

Use of Adenosine Cardiac Arrest to Facilitate NBCA Treatment of High Flow Vein of Galen Malformation in Two Infant Cases

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Introduction

Vein of Galen Aneurysmal Malformations (VGAM) are congenital high flow shunt systems that most commonly present in the neonatal and infancy stages of life and account for <1% of cerebrovascular lesions. The treatment paradigm has had many changes from open vascular to endovascular to combined treatment. The primary goal is reduction of clinical symptoms and not full obliteration of the lesion especially early in life. We present two infant (2 month and 3 month) patients with VGAM that developed evidence of worsening hydrocephalus related to venous congestion associated with a high flow mural type A-V shunt. To improve the distal treatment of each pedicle with NBCA glue; the high flow system was diminished by achieving temporary cardiac arrest using adenosine.

Methods

In the first case, a 4F catheter was advanced to the left internal carotid artery. Next, a microcatheter and microwire were used to access the right pericallosal artery. Once the level of the fistula was reached maximal Esmolol drip was used to decrease flow to the lesion without success. The decision was made to induce cardiac pause with adenosine to allow controlled embolization. NBCA glue was prepared and once half of the catheter dead space was reached adenosine provided five seconds of asystole and 5 seconds of bradycardia allowing complete occlusion of the pedicle. This process was repeated in two other pedicles resulting in significant flow reduction. This technique was replicated at the onset of treatment of our second patient with similar success. For each case we used a test dose of 1.5mg of adenosine to determine the extent and timing of conduction block and bradycardia provided prior to the treatment doses. First case had central venous access and the dosing for the treatment runs were 1.5mg, 1.5mg, and 2mg respectively. Second case did not have central venous access and required a larger treatment dose of 3mg and 6mg with still a less predictable time of arrest. Both cases the goal was to slow the flow to the malformation and not attempt complete cure.



Results

Following embolization follow-up imaging showed continued resolution of the hydrocephalus in each patient. The second patient was shunted by the pediatric service just after embolization. Both patients are well at 3 years and 10 months follow-up, respectively.

Conclusions

Adenosine induced cardiac arrest is a safe option to facilitate treatment of VGAM with NBCA glue in infants where balloons and multiple catheters are unable to be utilized. Test dosing should be used through a central venous catheter to determine dosing and timing of adenosine and glue administration.

Learning Objectives

By the conclusion of this session, participants should be able to 1) Describe the characteristics of high flow VGAM that may make them difficult to treat even with fast setting NBCA glue, 2) Identify which patient would benefit from adenosine cardiac arrest during endovascular treatment, 3) Discuss, in small groups how to prepare the anesthesia and endovascular teams for the complex steps required when using this technique.