Intrathecal Sodium Nitroprusside for the Treatment of Delayed Cerebral Ischemia Secondary to Refractory Vasospasm after Aneurysmal Subarachnoid Hemorrhage

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INTRODUCTION

Vasospasm is one of the most feared complications of aneurysmal subarachnoid hemorrhage (aSAH). It can be assymptomatic or lead to delayed cerebral ischemia (DCI), having a major impact on functional outcome.

OBJECTIVE

To report a case of a aSAH patient who developed refractory symptomatic vasospasm (VS) with DCI, treated with intrathecal sodium nitroprusside as a rescue therapy.

CASE REPORT

A 50 year-old hypertensive and diabetic female had a thunderclap headache 3 days before hospital admission, with symptoms of decreased level of consciousness, and a head CT scan (Figure 1) showing SAH (modified Fisher scale 3) and hydrocephalus, treated with external ventricular drainage.

***Figure 1**. Admission Brain CT of admission, with SAH classified as a modified Fisher Scale 3.*

Cerebral angiography showed a 2mm left superior cerebellar artery aneurysm, that was treated with endovascular coiling.

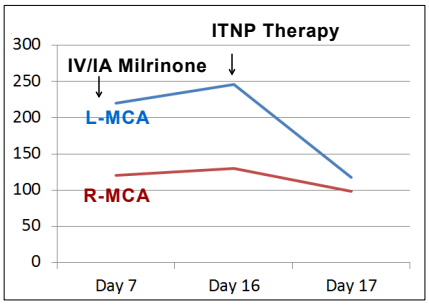

***Figure 2.** Mean flow velocities (cm/sec) in R-MCA and L-MCA during treatments for refractory VS. ITNP, intrathecal nitroprusside.*

Severe VS developed on day 7 of bleeding, with transcranial Doppler (TCD) parameters: Median flow velocity (MFV) of 220cm/s and Lindegaard index (LI) of 9.8 in the left middle cerebral artery (L-MCA); MFV of 120cm/s with LI of 4.6 in R-MCA.

Persistent VS was sequentialy treated with induced hypertension (intravenous noradrenaline), cerebral angiography with intraarterial infusion of milrinone, and subsequent continuous IV milrinone.

On day 16 of ictus, despite of aggressive VS treatment, TCD parameters worsened, as shown in Figure 2, with LI exceeding 10 in L-MCA, and 4,6 in R-MCA. Hypoperfusion in the left hemisphere was detected on CT perfusion study. Intrathecal administration of nitroprusside was therefore initiated (5 mg in bolus, through a solution of 2 mL of sodium nitroprusside and 8 mL of cerebral spinal fluid).

Soon after the administration, the patient developed unstable atrial fibrillation, requiring 5 electrical cardioversions.

On the next day, LI and TCD velocities improved (Figure 2 and 3), and there was only mild vasospasm on an angiogram.

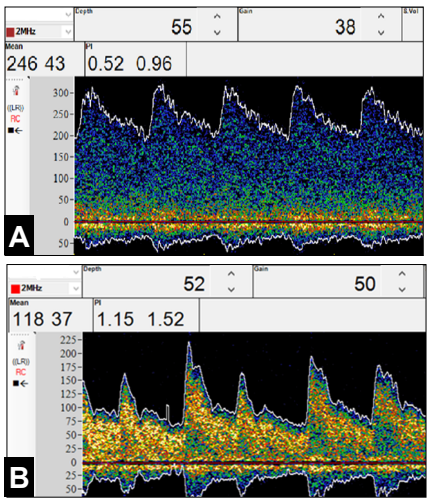

***Figure 3.** L-MCA transcranial Doppler, showing mean flow velocities before (A) and after (B) intrathecal Nitroprusside infusion.*

DISCUSSION

Intrathecal nitroprusside for the treatment of refractory severe VS is reported in the literature since 1999, with small case series showing improvement in up to 76% of patients. In our case, this treatment have led to improvement of TCD parameters in less than 24 hours after drug infusion.

Hypotension, nausea and vomiting are the most common adverse effects in previous studies. There are few descriptions of arrhythmia, but we could not find any other cause leading to severe instability in our case.

CONCLUSION

Intrathecal nitroprusside seems to be a promising therapy for refractory VS secondary do aSAH. Future studies may clarify its safety and clinical benefit for this population.

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