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Case Report

Bilateral Subclavian Artery Stenosis: Anaesthetic consideration

her emergency laparotomy for large bowel obstruction secondary to diverticular disease. She was also known to smoke heavily, and as a result, developed chronic obstructive pulmonary disease (COPD). She had a previous stroke which left no residual neurological deficit. Her previous exposures to general anaesthesia reported to be uneventful. All of her medical problems were controlled with medication although she was not taking any treatment for high blood pressure and AF. The preoperative investigations were consistent with her existing medical problems.

In the anaesthetic room a routine measurement of non-invasive brachial artery blood pressure in both arms recorded only 78 mm Hg systolic and 52 mm Hg diastolic. The low blood pressure was reconfirmed invasively with a left radial artery cannula. An unusually low blood pressure in both limbs lead to a suspicion of bilateral subclavian artery stenosis and the surgery was postponed for that day pending further investigations.

A CT angiography (Figure 1) confirmed the diagnosis of bilateral subclavian artery stenosis; slightly more marked on the right than the left. The stenosis appeared to be downstream

Introduction

Subclavian artery stenosis (SAS) is a relatively rare condition, even more so for its bilateral existence. In a study [1], the prevalence of SAS was 1.9% in the free-living cohorts and 7.1% in the clinical cohorts. SAS was significantly associated with smoking and higher levels of systolic blood pressure. Higher levels of high-density lipoprotein cholesterol were inversely and significantly associated with SAS. In regression analyses relating SAS to other cardiovascular diseases, the only significant finding was with peripheral arterial disease. The presence of this condition leads to erroneously low blood pressure recorded in the ipsilateral brachial artery or radial artery. The conventional anaesthetic challenge for these patients could be maintaining organ perfusion (especially the cerebral perfusion) and thus avoiding ischaemic damage when the actual blood pressure is unknown. We report an interesting patient with bilateral subclavian stenosis who underwent prolonged surgery for a repair of massive parastomal hernia. This case was detected incidentally based on clinical findings. It was confirmed subsequently by CT angiogram. The surgery was performed under general anaesthesia and the patient was discharged home unharmed.

Case

A 63 year old female (weight 60.8 kg, and height 158 cm) was scheduled to undergo repair of a massive parastomal hernia as an elective procedure. She was known to suffer from arteriosclerosis, peripheral vascular disease, hypertension and atrial fibrillation (AF). She underwent carotid endarterectomy on her left side and coronary artery bypass graft (CABG) the year before. Subsequently, a parastomal hernia developed following



Figure 1: CT Angiogram showing Bilateral Subclavian Artery Stenosis.

of the vertebral artery origins on both sides. The circle of Willis was seen to be supplied by the left internal carotid and both vertebral arteries. The right internal carotid was occluded from its origin. A vascular surgical opinion confirmed that preoperative angioplasty for subclavian stenosis was unwarranted because she was asymptomatic, as well as that the risks of angioplasty would outweigh the potential benefits. She was rescheduled for parastomal hernia repair.

On the day of surgery, she was connected to monitors including invasive arterial cannula in the left radial and right femoral artery, Electrocardiogram (ECG), pulse oximeter, Bispectral Index (BIS) monitor and non-invasive cardiac output monitor (CHEETAH NICOM™ - Bioreactance). A baseline measurement of left carotid blood flow was made using a Doppler probe by a vascular scientist. We used a standard and balanced anaesthesia technique using Propofol, Remifentanyl and Atracurium for induction and nitrous oxide, oxygen with Desflurane and Remifentanyl infusion for maintenance. A BIS in the range of 20 -40 was maintained throughout. The central venous pressure was monitored via a central line inserted in right subclavian vein after induction of anaesthesia. We aimed to maintain blood pressure and cardiac output close to her baseline values and that was achieved using vasoactive compounds and intravenous fluid as and when necessary. The carotid artery blood flow was monitored and measured throughout the procedure. The anaesthesia and surgery lasted for 8 hours with a blood loss of 500ml. She was normothermic at the end of the procedure. The surgical procedures performed on her included open strattice mesh repair of parastomal hernia, division of adhesion and Hartmann's procedure. Following an uneventful extubation in theatre, she was observed in a high dependency unit (HDU). During her 6 day stay in HDU, she needed respiratory support in the form CPAP and supplemental oxygen. She did not suffer any neurological deficit or any other organ dysfunction. A successful discharge from hospital was followed after 6 more days in hospital.

Discussion

Subclavian artery stenosis, especially the bilateral, is a rare condition [1,2]. A clinical diagnosis of one sided subclavian artery stenosis can be suspected if the brachial artery blood pressure of one side is 15 mm Hg more than other side [3,4]. However, the bilateral subclavian artery stenosis diagnosis could be more challenging.

The presence of subclavian artery stenosis leads to erroneously low blood pressure values when measured at the brachial or radial artery on the affected side. The error in blood pressure measurement could lead to inappropriate clinical management, especially if the existence of this condition is unknown either to the patient or the clinician. In the presence of erroneously low blood pressure, especially during anaesthesia where haemodynamic disturbances are very common, it is impossible to know the actual blood pressure and hence cerebral blood flow and cerebral perfusion. The challenge

for us, the anaesthetists, is to maintain actual level of blood pressure and thus maintain cerebral and other organ perfusion. This particular patient was more challenging because of the already compromised cerebral blood flow secondary to the complete blockage of her right internal carotid artery.

We aimed to maintain baseline haemodynamic parameters including radial artery pressure and cardiac output with or without the help of fluid, inotropes and vasoactive compounds. By doing that we successfully maintained internal carotid blood flow so that it was more or less constant throughout the procedure. There were a few episodes of significant fluctuations in blood pressure which were managed quickly to restore the near normal baseline values. However, we noticed that even in the presence of significant changes in blood pressure from the baseline values, the left carotid artery blood flow was well maintained throughout. In addition to the radial artery cannulation, we also cannulated the right femoral artery. But for the logistic surgical reason, we were unable to utilise it.

There are other alternative ways to monitor cerebral oxygenation non-invasively. These include cerebral pulse oximeter which measures the regional cerebral oxygenation (rSO₂), transcranial Doppler, Laser Doppler, Diffuse Correlation Spectroscopy (DCS) and quantitative frequency-domain near-infrared spectroscopy [5]. These are a few of the many techniques that have been used to measure cerebral perfusion in anaesthetised patients. We planned to use transcranial Doppler, but the attempt to locate the vascular 'window' a few days before surgery was a failure. Hence, we had to rely on internal carotid artery flow measurement.

Conclusion

This was a high risk case for anaesthesia, surgery and post anaesthetic organ (especially the brain) dysfunction. This is more so because of the presence of significant arteriosclerosis and the occluded right internal carotid artery. However, we have noted that in spite of the presence of vascular disease and low erroneous brachial artery blood pressure, it is possible to maintain carotid artery blood flow (hence cerebral perfusion) if we maintain the blood pressure nearer the baseline values measured from the stenotic side of the circulation. We also noticed that even in the presence of transient but significant haemodynamic disturbances; the flow through the carotid artery was well maintained.

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