An unreported life threatening vaso-respiratory disturbance during endovascular embolisation of Vein of Galen malformation- A case report

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Right ICA and left vertebral angiograms demonstrating arterial feeders, vein of Galen sac (arrow) and falcine sinus

The case:

- A 10 month old female weighing 9 kg
- Admitted for elective re-embolisation of her Vein of Galen Aneurysmal Malformation (VGAM).
- Presented 6 weeks earlier with seizures, diagnosed with VGAM and underwent her first embolisation, complicated by venous sinus thrombosis requiring therapeutic anticoagulation and anti-epileptics. Now, seizure free and well.
- Anaesthetic- Gas induction, endotracheal tube (ETT), radial arterial line, femoral central line.
- In the interventional radiology (IR) suite- Cerebral angiograms were obtained and a guide catheter positioned in the right ICA. A microcatheter was advanced into the posterior cerebral artery (PCA).
- The event- As soon as the microcatheter was in the PCA, a sudden significant drop in lung compliance was evidenced by a fall in end-tidal carbon dioxide (ETCO₂) and slow upstroke of the capnogram. Atracurium was given, sevoflurane was increased and equipment failure was ruled out. This was followed by severe hypotension and tachycardia.
- The management- Fluid boluses 10 ml/kg (x3), phenylephrine boluses (1-2 mcg/kg each) showed no effect. Adrenaline boluses (1 mcg/kg each) were given and an infusion was eventually needed (0.05- 0.5 mcg/kg/min). Noradrenaline infusion was also commenced.
- During this period, embolisation was performed in a right PCA feeder with Histoacryl glue uneventfully and the catheters were removed.
- Differential diagnoses and investigations- Pneumothorax, cardiogenic shock, internal bleeding, anaphylaxis were all ruled out by clinical means and investigations (CT head/chest, abdominal ultrasound, bed-side echocardiogram). Arterial blood gas analysis showed normal gas exchange and lactate.
- The procedure was abandoned. Within 20 minutes, the patient started recovering from the haemodynamic collapse and was weaned off of the vasopressor infusions. She was extubated and observed for half an hour. She was then sent to a high dependency bed.
- She came back for a repeat embolisation in 5 days. After a similar anaesthetic, her VGAM was approached via the vertebral artery instead of the ICA. She was noted to have a small, transient drop in lung compliance but was haemodynamically stable. She was electively transferred to an intensive care bed and was extubated the following day.

Background:

VGAM, although a rare condition (between 1 and 40 per 1,000,000 live births), forms about 30% of paediatric vascular malformations and about 1% of all congenital anomalies. It results from an abnormal shunting of arterial blood into the persistent median prosencephalic vein of Markowski, a precursor of the great cerebral vein of Galen. Neonates present in high output cardiac failure, whereas infants and children often present with symptoms secondary to hydrocephalus and/or venous hypertension. This second mode of presentation was the case with the child described in our case.

Treatment consists of supportive medical management (such as managing heart failure), reducing the arterio-venous shunt, and occasionally neurosurgical procedures to manage the hydrocephalus. For AV shunt reduction, embolization (with glue, ONYX or coils) is the main modality used in most centres.¹

We believe, this is the first case report illustrating an undescribed vaso-respiratory reaction during endovascular treatment of VGAMs

Discussion:

Autonomic disturbances, similar to trigeminocardiac reflex (TCR), have been reported in the past during superselective ophthalmic arterial chemotherapy (SOAC) for retinoblastoma² and selective angiography of the middle meningeal artery. These have been accompanied by bradycardia, hypotension and a decrease in respiratory compliance (40% or more) and have occurred when the catheter is in the cavernous part of the carotid artery or the ophthalmic artery. These may be due to stimulation of trigeminal afferents and abrupt changes in peripheral pulmonary vascular resistance³

We did not observe any bradycardia during our case. The complete resolution of the haemodynamic instability after removal of the intracranial catheters was very striking. It was interesting to note that during the second procedure, approaching the VGAM via the vertebral artery led to only a minor and momentary change in compliance but no hypotension or bradycardia.

Anaesthetists providing anesthesia for not just SOAC, but also VGAM embolisation and other intracranial IR procedures should be aware of the possibility of severe vaso-respiratory reactions. We believe that based on hitherto published cases, the existence of an autonomic pathway linking the intracranial vasculature and the respiratory system needs further investigation.

References:

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