Balancing act: anaesthesia considerations for pulmonary artery mass removal with inflow occlusion in a dog

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Abstract

The use of inflow occlusion to enable excision of a cardiac valvular myxosarcoma from the anterior leaflet of the pulmonary valve in a 10-year-old female spayed heading dog. The dog had a history of exertional syncope which was increasing in frequency. Inflow venous occlusion via lateral thoracotomy approach and a cerebral protective balanced anaesthesia protocol including hypothermia were utilised. In flow occlusion causing circulatory arrest was 2.27 minutes and the mass was successfully resected. Metabolic acidosis occurred post inflow occlusion which was corrected over the following 48 hours. The dog is doing well seven months post-surgery and there is no sign of metastasis.

Introduction

Cardiac tumors in dogs are fairly rare with the most common being hemangiosarcoma's, chemodectoma, ectopic thyroid carcinoma, and lymphoma. These neoplasms are generally primary tumors in dogs whereas in humans' cardiac tumors are generally secondary via metastasis. Primary valvular tumors are rarer still, with most reports describing benign valvular myxomas. Myxosarcomas represent the malignant counterpart of myxomas, having the ability to infiltrate the myocardium and metastasise via neoplastic emboli.

Surgical excision of cardiac masses can be done under a cardiac bypass technique with subsequent hypothermia or under inflow occlusion. In this case venous inflow occlusion was chosen due to the comparative simplicity of the technique, which does not require specialised equipment and has minimal cardiopulmonary, metabolic, and hematologic effects, issues that might be encountered with bypass surgeries. A cerebral protective balanced anaesthesia protocol to minimise the metabolic derangements of inflow occlusion was chosen including preloading in anticipation the blood loss of three cardiac cycles. Total duration of inflow occlusion between 4–8 minutes has been previously reported (Hunt *et al. 1*992).

Case presentation

A 10-year-old female spayed heading dog with a history of exertional syncope increasing in frequency of occurrence, over the past two months prior to presentation. Evaluation revealed a V/VI systolic murmur and a pulmonary artery valve mass, with moderate-severe obstruction of the outflow tract and mild right atrial dilation identified with echocardiography. The dog was referred to the author's institution for computed tomography (CT) to check for other metastasis and for surgical removal. The day before surgery a CT scan was undertaken under sedation revealing an intraluminal mass lesion associated with the pulmonary valve measuring approximately 18–19mm which appeared to almost entirely occlude the right ventricular outflow tract, with secondary hypertrophy of the right ventricular myocardium. There was no evidence of right sided heart failure or metastatic disease. Blood work including biochemistry panel, complete blood count, coagulation panel and blood typing was carried out. The dog was cross matched with two potential donors in preparation should there be excessive hemorrhage.

The dog was premedicated with medetomidine and methadone both given IV into a cephalic vein catheter placed the day before for the CT, monitoring equipment of an ECG, Doppler and NIBP were placed prior to induction and the dog was preoxygenated with a fitting mask and 100% oxygen. Induction with Midazolam and Propofol slowly to effect and the dog was connected to a rebreathing circuit and mechanical ventilation started to achieve low-normocapnia. A Total intravenous anesthesia protocol (TIVA) was started consisting of Propofol, Fentanyl and Dexmedetomidine continuous rate infusions, Lidocaine CRI and loading dose were prepared should there be excessive ventricular arrythmias. An arterial catheter to monitor invasive blood pressure was placed aseptically in the right dorsal pedal artery and a triple lumen central line placed in the right jugular vein. A second venous catheter was placed in the left lateral saphenous vein. The patient was preloaded with packed red blood cells (pRBCs) slowly increasing the rate during the patient prep time to achieve a precalculated loss volume of three cardiac cycles, which the cardiologist had calculated. A lactated ringers (LRS) of Hartmans was started at a rate of 10ml/kg/hr for the first hour, to be decreased to 5ml/kg/hr thereafter to assist with preloading volume prior to the inflow occlusion. Cefazolin was given at 22mg/kg 30 minutes prior to the first incision and continued every 60 minutes intra-operatively.

Active warming was not utilised to achieve mild hypothermia. Cerebral blood flow (CBF) is usually coupled to match cerebral metabolic oxygen consumption, more than half of the oxygen delivered to the brain is used to sustain electrophysiological functions whereas the rest supports cellular homeostasis. Suppression of cerebral metabolism leads to a reduction in cerebral blood flow which can be important should inflow occlusion exceed four minutes. Likewise, under normal anesthesia conditions mild hypercapnia is often permitted however hypercapnia has been associated with an increase in CBF and increased intracranial pressure, therefore in this case for additional cerebral protection during inflow a low-normocapnia was achieved with mechanical ventilation and maintained (Greene 2010). A blood gas was run to assess arterial carbon dioxide and end tidal carbon dioxide concentrations and address any ventilation-perfusion mismatch occurrence.

The patient was moved into the operating room (OR) and positioned in a right lateral recumbency. A Neuromuscular blocking agent of Rocuronium, was administered IV once the patient was at an appropriate plane of anesthesia for surgery. An intercostal nerve block with Lidocaine was administered once surgical drapes were positioned. Surgical dissection involved a left 4th intercostal thoracotomy, where the surgeon could identify the right ventricular outflow tract and the obstructive mass, a palpable thrill was present in the main pulmonary artery. The cranial vena cava, right azygous vein and caudal vena cava were isolated with blunt dissection through the mediastinum and Rummell tourniquets were placed using 0 Silk and 12G orange PCV catheters. The Rummels were left loose. A subphrenic pericardiotomy was performed and the left phrenic nerve isolated with a stay suture. 4-0 prolene stay sutures were placed on either side of the proposed incision in the main artery. The patient was gently hyperventilated prior to inflow occlusion to decrease ETC02 levels prior to inflow occlusion.

Then all infusions and mechanical ventilation ceased simultaneously, and the clock started. Inflow occlusion was then achieved with closure of all three Rummell tourniquets and three cardiac cycles observed before incision of the main pulmonary artery with an 11 blade. The surgical team were updated with timings every 30 seconds as well as an update on systolic blood pressure value. A large 20mm friable mass was identified on the anterior pulmonary valve leaflet, grasped and resected with sharp dissection. A partial occlusion clamp positioned, manual positive pressure breath holds were administered to hyperinflate the lungs in order to vent any air from the heart and flood out the incision for a couple of beats, mechanical ventilation recommenced the TIVA was restarted including the rRBCs and LRS. The arrythmia present was atrial fibrillation, which was self-resolving, and the clock was stopped. Total inflow occlusion time was 2.27 minutes.

A blood gas was taken showing metabolic acidosis, which was treated with sodium bicarbonate, potassium chloride was also added into LRS solution for hypokalemia. Active warming commenced slowly to begin to rewarm prior to recovery. The Arteriotomy site was closed with simple continuous prolene and the partial occlusion clamp was removed. The clamp was released without inflow due to the ventral aspect of the incision being caught in the jaws, whilst this was released and re-clamped there was a decrease in blood pressure and increase in heart rate due to hemorrhage. The pRBC rate was increased as well as giving a 5ml/kg LRS bolus over five minutes. Once the clamp was replaced parameters normalised and the rest of the closure was unremarkable. A thoracic drain was placed and secured and the chest closed.

The dog was moved out of the OR into recovery. Fentanyl CRI was continued into recovery as well as beginning a Lidocaine CRI and Paracetamol IV for additional analgesia. Cefazolin was continued overnight every eight hours. Maropitant was given on recovery and the following morning ondansetron started as the dog appeared nauseous. Metabolic acidosis was a complication associated with the inflow occlusion in this case, which was treated with sodium bicarbonate. This initially worsened in the immediate post-operative period but was corrected over the following 24 hours. Clopidogrel was started orally which was continued for three months.

A recheck echocardiography at one day postoperatively revealed the resected pulmonary valve cusp, minimal main pulmonary artery stenosis from arteriotomy site, no evidence of thrombus formation over surgical site or pulmonary hypertension. There was pharmacological bradycardia resulting in generalised cardiomegaly, or possible hypervolemia. The dog was discharged home after three nights hosptialisation.

Discussion

Cardiac surgery requires thorough presurgical planning and case selection is critical when considering the use of inflow occlusion. Advanced imaging is essential to determine the exact location, resectability and best approach for the case. Anesthesia protocols tailored around cerebral protection similar to a neurological patient approach with multimodal analgesia, the use of paralytics, controlled ventilation and mild-moderate hypothermia. A whole team approach is needed to ensure simultaneous actions intraoperatively from both the anesthesia and scrubbed surgical teams when under strict time pressures before, during and after the inflow occlusion. Potential risks which could have been encountered include, a mass which is not resectable, brain hypoxemia or arrest following inflow occlusion, or a heart which did not restart following inflow occlusion, pulmonary embolism following air introduction to the heart, arrythmias including atrial or ventricular fibrillation, reactive vasoconstriction and massive intra-operative haemorrhage.

Conclusion

This case showed a successful removal of a pulmonary leaflet mass under inflow occlusion with minimal resulting complications. The anaesthesia protocol was tailored to the individual patient with a multimodal approach and thoughtful considerations around ventilation and preloading for anticipated losses. This case highlights how a whole team approach with careful case consideration can contribute to a successful outcome. The dog remains asymptomatic seven months post operatively.

References

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