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CLINICAL PRACTICE

Postoperative visual loss due to complicated mediastinal dissection and haemorrhagic shock treatment during cardiac surgery

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Editor's key points

- This is a report of complete blindness after repeat open heart surgery.
- During mediastinal dissection, the superior vena cava was lacerated and it had to be clamped.
- The patient also suffered prolonged hypotension due to haemorrhage.
- The blindness could have been due to a combination of raised venous pressure and hypotension.

We report the case of a patient who underwent third time revision of double heart valve replacement. Mediastinal dissection for right atrium cannulation was complicated by laceration of the superior vena cava; this required temporary rescue clamping of the vessel. The patient suffered complete visual loss related to bilateral retrobulbar haematoma. Acute elevation of superior vena cava pressure due to vascular clamping and administration of large amounts of fluid through the central venous jugular catheter could have caused the postoperative visual loss.

Keywords: complications, neurological; shock, haemorrhagic; surgery, cardiac

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Postoperative visual loss after cardiac surgery is extremely rare but carries a catastrophic functional prognosis. We report a case of bilateral visual loss subsequent to rescue clamping of the superior vena cava for vascular injury during mediastinal dissection in a patient undergoing third time revision of heart valve replacement surgery.

Case report

The patient was a woman of age 60 yr, who had undergone two previous surgeries for double mitral and aortic valve replacement using bioprothesis, 17 and 10 yr ago, respectively. Paraprothethic leaks were responsible for haemolytic anaemia, and a third operation for double valve replacement was scheduled.

Treatment with oral coumadin was switched to unfractionated heparin 48 h before surgery. The preoperative assessment showed a haemoglobin of 9.3 g dl^{-1}, a platelet count of 272 g dl^{-1}, an international normalized ratio of 1.15, heparin level of 0.33 UI $litre^{-1}$, and a fibrinogen level of 3.5 g $litre^{-1}$. I.V. anaesthesia was induced through an 18 G cannulae inserted in the right forearm. The patient was supine during surgery. The eyes were protected using 1 ml of saline instillation and by keeping them taped shut. No extrinsic compression occurred. A central venous catheter was inserted in the right internal jugular vein using ultrasound guidance. After sternotomy, dissection of the anterior mediastinum for atrium cannulation was difficult because of major adhesions related to previous surgical procedures and was complicated by an acute dilaceration of the junction of the superior vena cava with the right atrium. This resulted in massive haemorrhage and shock: systolic arterial pressure decreased to <50 mm Hg, and remained so for 30 min despite resuscitation with norepinephrine >1 $\mu g\ kg^{-1}\ min^{-1}$, crystalloids 1000 ml, and colloid solution 500 ml. The haemoglobin decreased to 3.8 g dl^{-1}, and 7 units of packed red blood cells, together with 4 units of fresh-frozen plasma and 4600 ml of autologous blood from the blood saver were given. Fluids were infused through the internal jugular catheter. The superior vena cava was clamped for 30 min duration to allow successful repair of the defect. Venous cannulation was finally performed and extracorporeal circulation started. Double valve replacement was completed without surgical difficulty.

Coagulopathy persisted at intensive care unit (ICU) admission, with a haemoglobin of 12.5 g dl^{-1} but a platelet count

of 117×10^3 μl^{-1}, fibrinogen 1.9 g litre^{-1}, and prothrombin time 59%. The initial ICU stay was complicated with a severe postoperative vasoplegic and cardiogenic shock, which required intense vasopressor and inotropic pharmacological support. Massive volume expansion with 16 litres of crystalloid was required to stabilize haemodynamics over the first 72 h. Significant bilateral and symmetrical exophthalmos became apparent on the third postoperative day (POD). Before this observation, upper thorax swelling including the head and face was seen, and was attributed to massive transfusion and large i.v. volume infusion.

After stabilization of haemodynamics, the sedation was stopped on POD 5. However, on POD 6, the patient complained of bilateral visual loss. Whereas the neurological examination revealed no abnormalities, ophthalmological examination showed symmetrical and intermediate pupils, without pupillary light reflex or blink response. Fundal appearances showed normal discs and maculae, but displayed moderate intravitreal haemorrhage in the right eye. Intraocular pressures were normal. Visual loss was complete, bilateral, without any light perception. Head computed tomography (CT) scan at POD 7 revealed the presence of bilateral intraorbitary retrobulbar haematoma, on the top of the orbits, close to the optic nerves (Fig. 1): no intracranial cause of blindness was identified. The chest CT scan showed a residual 50% stenosis of the superior vena cava with a parietal thrombus. Decompressive ocular surgery was not performed because of the time elapsed between presumed onset and diagnosis, and the need to continue effective anticoagulation. Specific treatment was limited to instillation of drops of vitamin A.

On POD 55, the patient had a persistent blindness, despite the disappearance of intravitreous haemorrhage and retrobulbar hematoma on control head CT scan. The patient was finally discharged from hospital on POD 60.

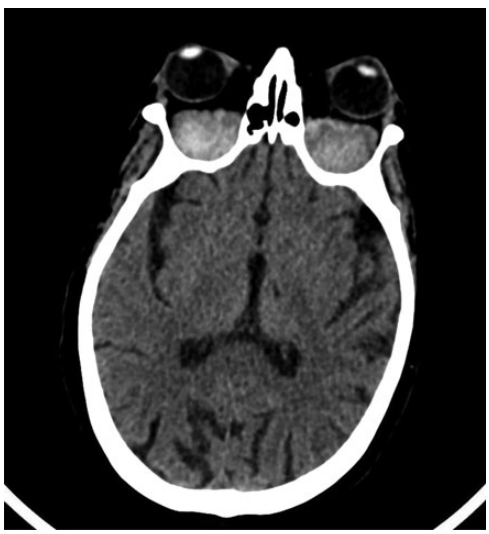

Fig 1 Head CT scan without contrast injection performed at POD 7 showing bilateral intraorbital and retrobulbar haematoma.

Discussion

Postoperative visual loss may result from three main pathophysiological mechanisms that are: ischaemic optic neuropathy, occlusion of the central retinal artery, and cortical occipital ischaemia or haemorrhage.[1–3] The fundus of the eyes and the head CT scan are the two major discriminating morphological evaluation tests. After cardiac surgery, the incidence of visual loss, dominated by ischaemic optic neuropathy, is estimated between 0.06% and 1.3% of interventions.[4–6] Thrombotic and embolic phenomena are more frequently encountered in the context of atherosclerotic infiltration.[7] Similarly, aortic manipulation may promote atherosclerotic plaques fragmentation that can occlude vessels in the vertebro-basilar territory or ophthalmic arteries. Major hypovolaemia and low flow can cause cerebral hypoperfusion, generating visual problems if it involves the occipital lobes.[4]

Bilateral and complete visual loss due to acute intraorbital retrobulbar bleeding has never been reported after non-ophthalmic and non-sinus surgery. Only one case of visual loss was described after resection of part of the superior vena cava during extraction of a mediastinal tumour, but no intraorbital haematoma occurred.[8] In our case, acute and sustained major increase in central venous pressure due to both proximal superior vena cava surgical clamping and large fluid loading in superior vena cava territory is the most likely cause for the postoperative visual loss. The iatrogenic sudden increase in central venous pressure would have been transmitted to the venous drainage of the eye through cavernous sinus central and central retinal vein, without possible blood re-routing option, since the orbit veins such as supratrochlear, nasal, facial, and ciliary veins were all exposed to the same level of elevated pressure. This acute increase in pressure within the venous drainage system of the eyes would have promoted vascular rupture and haematoma formation in the anterior and posterior segment of both eyes. Moreover, intraoperative coagulopathy induced by the haemorrhagic shock could also contribute to intraorbital bleeding, and thus enhanced acute optic nerve ischaemia. We falsely considered the immediate postoperative upper thorax and head swelling as the consequence of massive fluid administration. Moreover, this aspect hid exophthalmos which became apparent on POD 3, thus explaining delayed head CT scan. Early intraoperative reestablishment of normal superior vena cava pressure and misleading clinical presentation were certainly responsible for the delay of our investigations. The clinical features showing initially a superior vena cava syndrome aspect, then evident exophthalmos and the findings of the fundus of the eyes corroborate our diagnostic hypothesis.

More importantly, our case report questions the prevention of such complication. It raises the possibility that in the case of anticipated difficult mediastinal dissection (history of mediastinal radiotherapy, mediastinal, or vascular venous tumour surgery) or significant venous accesses should be secured before sternotomy in both the inferior and superior vena cava territories. This strategy applied to our patient might have prevented the massive increase in superior vena cava pressure,

and reduced the risk of optic nerves ischaemia while the mean pressure within the central retinal artery was certainly very low, due to haemorrhagic shock. Conversely, fluid loading and vasoactive drug infusion in inferior vena cava territory would have improved haemorrhagic shock treatment, thus improving vascular perfusion balance of the eyes.

In conclusion, the definitive bilateral visual loss in this case resulted from acute and intense increase in venous pressure within the drainage system of the eyes due to the combination of clamping superior vena cava and the treatment for haemorrhagic shock. This emphasizes the need for a preventive strategy in complex cardiac surgery, which would require securing large venous accesses, before sternotomy, in both superior and inferior vena cava territories. In addition, the rare nature of this postoperative visual loss justifies establishing registers, like the one proposed by the American Society of Anesthesiologists (ASA Postoperative Visual Loss Registry available from www.asaclosedclaims.org site) to better define the incidence and risk factors.

Authors' contributions

N.M.: clinical management of the patient, data acquisition and analysis, and wrote the manuscript; S.Z.: clinical management of the patient, data acquisition and analysis, and approved the final version of the manuscript; H.H.: clinical management of the patient, data acquisition and analysis, and approved the final version of the manuscript; W.K.: clinical management of the patient, data acquisition and analysis, and approved the final version of the manuscript; V.S.: clinical management of the patient, data acquisition and analysis, and approved the final version of the manuscript; M.D.: clinical management of the patient, data acquisition and analysis, and approved the final version of the manuscript; J.-P.C.: clinical management of the patient, data acquisition and analysis, and approved the final version of the manuscript; G.D.: clinical management of the patient, data acquisition and analysis, and wrote the manuscript.

Declaration of interest

None declared.

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