Giant ICA Cavernous Aneurysm with Coarctation of Aorta and Autoimmune Hemolytic Disease Posted for Flow Diverter Stenting

Yagna M. Gali[1] Moparthi R. Bhushanam[1] Shekhar R. Gurrala[1] Sanna R. Reddy[2] Vipul Pathak[2]

[1] Department of Neuroanaesthesia, Apollo Hospitals, Jubilee Hills, Hyderabad, Telangana, India
[2] Department of Neurosurgery, Apollo Hospitals, Jubilee Hills, Hyderabad, Telangana, India

Address for correspondence Yagna Munesh Gali, MD, Department of Neuroanaesthesia, Apollo Hospitals, Jubilee Hills, Hyderabad 500096, Telangana, India (e-mail: gmunesh@gmail.com).

J Neuroanaesthesiol Crit Care 2023;10:199–201.

Abstract

Anesthetic management of a case of intracranial aneurysm with coarctation of the aorta poses a formidable challenge to the anesthesiologist. The perioperative risks associated with such cases are rupture of an aneurysm, cardiac arrhythmias, stroke from cerebral insufficiency, intracerebral hemorrhage, myocardial ischemia, thromboembolic stroke, and aortic dissection. Endovascular stenting is preferred over surgical clipping of an aneurysm as flow diverter stenting has less morbidity and mortality compared to the latter, and the parent vessel can also be preserved. Despite the perioperative risks, successful stenting of an internal carotid artery aneurysm is beneficial in preventing impending aneurysm rupture and the progressive worsening of neurological symptoms. Therefore, it is imperative to understand the physiological basis behind the unforeseen complex hemodynamic fluctuations occurring during the procedure, as well as the anesthetic goals and complications encountered during the procedure. We describe and discuss the successful anesthetic management of a case of a giant internal carotid artery aneurysm with coarctation of the aorta and autoimmune hemolytic disease posted for flow diverter stenting.

Keywords
- internal carotid artery aneurysm
- coarctation of aorta
- flow diverter stenting
- case report

Introduction

Intracranial aneurysms (IA) greater than 25 mm in diameter are called giant intracranial aneurysms (GIA).[1] The prevalence of IA in patients with coarctation of the aorta (CoA) is 10%.[2] The incidence of IA rupture in CoA patients is 4.8%.[3]

This case report highlights the anticipated as well as unanticipated challenges faced during anesthetizing a CoA patient and underlying autoimmune hemolytic disease for flow diverter (FD) stenting of the GIA.

Case Report

A 60-year-old female patient presented with progressive deterioration of diplopia for 2 months in January 2023. Upon examination, she had left eye proptosis and left lateral rectus palsy. Her Glasgow coma score was 15/15, with no other discernible focal neurological deficit. Magnetic resonance imaging and cerebral angiography were suggestive of the left cavernous internal carotid artery (ICA) aneurysm. Cerebral digital subtraction angiography (DSA), when

DOI https://doi.org/10.1055/s-0043-1770780.
ISSN 2348-0548.

Thieme Medical and Scientific Publishers Pvt. Ltd., A-12, 2nd Floor, Sector 2, Noida-201301 UP, India

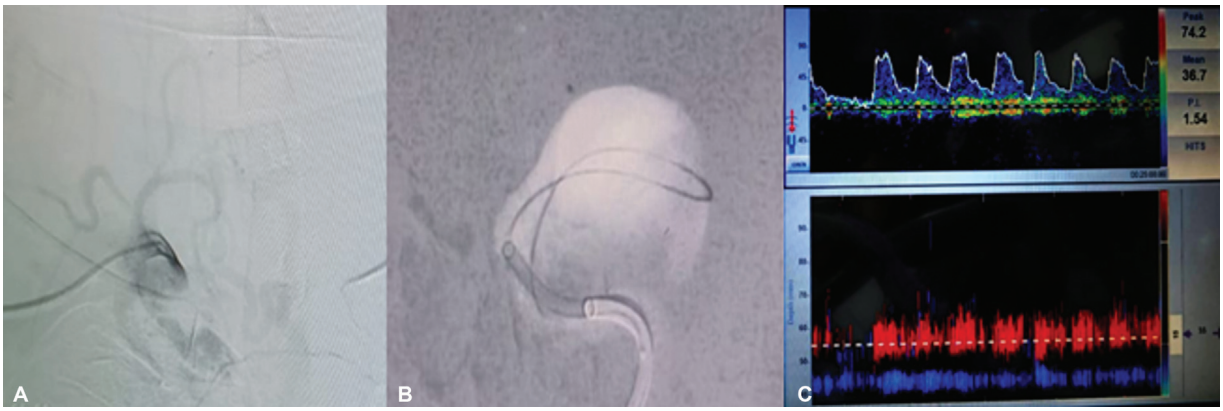

Fig. 1 (A) Digital subtraction angiography (DSA) showing coarctation of the aorta resulting in formation of collateral vessels when dye injected into arch of aorta on anteroposterior view. (B) DSA showing microcatheterization of left giant internal carotid artery aneurysm in anteroposterior view. (C) Left middle cerebral artery velocity monitoring with transcranial Doppler during arteriotomy closure.

attempted through transfemoral route, showed the existence of CoA (►**Fig. 1A**). Therefore, right radial artery access was sought to perform DSA, which confirmed the presence of a giant left paraclinoid ICA aneurysm measuring 27 × 25 mm.

During the pre-anesthetic evaluation, blood pressure (BP) recorded in the right arm and right calf was 170/90 mm Hg, and 110/70 mm Hg, respectively. The pulse rate was 82 / minute in the right radial artery, with radiofemoral delay and feeble pulses in the lower limb. Her electrocardiogram (ECG) showed sinus rhythm and echocardiography implied severe aortic stenosis, a bicuspid aortic valve, and left ventricular hypertrophy with normal ejection fraction. Her baseline hemoglobin (Hb) was 12.6 g/dL and her hematocrit was 41%. The patient was primed with Aspirin 150 mg, clopidogrel 75 mg, and cilnidipine 10 mg orally for 3 days. An indirect agglutination test done on a cross-match blood sample for the preservation of blood products turned strongly positive. On further evaluation, there was the presence of four red blood cell (RBC) antibodies, namely anti-M, Kell Ab, anti-Jk^a and anti-Fy^a. Hence, acute normovolemic hemodilution was planned. On the day of surgery, all standard monitors and defibrillator with ECG were connected. A Ryle's tube, right subclavian central venous catheter, and radial and dorsalis pedis arterial lines were placed before induction. The patient was given antibiotic prophylaxis with intravenous (IV) ceftriaxone 2 gm and premedicated with IV esomeprazole 40 mg and midazolam 2 mg. She was anesthetized using IV fentanyl, propofol, and vecuronium with the aim to blunt the intubation response. Acute normovolemic hemodilution was done, and two units (1 unit equivalent to 300–350 mL) of whole blood were removed and blood volume was restored with 500 mL of hydroxyethyl starch and 1500 mL of PlasmaLyte A. She was maintained on total IV anesthesia with standard doses of propofol and fentanyl infusion.

Owing to severe CoA and difficult radial access to maintain a stable coaxial system, the endovascular procedure was carried out by left transcarotid approach via arteriotomy. The arteriotomy was done nearly 3 cm distal to the bifurcation of the common carotid artery (CCA). After loading with 325 mg Aspirin, 30 mg prasugrel through Ryle's tube, and 5000 IU heparin IV, a guiding catheter was passed till petrous ICA. The activated clotting time was maintained above 200 seconds with intermittent doses of heparin throughout the procedure. As the microcatheter was passed across the cavernous segment of the ICA, an unusual occasional drop of around 30 mm Hg in invasive BP monitoring was observed approximately every 2 minutes from the baseline. It reverted within a few seconds, and this vicious cycle continued till the end of FD deployment (►**Fig. 1B**). During the passage of the microcatheter across the cavernous and ophthalmic segments, there was bradycardia and the appearance of multiple ventricular ectopics. The surgeon was notified and the rhythm reverted to normal once the traction of the microcatheter was relieved. To facilitate arteriotomy closure, the ICA, CCA, and external carotid artery (ECA) were occluded in this order. Cooley's clamp was used to occlude the CCA, and vascular silastic tapes were used to occlude and retract external and internal carotid arteries. The vessels were occluded proximally and distally to the arteriotomy in order to prevent leakage of blood, flowing from the aorta and the circle of Willis through the arteriotomy site. Transcranial Doppler (TCD) was used to monitor left middle cerebral artery (MCA) velocity during arteriotomy closure (►**Fig. 1C**). Postcarotid clamping, BP shot to 240/120 mm Hg and remained high. An acute rise in BP could not be controlled despite the administration of labetalol and propofol boluses. It normalized once the clamp was released. There was a blood loss of around 700 mL during the procedure. Considerable blood loss happened during arteriotomy closure due to high BP and intraoperative anticoagulation. The occlusion of vessels was relieved in the reverse order, that is, ECA, CCA followed by ICA once the arteriotomy closure was done. Sequestered blood was reinfused into the patient. She was extubated in the neurointensive care unit after her hemodynamic parameters returned to baseline an hour later. Postextubation, she was observed in the intensive care unit. There was no evidence of new focal neurological deficits for the next 48 hours. Postoperative Hb was 9.4 g/dL with a hematocrit of 28%. She had an uneventful recovery in the hospital and was discharged 6 days later.

Discussion

Hyperdynamic circulation in patients with CoA significantly increases vessel wall shear stress and poses a great threat to aneurysm rupture.[4] Aggressive antihypertensive measures to control BP may cause reduced perfusion to abdominal organs and lower limbs. Hyperstimulation of the ICA nerve plexus in the vicinity of the aneurysm during various stages of FD placement may predispose to unprecedented hemodynamic and cardiac rhythm fluctuations. There is an increased probability of the formation of microthrombi, which may disseminate, necessitating the need for high-dose antiplatelets and anticoagulants. Patients with CoA are accustomed to high cerebral perfusion pressure, and carotid clamping may prove deleterious. Release of the carotid clamp may also cause reperfusion injury. Blood transfusion in patients of pre-existing RBC alloimmunization disorder poses a serious threat to life by causing autoimmune hemolytic reactions.

Considering all the complications associated with this case, we set anesthetic goals before anesthetizing the patient. They were to prevent overt hemodynamic fluctuations, maintain mean arterial pressure over 55 mm Hg in the lower limb and under 90 mmHg in the upper limb, maintain adequate depth of anesthesia and analgesia, maintain anticoagulation, avoid blood transfusion, and target normocarbia to prevent cerebral vasoconstriction.

In our case, we postulate that constant irritation of sympathetic fibers of the ICA plexus augmented BP, which activated the baroreceptor reflex, causing occasional episodes of sudden BP drop.[5] Acute normovolemic hemodilution was done, in case of the need for a blood transfusion. The reason for RBC alloimmunization was attributed to the patient having six pregnancies in the past.[6]

In our case, the transcarotid approach was preferred over transfemoral or transradial approaches due to the presence of severe CoA and difficult radial access. It is seldom used for neuroendovascular procedures owing to the risk of carotid artery dissection, carotid artery stenosis, cervical hematoma, and peripheral intracranial embolism. Monitoring of MCA flow velocity during carotid clamping was done with TCD to watch for compromise in cerebral perfusion. However, baseline MCA velocity was not recorded as it was decided intraoperatively.

Through this case report, we highlight the significance of devising a meticulous anesthetic plan customized to the pathophysiological condition of the patient. We also emphasize on the importance of understanding the steps of the endovascular procedure and procedure-related complications for early anticipation, recognition, and timely intervention for safe and successful management.

Conclusion

Anesthetizing a patient of CoA with GIA is like walking on a tight rope. Pre-existing RBC alloimmunization makes it, even more, a formidable challenge. Understanding the preoperative status of the patient, pathophysiology, and striking the right cord in managing the hemodynamics of these patients is of prime importance.

Conflict of Interest
None declared.

References

1 Lonjon M, Pennes F, Sedat J, Bataille B. Epidemiology, genetic, natural history and clinical presentation of giant cerebral aneurysms. Neurochirurgie 2015;61(06):361–365
2 Yu X, Xia L, Jiang Q, Wei Y, Wei X, Cao S. Prevalence of intracranial aneurysm in patients with aortopathy: a systematic review with meta-analyses. J Stroke 2020;22(01):76–86
3 Singh PK, Marzo A, Staicu C, et al. The effects of aortic coarctation on cerebral hemodynamics and its importance in the etiopathogenesis of intracranial aneurysms. J Vasc Interv Neurol 2010;3(01):17–30
4 Rafieianzab D, Abazari MA, Soltani M, Alimohammadi M. The effect of coarctation degrees on wall shear stress indices. Sci Rep 2021;11(01):12757
5 Cao Q, Zhang J, Xu G. Hemodynamic changes and baroreflex sensitivity associated with carotid endarterectomy and carotid artery stenting. Intervent Neurol 2015;3(01):13–21
6 Webb J, Delaney M. Red blood cell alloimmunization in the pregnant patient. Transfus Med Rev 2018;32(04):213–219