Annals of
Medical & Surgical Case Reports

Case Report

Malik I and Agarwal A. Ann Med & Surg Case Rep: AMSCR: 100005

Anesthetic Management of a Neurosurgical Emergency in a Patient with a Recent Bentall Procedure

Indira Malik[1*] and Aakash Agarwal[2]

[1]Department of anesthesiology, PGIMS, Rohtak, Haryana, India

[2]Department of anesthesiology and critical care, BL Kapur Memorial Hospital, New Delhi, India

[*]**Corresponding author:** Indira Malik, Department of anesthesiology, PGIMS, Rohtak, Haryana 124001, India, Tel: +919350224435, Email: mdr.indira@yahoo.in

Citation: Malik I and Agarwal A (2019) Anesthetic Management of a Neurosurgical Emergency in a Patient with a Recent Bentall Procedure. Ann Med & Surg Case Rep: AMSCR: 100005

Received Date: 08 April, 2019; **Accepted Date:** 09 May, 2019; **Published Date:** 15 May, 2019

Abstract

Lifelong anticoagulation is often required in patients undergoing major cardiovascular surgery such as the Bentall procedure. Such patients are at high risk for hemorrhagic and thrombotic complications which can result in serious disability and even death. Patients presenting with intracranial hemorrhage may require urgent neurosurgical intervention, which in turn becomes a challenging scenario for the anesthesiologist. Thus, a sound knowledge of regulating perioperative anticoagulation and accepting increased risk of intraoperative and postoperative bleeding is mandatory for the anesthesiologist.

Keywords: Anticoagulants; Hemorrhage; Neurosurgical; Thrombosis

Introduction

The Bentall cardiac surgical procedure involves replacement of the aortic root with a mechanical valve conduit and re-implantation of the coronary buttons [1]. After surgery, the patients are put on life-long oral anti-coagulant (OAC) therapy because of the high risk of systemic thromboembolism. Despite stringent monitoring of OAC doses, hemorrhagic and thrombotic complications are well-known. Spontaneous intracranial hemorrhage (ICH) is one such devastating, albeit rare (incidence: 0.4-1.8%) [2], complication in cardiac surgical patients on OACs that is reported to cause serious disability or death in more than 60% ofcases.[3] Emergency neurosurgical intervention for ICH may be necessary in some patients. However, anesthetizing these patients is challenging in view of the anticipated increased surgical bleeding and the need for precisely regulating their coagulation status to prevent postoperative hemorrhage or thrombosis; prevention of intraoperative hemodynamic instability in patients with cardiac dysfunction is a further concern. We report a post-Bentall surgery patient on OACs who underwent emergency craniotomy and hematoma evacuation following spontaneous ICH and highlight the conflicts in managing perioperative coagulation in such cases.

Case Report

A 27 years old, male patient was admitted with a history of sudden-onset right hemiparesis and decreased consciousness. A non-contrast computed tomography (NCCT) scan head revealed intraparenchymal bleed, measuring 48 × 41 × 40 mm in the left frontoparietal lobe with mass effect on the ipsilateral lateral ventricle and a midline shift of 7.5 mm; urgent hematoma evacuation was considered. Preoperative anesthesia check-up revealed that the patient had undergone a Bentall procedure for a severely calcified and thickened bicuspid aortic valve (AV) 2 months back and was currently on aspirin 75 mg and warfarin 5/2.5 mg daily (alternate day) targeted to an international normalized ratio (INR) of 2.0 to 3.0. There was no history of trauma or prior bleeding episodes. He had a Glasgow Coma Scale (GCS) of E4V1M4, right-sided weakness and normal vital parameters. His INR was 2.8, and echocardiography showed normal AV prosthesis function, ejection

fraction of 40%, peak AV gradient of 27 mm Hg and mild aortic regurgitation; remaining investigations were normal. In consultation with the cardiologists, the OACs were stopped and the patient administered 4 units of fresh frozen plasma (FFP). He was taken up for emergency neurosurgery as grade 3E American Society of Anesthesiologist (ASA) grading under a high-risk consent. In the operating room, radial artery catheterization and intravenous (IV) cannulation was done under local anesthesia and routine monitoring instituted. For anesthesia, he received IV midazolam 1 mg, fentanyl (3 mcg/kg initial;1 mcg/kg hourly), thiopentone 4 mg/kg, infusions of propofol (50-100 mcg/kg/min) and vecuronium (0.1 mg/kg bolus;1 mcg/kg/min infusion), and oxygen-air mixture (50:50), and was mechanically ventilated. Central venous cannulation was avoided considering the raised INR. IV mannitol 40 gm, phenytoin 300 mg and tranexamic acid (10 mg/kg loading dose; 1 mg/kg/hour infusion) were also administered. The patient remained hemodynamically stable throughout surgery. He had a blood loss of around 400ml; one unit packed red blood cells and two units FFP were transfused. Following surgery, the patient was electively ventilated and closely monitored for his neurological, cardiovascular and coagulation status. Tracheal extubation was done on the 3rd postoperative day (POD) following a good recovery. The patient did not receive any bridging therapy with heparin as the neurosurgery team was apprehensive about rebleeding. On the 5th POD, his INR was 1.5, and in consultation with the cardiologist, OACs (aspirin 75 mg and warfarin 5/1.25 mg daily) were re-started to maintain the INR at 2.0-3.0, since the patient was having a valved conduit in- situ and was at high risk for valve thrombosis. However, on the 8th POD the patient had generalized tonic-clonic seizures; anti-seizure treatment and mechanical ventilation were initiated. NCCT head revealed an intraparenchymal bleed and surrounding edema, again in the right frontoparietal region. No further neurosurgical intervention was undertaken; the patient eventually died on the 23rd POD.

Discussion

Patients with recent cardiac operations like valve replacement, coronary artery bypass grafting and aortic root replacement are considered high-risk cases for non-cardiac surgeries [4]. These patients may have persistent cardiac dysfunction or new systemic and cardiac complications from cardiac surgery and cardiopulmonary bypass leading to perioperative cardiovascular instability and poorer outcomes in subsequent operations. Dialysis-dependent renal dysfunction, congestive cardiac failure and sudden death were also reported [5,6]. Bentall surgery patients are also known to develop serious late complications like stroke, low cardiac output syndrome, hemorrhage, multiorgan failure, myocardial infarction and arrhythmias [1]. Ascending aortic pseudoaneurysm (APA) can develop after aortic surgery; though mostly silent, APA can potentially rupture during other surgeries [7,8].

Preoperative OAC therapy in patients with mechanical valves increases their tendency to bleed more and exaggerates the risk of subsequent surgeries. Neurosurgery can be particularly risky due to the likelihood of postoperative re-bleeding with disastrous consequences. Hence, conservative management of neuro-emergencies like ICH is often preferred and surgery undertaken only in patients with hemodynamic instability or significant midline shift with impending herniation [9].

The anesthetic management of a patient requiring emergency neurosurgery after a recent heart surgery necessitates a thorough preoperative cardiac evaluation and optimum management of any cardiac dysfunction within the limited time constraints. The preoperative X ray chest should be scrutinized for APA suggested by mediastinal widening and blurred aortic junction [10]. The details of preoperative OAC therapy, history of prior bleeding episodes and derangements in coagulation profile need to be ascertained. The commonest OACs are vitamin K antagonists (VKAs); aspirin, unfractionated heparin (UFH), subcutaneous (SC) low molecular weight heparin (LMWH) and direct thrombin inhibitors (DTI) are also used. A higher risk of early postoperative bleeding with life-threatening consequences is seen with VKAs targeted to INR more than 3 in comparison to aspirin [11]. Use of UFH and LMWH is also associated with stroke [12,13]. Use of VKA with a low INR target of 1.5-2.0 along with low dose aspirin (100mg) had a lower risk of bleeding [14]. The OACs should always be discontinued preoperatively and surgery deferred till the coagulation profile is normal. However, in emergency situations, reversal of effects of oral VKAs can be done with intramuscular (IM) phytonadione 30 mg and administration of FFP [15]. Recombinant factor 7a (5-320 mcg/kg) has also been used before an urgent neurosurgery [16]. Reversal of DTI can be done with prothrombin complex concentrate (PCC), factor eight inhibitor bypassing activity (FEIBA) and hemodialysis [17]. In patients with a high risk of thromboembolic events intraoperative bridging therapy with UFH, LMWH or glycoprotein IIb/IIIa inhibitors is recommended after stopping OACs. The intraoperative management includes minimizing blood loss, adequate replacement of blood and blood products, maintenance of hemodynamic stability and prompt detection and treatment of cardiac emergencies. Close postoperative monitoring is recommended for early detection of intracranial hematoma or infarct. Mortality in these patients may be related to bleeding as a result of OAC use. In this case however, OAC was restarted in accordance with majority of the accepted international guidelines. Despite that the patient suffered rebleeding and his condition deteriorated, he had to be put back on mechanical ventilation and expired on the 23rd POD. The most vexing issue in patients with artificial cardiac valves is the decision regarding the optimal timing of restarting anticoagulants after neurosurgery. Early resumption increases the chances of cerebral re-bleeding and is not agreeable to most neurosurgeons. Conversely, a delay in anticoagulation can cause thromboembolic complications, of which, a stuck cardiac valve and stroke are the most dangerous. Opinions and practice on this issue continue to vary till date.

Majeed et al [18] reported that the optimal timing for resuming OACs was between 10-30 weeks after anticoagulation-associated ICH in a study of 177 ICH survivors. A survey of 504

physicians showed a preference for resumption between 4 and 14 days after ICH [19]. The European Stroke Initiative recommends that patients with a strong indication for anticoagulation, such as a history of embolic stroke with atrial fibrillation, should be restarted on VKA after 10 to 14 days, depending on the risk of thromboembolism and ICH recurrence [20]. The American Heart Association suggests that, in patients with a very high risk of thromboembolism VKA may be restarted 7 to 10 days after ICH onset [21]. The American College of Chest Physicians recommends starting prophylactic-dose heparin the day after ICH, with no clear guidance on restarting VKA [22]. A large, recent meta-analysis, including 8 studies of over 5000 patients of ICH, observed that re-initiation of VKA had been done within 10-40 days after surgery. They concluded that restarting of OACs led to lower risk of arterial thromboembolism without significant increase in the risk of recurrence of ICH [23]. The timing of restarting VKA depends on individual clinical condition (i.e., risks of thromboembolism and likelihood of recurrent ICH) and a brain CT scan/MRI to confirm the resolution of ICH [24]. In patients with prosthetic mechanical valves, with very high risk of thromboembolism, resumption of VKA is suggested at 2 weeks after the onset of ICH or sooner, if the hemorrhage burden is small and causative mechanism treated or stabilized.

Conclusion

To conclude, a high index of suspicion for post-cardiac surgery complications, high bleeding risks due to long-term use of OACs, especially for neurosurgical procedures, are the major considerations in such cases and a carefully tailored perioperative anaesthetic management is desirable.

References

1. Mookhoek A, Korteland NM, Arabkhani B, Centa ID, Lansac E, et al. (2016) Bentall Procedure: A Systematic Review and Meta-Analysis. Ann Thorac Surg 101: 1684-1690.
2. Askey JM (1966) Haemorrhage during long term anticoagulant drug therapy-part 1: Intracranial haemorrhage. Calif med 104: 6-10.
3. Kase CS, Robinson RK, Stein RW, Mohr JP, Hier DB, et al. (1985) Anticoagulant-related intracerebral hemorrhage. Neurology 35: 943-948.
4. Levy JH, Faha Ghadimi K, Bailey JM, Ramsay JG (2017) Postoperative cardiovascular management. In Kaplan JA editor. Kaplan's Cardiac Anesthesia, 7th ed. Philadelphia Elsevier; 2017: 1327-1357.
5. Benke K, Agg B, Szabo L, Szilveszter B, Odler B, et al. (2016) Bentall procedure: quarter century of clinical experiences of a single surgeon. J Cardiothorac Surg 11: 19-28.
6. Hanak V, Troubll M, Santavy P, Lonsky V (2016) Pseudoaneurysm of the ascending aorta following Bentall procedure. J Cardiovasc Dis Diagn 4: 5-8.
7. Malvindi PG, van Putte BP, Sonker U, Heijman RH, Schepens MA (2013) Reoperation after acute type A aortic dissection repair: a series of 104 patients. Ann Thorac Surg 95: 922-927.
8. Duraes AR, Schonhofen IS, Frietas CPA, Bitar YSL (2017) Giant pseudoaneurysm of ascending aorta. J Gen Emerg Med 2: 29-31.
9. Maeda K, Gotoh H, Chikui E, Furusawa T (2001) Intratumoral Haemorrhage from a Posterior fossa tumor after Cardiac valve surgery: case report. Neurol Med Chir 41: 548-550.
10. He S, Chen X, Zhou X, Hu Q, Ananda S (2015) Sudden death due to Traumatic Ascending Aortic Pseudoaneurysms ruptured into the esophagus. Medicine 94: 1-4.
11. Gherli T, Colli A, Fragnito C, Nicolini F, Borrello B, et al. (2004) Comparing Warfarin With Aspirin After Biological Aortic Valve Replacement. Circulation 110: 496-500.
12. Montalescot G, Polle V, Collet JP, Leprince P, Bellanger A, et al. (2000) Low Molecular Weight Heparin after Mechanical Heart Valve Replacement. Circulation 101: 1083-1086.
13. Caird J, Chukwunyerenwa C, Ali Z, Rawluk D (2006) Craniotomy with prosthetic heart valves: a clinical dilemma. British Journal of Neurosurgery 20: 40-42.
14. Teshima H, Ikebuchi M, Miyamoto Y, Tai R, Sano T, et al. (2017) 10-year results of On-X bileaflet mechanical heart valve in the aortic position: low target INR regimen in Japanese. GenThorac Cardiovasc Surg 65: 435-440.
15. Nagano N, Tabata H, Hashimoto K (1991) Anticoagulant-related Intracerebral Hemorrhage in Patients with Prosthetic Heart Valves; Report of Two Cases. Neurol Med Chir 31: 743-745.
16. Veshchev I, Elran H, Salame K (2002) Recombinant coagulation factor VIIa for rapid preoperative correction of Warfarin related coagulopathy in patients with acute subdural haematoma. Med Sci Monit 8: CS98-100.
17. Awad AJ, Walcott BP, Stapleton CJ, Yanamadala V, Nahed BV (2013) Dabigatran, intracranial hemorrhage, and the neurosurgeon. Neurosurg Focus 34: 1-6.
18. Majeed A, Kim YK, Roberts RS, Holmstrom M, Schulman S (2010) Optimal timing of resumption of warfarin after intracranial hemorrhage. Stroke 41: 2860-2866.
19. Alkherayf F, Xu Y, Gandara E, Westwick H, Moldovan ID, Wells PS (2016) Timing of vitamin K antagonist reinitiation following ICH in mechanical heart valves: systematic review and meta-analysis. Thrombosis research 144: 152-157.
20. Kaste M, Kwiecinski H, Steiner T, Mendelow D, Juvela S, et al. (2006) Recommendations for the management of intracranial hemorrhage. Cerebrovasc Dis 22: 294-316.
21. Broderick J, Connolly S, Feldmann E, Hanley D, Kase C, et al. (2007) Guidelines for the management of spontaneous ICH in adults: 2007 update. Stroke 38: 2001-2023.
22. Albers GW, Amarenco P, Easton JD, Sacco RL, Teal P (2008) Antithrombotic and Thrombolytic therapy for Ischemic stroke. CHEST 133: 630S-669S.
23. Murthy SB, Gupta A, Merkler AE, Navi BB, Mandava P, et al. (2017) Restarting anticoagulant therapy after intracranial hemorrhage. Stroke 48: 1594-1600.
24. Li YG, Lip GYH (2018) Anticoagulation resumption after Intracerebral hemorrhage. Current atherosclerosis Reports 20: 1-10.