Carotid endarterectomy: A case study and literature review

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ABSTRACT

Carotid endarterectomy (CEA) is a preventative surgery that is performed in patient populations at risk of emboli due to an atherosclerotic plaque, located at the carotid bifurcation. Despite the advances in overall treatment, no consensus has been reached on the preferred anaesthetic for CEA. Improved procedural outcomes are due to the advances in medical therapy, cerebral monitoring, improved timing of surgery with improved surgical techniques and the use of ultrasound guided regional techniques. However, the choice of anaesthetic should consider the patient, the hospital's resources and the preferences of the anaesthesiologist and surgeon. SAHeart 2022;19:20-23

CASE REPORT

A 74-year-old female was scheduled for a left CEA. She presented 7 weeks prior to surgery with a 4 day history of dysarthria and expressive aphasia. Her symptoms improved within 2 weeks of her initial presentation without residual neurology. She has a background history of hypertension and dyslipidaemia controlled on an ACE inhibitor, calcium channel blocker and a statin, complicated by the above-mentioned non-disabling cerebral vascular accident (CVA). She was subsequently started on clopidogrel. She has no history of ischaemic heart disease or congestive cardiac failure. She was diagnosed in 2008 with rheumatoid arthritis, currently on disease modifying agents. She is a smoker with a 5 pack year history but reports cessation following her cerebrovascular insult, 7 weeks prior.

On physical examination she was non-distressed on room air, saturating at 92%. Her right and left arm blood pressure measurement was 120/80 and 115/65 respectively, showing minimal discrepancy. Her heart rate was regular at 77 beats per minute. Cardiovascular exam was unremarkable. On respiratory examination, there was no wheezing or features of hyperinflation. She had no neurological fallout and was oriented to person, place, and time. There was evidence of rheumatoid arthritis noted with radial deviation of both wrists and ulna deviation of fingers, with preservation of her airway, cervical spine, pulmonary, cardiovascular, and haematological systems. Laboratory findings were unremarkable, her haemoglobin and platelets were 12.3g/dL and 315 x 10^9/L respectively with a normal HBA1C and renal function.

A 12-lead ECG revealed left ventricular hypertrophy with inverted T waves in the inferolateral leads. Her chest X-ray showed fibrotic changes and calcification of the arch of the aorta. A 2D echocardiography was normal. A computed tomography angiogram of the carotids showed extensive atherosclerotic plaques in bilateral internal carotid arteries (cervical segment) and vertebral arteries (distally) with at least 55% stenosis (Figure 1). The carotid doppler revealed severe stenosis at the level of the left carotid artery bifurcation with 80% - 90% stenosis. Small plaques were visualised in the right common carotid artery and bifurcation.

The patient was discussed prior to surgery at a multidisciplinary team (MDT) meeting. A detailed plan was made regarding the perioperative management of the patient. One day prior to surgery the patient was re-examined, counselled, and consented. A thorough history was documented with all relevant investigations. Adequate advice was given on preoperative starvation. Two packed red cells were ordered for delivery to theatre prior to commencement of the case.

Clopidogrel was continued perioperatively as agreed at the MDT meeting.

The patient was cleaned and draped with her head slightly extended and turned to the right for adequate visualisation and dissection by the surgeons (Figure 2). Forty minutes after the start of surgery and dissection of the neck, a Javid shunt was inserted (Figure 3). There was maintenance of cerebral oxygenation throughout the procedure. She was transferred to ICU post operatively, awake with normal vitals and no neurological fallout. Twenty four hours after her surgery, she presented with increased blood pressures of 194/80. A labetalol infusion was started. Within 36 hours she was discharged to the ward, where she started to complain about a unilateral headache. On examination she was haemodynamically stable without any focal neurological signs or reported seizures. She was discharged home on day 5.

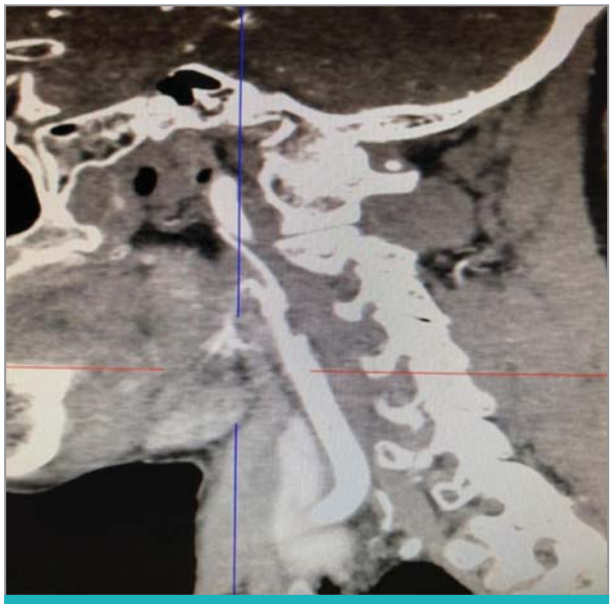

FIGURE 1: Sagittal plane CT angiogram showing narrowing/stenosis of the internal carotid artery.

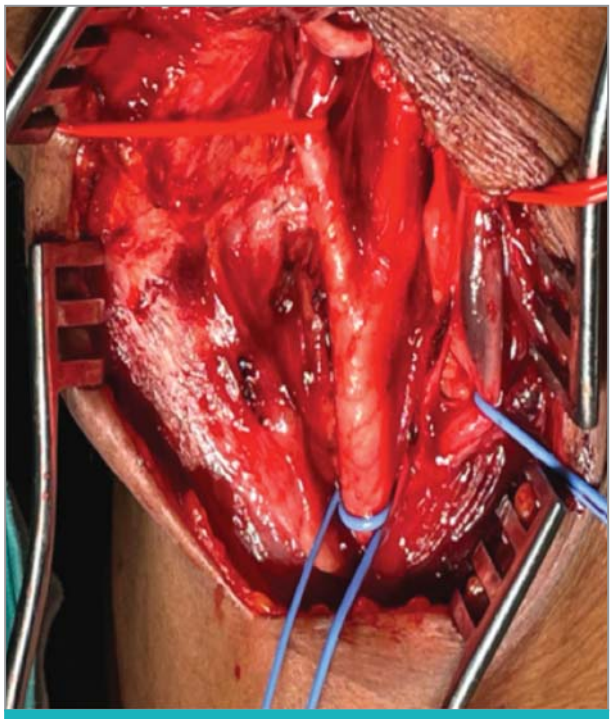

FIGURE 2: Exposure of the carotid bifurcation with the large atherosclerotic plaque causing stenosis.

DISCUSSION

In the developed world, CVA is the most common cause of adult neurological deficit.[1] CEA is a preventative surgery to prohibit disabling or terminal strokes in patient populations with significant carotid stenosis.[1]

There is indisputable evidence that promotes CEA in symptomatic patients with carotid stenosis of above 70% in the applicable carotid region.[1] Symptomatic patients are those that present with transient ischaemic attacks and non-disabling, reversible cerebral vascular accidents.[1,2] The most pronounced benefits of performing a CEA are obtained if the surgery is performed within 2 weeks of the last neurological symptom.[1,3]

The above-mentioned patient was referred to the vascular surgeons shortly after her CVA, which is uncommon in our hospital. It appears that most patients in our setting who do present, either present late after their CVA or are unfortunately

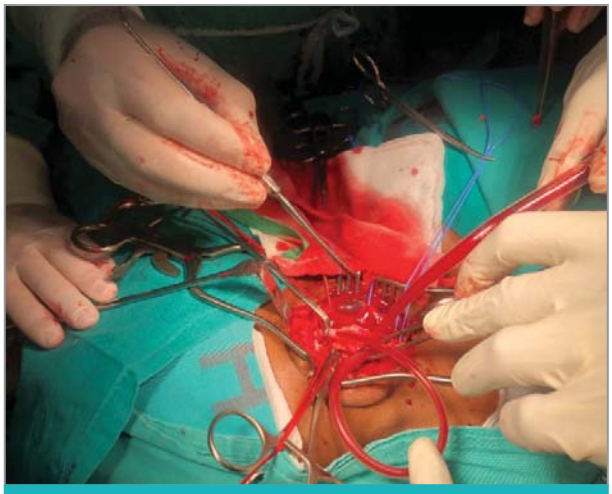

FIGURE 3: Shunt inserted across the clamped section of the carotid artery.

referred very late after their initial stroke to the vascular surgeons, due to a large patient volume, resulting in long waiting lists for investigation. This is evidenced by this case being our third in 7 years. Her neurological fallout was non-debilitating and reversible. She was subsequently medically managed and a stenosis of above 80% was noted on carotid doppler, considering her a good candidate for surgery.

CEA is not without significant complications.[1,4] Strokes and myocardial infarctions are amongst others serious adverse events in the perioperative period.[1,4] Carotid clamping, embolisation and thrombosis often result in perioperative neurological deficits.[1,4] Patients presenting with carotid disease commonly have related coronary artery disease.[1]

CEA is successful under local or general anaesthesia.[1,3] Superficial cervical plexus nerve blocks combined with deep cervical plexus nerve blocks are usually adequate with supplementation of local anaesthetic filtration by the surgeon.[1,3] There are however dangers to the use of deep plexus blocks.[3] Intra-arterial injection, injection into the cerebral spinal fluid, arterial injury and phrenic nerve damage are common complications.[3] Cervical epidurals may also be considered, although dural puncture, epidural venipuncture and respiratory muscle paralysis has been reported as major adverse anaesthetic complications.[1] As a result both are no longer performed. The greatest benefit of local anaesthesia is prompt neurological assessment of an awake patient.[3] Other advantages to local anaesthesia include preservation of coronary and cerebral autoregulation, avoidance of airway manipulation and general anaesthesia complications. The risk of excess sedation, patient stress and pain, limited access to the airway once surgery has commenced and possible patient claustrophobia are limitations to local anaesthetic techniques.[3,5]

Due to our surgical preference, our patient received a combination of local and general anaesthesia. A superficial and intermediate cervical plexus block was done with an addition of local anaesthetic by the surgical team upon closing of the operative site. No supplemental analgesia was necessary intraoperatively, which proved the substantial benefit of the combined technique.

There is no data advocating for a particular general anaesthetic technique.[1] The secure airway, decreased patient anxiety and neuroprotection with volatile anaesthesia are of additional benefit. Sevoflurane has been argued to be the most favourable volatile for neuroanaesthesia.[1,3] Nitrous oxide should be avoided due to the increase in cerebral metabolic rate and risk of air embolism.[1,3] Cardiovascular instability is commonly observed during CEA due to the impairment of autoregulation, decreased sensitivity of the baroreceptors due to atherosclerosis, increased age, the haemodynamic effects of carotid cross clamping, anaesthesia, diabetes and antihypertensive drugs.[6] Invasive arterial blood pressure monitoring is essential and maintenance of systolic blood pressures below 170mmHg and within 20% of the patient's baseline pressures is recommended.[1,3]

Our patient had increased preoperative blood pressures, was elderly and on antihypertensive agents which accounted for her cardiovascular lability on induction. The agents needed to maintain and induce general anaesthesia also contributed to the cardiovascular liability. It is therefore important to have vasoactive drug infusions pre-drawn in anticipation of haemodynamic instability perioperatively and access to a central venous line. On discharge from ICU, she complained about a severe unilateral headache which was assessed as a lack of adequate analgesia prescribed following discharge from ICU. Cerebral hyperperfusion syndrome was excluded as she responded well to the analgesia. She had no neurological fallout.

Cerebral hyperperfusion syndrome is an immoderate form of cardiovascular instability which occurs 2 - 7 days after surgery. These patients commonly present with severe ipsilateral headaches, hypertension, seizures and focal neurological deficits.[6] Literature attributes the pathophysiology of this syndrome to ischaemic reperfusion with impaired cerebral autoregulation.[6] Tight arterial blood pressure control is mandatory to prevent cerebral oedema.[6] Measures to mitigate hypertension associated with extubation should be taken as well as postoperative nausea and vomiting associated with head and neck surgery. Neurological monitoring is of great importance during a carotid endarterectomy.[1] Stump pressure is the pressure measured in the internal carotid artery after the common and external carotids are clamped. This pressure reflects the perfusion pressure of the Circle of Willis.

Thresholds between 25mmHg and 75mmHg have been suggested, below which shunting is appropriate.[1,7,8] Near infrared spectroscopy reports regional cerebral oxygenation. Carotid cross clamping results in a decrease in regional cerebral oxygenation. Other documented neurological monitors described in literature are: EEG, somatosensory evoked potentials and transcranial dopplers. All of which have benefits and disadvantages.[1]

In this case, near infrared spectroscopy was the neurological monitor used (as it was the only monitor available in our setting). It provided prompt identification of decreases in cerebral blood flow. Oxygen saturation of predominantly 75% venous and arterial blood is measured.[8] This provides a non-invasive continuous monitor which correlates well with cerebral metabolism. A reduction in 12% from baseline suggests a decline in cerebral metabolism.[8] A disadvantage is that there is no distinction between intracranial and extracranial blood.[8] Improved procedural outcomes are due to the advances in medical therapy, cerebral monitoring, improved timing of surgery, improved surgical techniques and the use of ultrasound guided regional techniques.[9]

In this case study we found that a combined general and local anaesthetic technique worked well minimising our use of opiates and allowing prompt emergence and neurological assessment.

Ethical approval

Approval (Approval number M210679) for this case study was granted by the Human Research Ethics Committee (Medical) of the University of Witwatersrand, Johannesburg, and other relevant authorities.

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