	MCA R	ACA L	MCA L	Milrinone	IA therapy	Clinical status	Horner
Day 13 pre-block	203 (severe)	149 (mild), LI 4.5	No VS	149 (mild), LI 3.6	1.0	Milrinone (morning)	Headache, confusion, GCS 14	—
Day 13 post block (First SGB)	118 (no VS; −42%)	132 (mild; −11%), LI 3.2	130 (mild)	No VS	1.0	—	No headache, GCS 15	Present
Day 14	151 (mod)	No VS	No VS	124 (mild), LI 3.5	1.5	—	Mild headache, GCS 15	Absent
Day 15 pre-block	150 (mod)	135 (mild), LI 3.2	No VS	No VS	1.5	—	Headache, confusion, GCS 14	Absent
Day 15 post block (second SGB + dexamethasone 8 mg)	134 (mild; −11%)	No VS	No VS	No VS	1.5	—	No headache, GCS 15	Present
Day 16	139 (mild)	No VS	No VS	No VS	1.0	—	No headache, GCS 15	Present
Day 17	139 (mild)	No VS	No VS	No VS	0.75	—	No headache, GCS 15	Resolved (morning, ~43 h)
Day 18	134 (mild)	No VS	No VS	138 (mild), LI 3.4	Stopped	—	No headache, GCS 15	Absent
Day 19	122 (mild)	No VS	No VS	No VS	Stopped	—	No headache, GCS 15	Absent

TABLE 1: Chronological evolution of TCD flow velocities, milrinone dose, and clinical/neurological status.

Mean flow velocities in cm/s (severity; percentage reduction from the preceding pre-block value); Systemic milrinone in mcg/kg/min. Both blocks were performed on the right side. Intra-arterial verapamil was administered during aneurysm treatment on Day 6, and intra-arterial milrinone on Day 11 and on the morning of Day 13; no endovascular therapy was performed after Day 13. VS thresholds: no VS <120 cm/s, mild 120–149 cm/s, moderate 150–199 cm/s, severe ≥200 cm/s; these thresholds were validated for the MCA and were applied to the ACA by extrapolation. The LI was calculated for the MCA only; all values were >3, consistent with vasospasm. Changes in TCD velocities do not necessarily reflect changes in cerebral perfusion.

Abbreviations: ACA, anterior cerebral artery; MCA, middle cerebral artery; R, right; L, left; LI, Lindegaard index; VS, vasospasm; mod, moderate; IA, intra-arterial; SGB, stellate ganglion block; TCD, transcranial Doppler; GCS, Glasgow Coma Scale; h, hours

On Day 15, the patient's condition worsened again, with headache and confusion (GCS 14), and TCD showed moderate vasospasm of the right ACA. Because the first block had lasted less than 24 hours and was followed by recurrence, a second right SGB was performed with intravenous dexamethasone 8 mg, given immediately after the block, with the aim of prolonging its duration; this dose lies within the range associated with the maximal block-prolonging effect of intravenous dexamethasone [7]. As adequate spread around the cervical sympathetic trunk was observed under ultrasound, 7 mL of 0.75% ropivacaine was used. No endovascular therapy was performed after Day 13. The second block was followed by an ipsilateral Horner's syndrome that persisted for approximately 43 hours, until the morning of Day 17, with resolution of headache and confusion (GCS 15), and by an 11% fall in right ACA mean flow velocity, with no vasospasm in the remaining vessels (Table *1*). Milrinone was weaned from Day 16 and stopped on Day 18, without recurrence of neurological symptoms. After both blocks, mean arterial pressure remained above 70 mmHg, with a normal heart rate in sinus rhythm, and no hypotension, bradycardia, haemorrhagic complications or hyperglycaemia occurred.

The patient was transferred to the ward on Day 18. A mild left hemiparesis was identified later during the hospital stay; its onset was not documented, and follow-up computed tomography showed no new infarction. She was discharged on Day 24, able to perform all her usual activities. Six months after discharge, her modified Rankin Scale [12] score was one, and her Glasgow Outcome Scale [13] score was five (good recovery), with mild left hemiparesis as the only residual deficit.

Discussion

The cerebral vasculature receives dense sympathetic innervation from the cervical sympathetic trunk, and experimental work has shown that sympathetic activation constricts cerebral arteries, whereas its inhibition produces vasodilation [5]. The sympathetic surge that follows aSAH provides the physiological rationale for SGB as a bedside modulator of the neurogenic component of vasospasm. The vascular effect of SGB appears to act predominantly on distal and microvascular beds: angiographic studies have reported improved cerebral perfusion without a measurable change in the diameter of proximal vessels, whereas TCD studies have shown a fall in flow velocities of approximately 15%, consistent with downstream vasodilation [5,14]. Falls of similar magnitude were observed in this patient. Reduced TCD velocities, however, do not necessarily indicate improved cerebral perfusion, and perfusion was not measured in this case. The clinical improvement despite persistently elevated velocities in the right ACA is consistent with an effect not fully captured by TCD, but cannot confirm it. Although both blocks were performed on the right side, velocities also fell in the contralateral MCA after the first block. This may reflect the natural course of vasospasm, the same-day intra-arterial milrinone or the operator-dependent variability of TCD, and only raises the possibility of an effect extending beyond the injected side.

The clinical evidence for SGB in this setting is limited to small and heterogeneous studies, but it is generally favourable. A systematic review and meta-analysis by Brenner et al., pooling eight studies and 182 patients, reported favourable outcomes in 52% of patients, a decrease in cerebral blood flow velocity in the studies that measured it, and a complication rate of 2% [6]. Wendel et al. documented a reduction in MCA velocities after SGB [14], Samagh et al. reported a fall in mean MCA velocity after the block [15], and a pilot randomised trial suggested improved flow velocities when SGB was applied early after aSAH [16]. Treggiari et al. had earlier shown that cervical sympathetic block could reverse delayed ischaemic neurological deficits [17], and SGB combined with intra-arterial therapy has been proposed as a single-session strategy for refractory cases [18]. This case adds a bedside observation: the duration of an ipsilateral Horner's syndrome, a clinical marker of cervical sympathetic blockade, paralleled the duration of the clinical and sonographic response.

Continuous cervical sympathetic blockade through an indwelling catheter has been described in this setting and avoids repeated procedures, but whether it improves vasospasm and DCI outcomes or simply adds risk remains undetermined [5]. In a patient requiring serial neurological examination, a continuous infusion at the level of the sixth cervical vertebra carries a cumulative risk of spread to the recurrent laryngeal and phrenic nerves, with hoarseness, dysphagia or respiratory compromise that could confound neurological assessment, in addition to the risks of catheter displacement and infection in the neurocritical care setting. A single-injection block, by contrast, is rapidly performed at the bedside, can be repeated and produces a Horner's syndrome with a discrete onset and offset. In this patient, both blocks were performed under dual antiplatelet therapy without haemorrhagic complications, with ultrasound and colour Doppler used to avoid vascular structures along the needle path.

Two distinct mechanisms must be considered separately when interpreting the response to the second block: a possible prolongation of sympathetic blockade by dexamethasone, and a direct anti-inflammatory effect of dexamethasone that is independent of the block. These cannot be disentangled in this case. Regarding the first, intravenous dexamethasone prolongs the duration of peripheral nerve blocks [7]; it was added because the first block lasted less than 24 hours and was followed by recurrence of symptomatic vasospasm while the patient remained within the period of risk. The Horner's syndrome lasted less than 24 hours after the first block and approximately 43 hours after the second, and the second block coincided with a sustained response that allowed milrinone to be weaned. Two features of the sequence bear on this comparison. The second block used a lower volume of local anaesthetic (7 vs 10 mL of 0.75% ropivacaine), which would be expected to shorten rather than prolong it, and, unlike the first, it was not accompanied by any endovascular therapy. The difference between the two blocks is therefore consistent with the intended effect of the adjuvant, but it also coincided with the later phase of vasospasm, when spontaneous resolution becomes more likely, and a single intra-patient comparison cannot establish that the combination is superior to SGB alone. Perineural dexamethasone could be considered as an alternative, but it offers no greater maximum effect than the intravenous route [7].

The second mechanism is independent of the block. Inflammation contributes to the pathophysiology of DCI, with elevated inflammatory mediators in the cerebrospinal fluid and a systemic inflammatory response that predicts poor outcome [8]. In a propensity-score analysis of 309 patients, Mohney et al. found that dexamethasone was associated with better functional outcome at discharge and a reduced systemic inflammatory response, but not with a significant reduction in angiographic vasospasm, sonographic vasospasm or DCI [19]. A randomised trial of high-dose methylprednisolone similarly found no reduction in DCI but an improvement in long-term outcome [20]. Part of the favourable evolution in this patient may therefore reflect an anti-inflammatory effect of dexamethasone acting in parallel with, rather than through, the sympathetic block. Conversely, the evidence that dexamethasone does not reliably reduce vasospasm argues against attributing the fall in TCD velocities to the drug itself. The longer-lasting Horner's syndrome is the most direct available sign that the adjuvant may have extended the sympathetic blockade, but it is a clinical surrogate and not a validated measure of cerebral sympathetic blockade. The possible combined effect of sustained sympathetic blockade and systemic anti-inflammatory action is therefore a hypothesis

generated by this case, not a conclusion demonstrated by it.

This report has several limitations. It describes a single patient, and the natural course of vasospasm, which typically resolves after the second week, may have contributed to the improvement that followed the second block. The patient received multiple concurrent treatments, including nimodipine, escalating systemic milrinone, intra-arterial verapamil and milrinone, and two SGBs, so the effect of any single intervention cannot be isolated; in particular, the response to the first block cannot be separated from the intra-arterial milrinone given on the same morning. The response was monitored with TCD and clinical examination only: cerebral perfusion imaging was not performed, and the velocity thresholds used for the ACA were extrapolated from those validated for the MCA. The duration of Horner's syndrome was assessed clinically at the bedside and was not precisely timed; exact clock times of measurements were not systematically recorded, and Horner's syndrome has not been validated as a measure of cerebral sympathetic blockade. Finally, a mild left hemiparesis was identified later in the hospital stay; although follow-up computed tomography showed no new infarction, its onset was not documented, and DCI cannot be excluded. These limitations confirm that the value of this report lies in generating a hypothesis that warrants evaluation in controlled studies.

Conclusions

This case describes two ultrasound-guided SGBs used as a bedside rescue therapy for symptomatic cerebral vasospasm refractory to systemic and endovascular treatment. After intravenous dexamethasone 8 mg was added to the second block, the Horner's syndrome lasted longer (approximately 43 vs less than 24 hours), consistent with sustained sympathetic blockade, and coincided with sustained clinical improvement that allowed milrinone to be weaned. These are temporal associations. The favourable outcome may reflect the combined effect of concurrent therapies and the natural resolution of vasospasm, and intravenous dexamethasone has anti-inflammatory effects independent of the block. The combination of prolonged sympathetic blockade and anti-inflammatory action is therefore a hypothesis to be tested in prospective controlled studies, not a basis for any recommendation regarding the efficacy of intravenous dexamethasone as an adjuvant to SGB.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Francisco Vaz Pereira, José Miguel Guerreiro, Joana Rodrigues

Acquisition, analysis, or interpretation of data: Francisco Vaz Pereira

Drafting of the manuscript: Francisco Vaz Pereira

Critical review of the manuscript for important intellectual content: José Miguel Guerreiro, Joana Rodrigues

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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