

CASE REPORT

Unusual complication after cardiac surgery: iatrogenic Ortner's syndrome

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Abstract

A case of rapid unilateral vocal cord paralysis a few days after cardiac surgery is presented. On postoperative day 3, the patient developed severe hoarseness within a few hours. After evacuation of 300 ml blood from an intrathoracic haematoma, the hoarseness resolved quickly. The mechanism is supposedly direct pressure on the laryngeal nerve. In an intubated patient this complication would not have been noted.

Introduction

Annually, roughly 13,000 patients undergo cardiac surgery in the Netherlands.^[1] The perioperative complications after cardiac surgery in general are well known and consist mainly of myocardial infarction, wound infections, early or late cardiac tamponade, decline in renal function and neurological complications. To a certain extent, postoperative bleeding is common and thorax drains are left in pleural, pericardial and substernal areas to drain any fluid that might accumulate in the first postoperative day(s). We describe a case of intrathoracic haematoma that caused direct pressure on the laryngeal nerve resulting in hoarseness and severe weakening of the voice on postoperative day 3. If the patient had been mechanically ventilated at that moment, the complication would not have been noted.

Case

An 80-year-old woman was admitted to the intensive care department post cardiac surgery. Her cardiac medical history consisted of chronic atrial fibrillation and angina pectoris since 2018. During the work-up for atrial fibrillation, transthoracic echocardiography showed severe aortic stenosis, mild aortic regurgitation and severe tricuspid regurgitation with annular dilatation. In addition, coronary angiography showed significant main stem stenosis. CT scan showed extensive calcifications of the ascending aorta. These calcifications prevent safe clamping

of the ascending aorta during cardiac surgery. After extensive consultation, she underwent a hybrid cardiac procedure, consisting of off-pump coronary artery bypass grafting with the left internal mammary artery on the left anterior descending artery, direct aortic access transcatheter aortic valve replacement (TAVR) with a 26 mm Corevalve through a mini-sternotomy, and tricuspid annuloplasty with implantation of a 34-mm annuloplasty ring. According to our institutional protocol, all patients undergoing valve surgery are equipped with a pulmonary artery catheter enabling continuous cardiac output monitoring.

After the TAVR procedure, the mean gradient over the aortic valve was 10 mmHg and there was no paravalvular leakage. Total bypass time was 56 minutes, no cross clamping was performed. By protocol, 1 gram of tranexamic acid was administered. The heparin dose, which was administered by protocol before starting cardiopulmonary bypass, was completely reversed at the end of surgery. During the entire operation, diffuse microvascular bleeding was noted and one unit of packed red blood cells was administered. Four drains were left in situ, one in each pleural cavity, one pericardial drain and one substernal drain. External cardiac pacing was applied in DDD mode because of complete heart block. The postoperative left ventricular function was described as reasonable with grade II diastolic dysfunction; right ventricular function was good. The resulting tricuspid regurgitation was minimal. The patient was postoperatively admitted to our ICU, with low-dose dobutamine (2.3 µg/kg/min) and noradrenaline (0.04 µg/kg/min).

Within three hours after admission, she was extubated and within the next 12 hours the noradrenaline infusion was discontinued. X-ray of the thorax immediately after arrival to the ICU was unremarkable (*figure 1a*). The cardiac index was stable at around 1.8 l/min/m². On the morning of the first postoperative day, drain production averaged 100 ml/

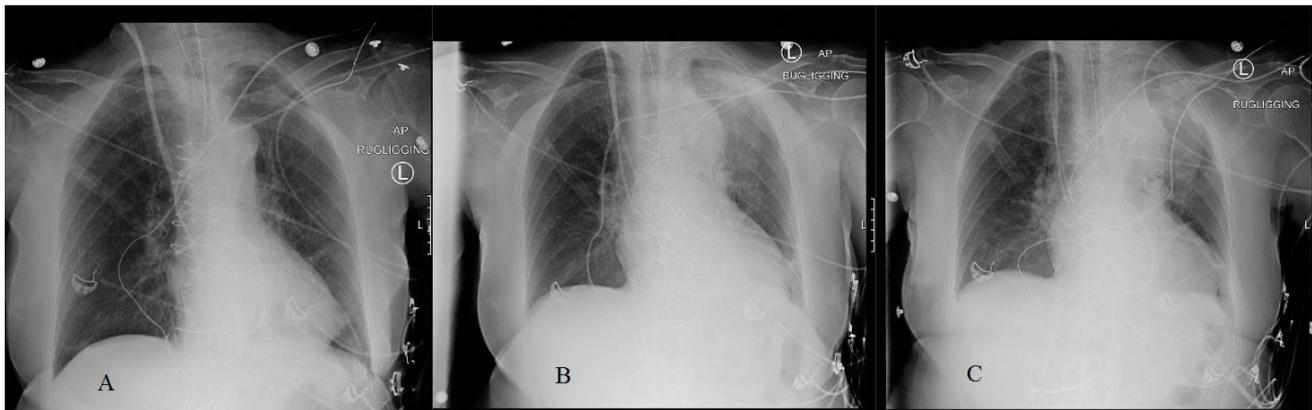


Figure 1a, b and c. A: Chest X-ray on arrival to ICU; B: Postoperative day 1; C: Postoperative day 2. A developing haematoma is visible in the left upper hemithorax

hour and with haemoglobin levels decreasing from 5.8 mmol/l to 4.9 mmol/l, one unit of packed red blood cells was given. Guided by rotational thromboelastometry, one unit of platelets and 2 grams of fibrinogen were administered, which resulted in reduced drain production (from 100 to 30 ml/hour). Anticoagulation therapy was withheld due to this continuous drain production. Chest X-ray showed a new shadowing in the left superior lobe of the lung (*figure 1b*). The cardiac surgeon was consulted for possible redo thoracotomy: in his opinion conservative treatment was indicated at this stage.

Chest X-ray on the second postoperative day showed that the shadowing in the left superior lobe was progressive (*figure 1c*). CT of the thorax without contrast administration revealed an organised haematoma along the medial and dorsal aspects of the left upper lobe and along the major fissure of the left lung. The haemoglobin levels decreased again from 5.9 to 5.3 mmol/l. Two units of packed red blood cells were given. The platelet count was $146 \times 10^9/l$ which was considered sufficient. Blood gas analyses remained unremarkable. Again, in the opinion of the cardiothoracic surgeon, conservative treatment was the best option given the fragile tissues and sternum. The thorax drains were still in situ (but not located near the haematoma) and production was negligible.

On the third postoperative day, the patient developed severe progressive hoarseness within two hours. Her voice beforehand had been unremarkable. In combination with the localisation of the haematoma we suspected progressive development of the haematoma causing pressure on the left recurrent laryngeal nerve with palsy. The consulted ENT specialist confirmed our diagnosis and urgent decompression was warranted in his opinion. The decision was made to perform video-assisted thoracoscopic surgery to evacuate the haematoma. Approximately five hours after the start of her symptoms, she underwent video-assisted thoracoscopic surgery and 350

ml of blood clots localised in the proximal part of the left internal mammary artery was evacuated. No active bleeding sites were found on inspection. A few hours after surgery, she was extubated. Immediately it was noted that her hoarseness and weak voice had disappeared completely. Anticoagulation therapy (acetylsalicylic acid and acenocoumarin) was started and she was transferred to the cardiac unit the next day. Unfortunately, a follow-up by ENT was not performed. She was discharged one week later after DDD pacemaker implantation. Her voice remained normal, without stridor or aspiration.

Discussion

Vocal cord paralysis is a well-known complication after cardiac surgery. Reports vary from 1.1% clinically apparent vocal cord paralysis in all adult cardiac surgery,^[2] to 5% clinically apparent after thoracic aortic and aortic arch surgery.^[3] When all patients were examined by ENT specialists after cardiac surgery an incidence of 10.7% was found.^[4] A review of the literature between 1980 and 2011 reported an incidence of 10.15% of vocal cord paralysis following cardiovascular surgery.^[5] The left recurrent laryngeal nerve is more often affected (70%) than the right recurrent laryngeal nerve (30%). Symptoms are hoarseness (100%) associated with stridor (49%) and aspiration (15%).^[5] The combination of left recurrent laryngeal nerve palsy with a cardiovascular disease or cardiovascular intervention surgery is called Ortner's syndrome or cardiovocal syndrome. Norbert Ortner first described hoarseness of voice due to left recurrent laryngeal nerve palsy in three patients with mitral stenosis in 1897. He postulated that an enlarged left atrium was the culprit, pressing the left recurrent laryngeal nerve against the aortic arch. In that same light, Ortner's syndrome can be the first symptom of diseases involving dilatation of the aortic arch to put traction on the left recurrent laryngeal nerve and produce hoarseness.^[6]

Nowadays, various mechanisms are known to attribute to vocal cord paralysis after cardiac surgery. First of all, median sternotomy with sternal retraction can result in longitudinal strain on both subclavian arteries and hence to both laryngeal nerves. This is enhanced by unilateral or bilateral traction on the rib cage for harvesting of the internal mammary artery.^[7] It has also been postulated that thermal injury by electrocautery during dissection of the internal mammary artery near its origin from the subclavian artery might contribute.^[2] Also the use of cardioprotective ice slush might play a role.^[2] In addition, direct trauma or neuropraxis to the left recurrent laryngeal nerve in the minimal aortic window (4 mm) where it curves around the aortic arch just above the pulmonary artery is a well-described mechanism. This can be the case in aortic aneurysms and their repair, left atrial enlargement, pathology of the ductus arteriosus or dysrhythmia ablation procedures.^[6] Also direct (iatrogenic) trauma to the larynx or the arytenoid joints or vagus nerve in the carotid sheath has been described. This involves traumatic endotracheal tube placement which can cause dislocation of the cricothyroid or cricoarytenoid joints. Repeated unsuccessful attempts to insert a transoesophageal echocardiography probe can lead to laryngeal oedema or to laryngeal recurrent nerve palsy due to haematoma of the oesophageal wall in the tracheoesophageal groove.^[7] Lastly, central venous catheterisation can cause laryngeal nerve injury, especially in the case of inadvertent arterial puncture because of close proximity of the vagal nerve in the carotid sheath. The prognosis of laryngeal recurrent nerve palsy depends on the cause. In general it is believed that the chance of recovery depends on the degree and duration of the injury. Ischaemic causes due to manipulation during cardiac surgery are not associated with a good outcome. Patients are assessed for aspiration, increased vocal effort, altered voice quality, dyspnoea on exertion and decreased quality of life. If the symptoms are well tolerated, only follow-up is warranted. Otherwise, medialisation of the affected vocal cord can be an option.^[6]

In our patient, none of the causes of left recurrent laryngeal nerve palsy as described above applied. After extubation her voice was unremarkable, excluding most direct injury mechanisms as described. A transoesophageal echocardiography probe, endotracheal tube and central line were placed uneventfully. We postulate that in the course of three postoperative days, the progression of the haematoma from the harvesting left internal mammary artery bed (*figures 1 and 2*) with open pleura caused direct pressure on the left recurrent laryngeal nerve just where the nerve loops around the arch. This caused the quickly developing hoarseness. Hence, this complication can be classified as iatrogenic Ortner's syndrome. This postulation is supported by the fact that after swift intrathoracic evacuation of the haematoma via video-assisted thoracoscopic surgery, all the symptoms disappeared and she made an unremarkable recovery. Urgent re-exploration was warranted and successful.

Disclosures

All authors declare no conflict of interest. No funding or financial support was received.

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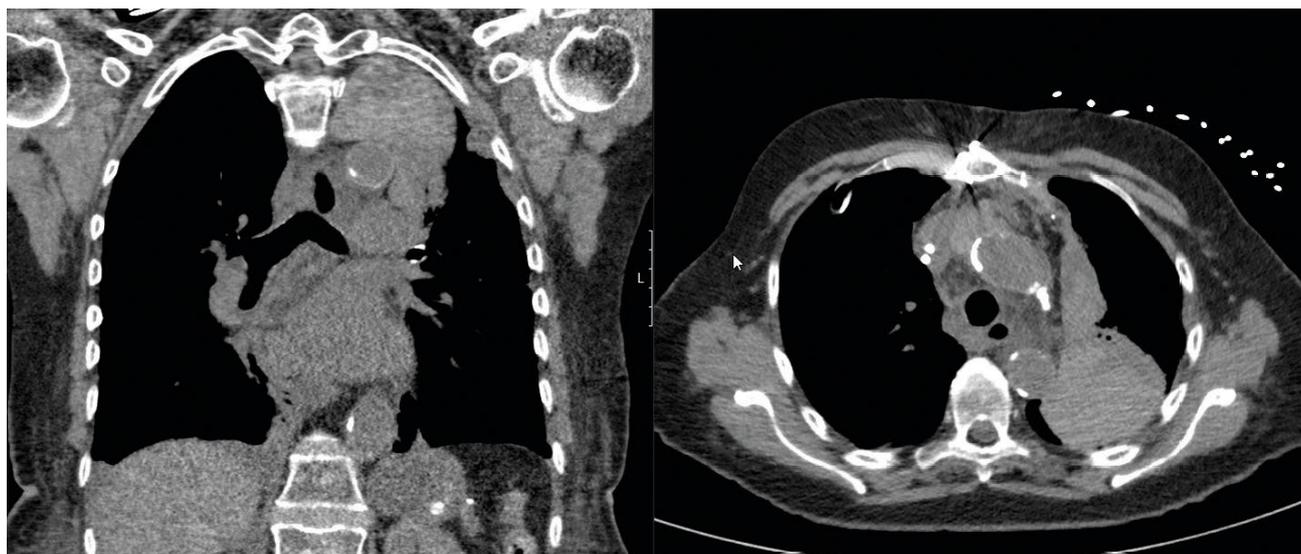


Figure 2. Coronal and axial CT showing intrathoracic haematoma pressing on aortic pulmonary window