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REVIEW

Paraganglioma review: A clinical case

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SUMMARY

Introduction: Paragangliomas are rare neuroendocrine tumours that can release catecholamines giving place to hypertensive crises and arrhythmias. Haemodynamic instability suppose a challenge for anaesthesiologists.
Clinical case: 54 years old woman scheduled for resection of a mediastinal paraganglioma located between vital structures. α-blockage was prescribed before surgery. Advanced monitoring was made during anaesthetic management. Extracorporeal circulatory support was necessary due to the haemodynamic instability and location of the tumour near vital structures.
Discussion: We present a review about general management in paragangliomas, focussing on anaesthetic monitoring. The mediastinal location of the tumour requires a special evaluation of the airway and the possibility of extracorporeal assistance.
Conclusion: Paraganglioma surgery requires and advanced monitoring and knowledge of physiopathology to reduce the mortality and morbidity.

1. Introduction

Paragangliomas are tumours arising from the neural crest located in extra-adrenal locations. They are derived from chromaffin cells, so it may be secretory tumours of catecholamines, giving rise to hypertension crisis and arrhythmias triggered by a massive release of these substances. Added to this, secondary symptoms to the location and compression of surrounding structures to the tumour occur; that it may be vitally important in the case of mediastinal locations. The frequency of these tumours is low and therefore the literature available and updated is scarce. However, advances in the preoperative management of these patients and the progress in anaesthesia have led to a reduction in the morbidity and mortality from this type of pathology.

Therefore, we present a review of the anaesthetic and perioperative management of pheochromocytomas and paragangliomas, following a report of a clinical case scheduled for surgery in our hospital.

2. Clinical case

A 54 year-old woman, weight: 66 kg, height: 170 cm, with a history of primary hyperparathyroidism, hypothyroidism and colloid breast tumour operated on in 2002. Her regular treatment was vitamin D and levothyroxine. She started having symptoms of hot flushes for 2–3 min accompanied by palpitations without objective hypertension. Given the patient's history of hyperparathyroidism, a primary study was conducted to calculate the quantity of catecholamines in her blood and urine fractionated metanephrines. There was an elevation of dopamine (407 ng/L; normal range: <85) and noradrenaline (941 ng/L; normal range: 420) in the blood, and an elevation in 24 h urine of norepinephrine (206 μg; normal range: 23–105), dopamine (838 μg; normal range: 190–450) and adrenaline (22 μg; normal range: 4–20). PTH, calcium and phosphorus levels were also high.

A cervical-thoracic-abdominal CT was performed and it identified a mediastinal mass, high uptake, of 7 × 6 cm that compresses the left main bronchus and presented a plane of cleavage between the aorta and vena cava. In the chest CT study, contrast was observed in the separation plane of the mass within the main pulmonary artery and pulmonary arteries. The mass presents extensive mediastinal, paratracheal, para-aortic, left hilar and subcarinal circulation (Fig. 1).

After the diagnosis, the patient was scheduled for surgery and hospitalized to receive alpha-blockage therapy prior to surgery. An initial dose of phenoxybenzamine 10 mg, was administered 3 times a day. Arterial pressure values remained stable between 130 – 98/65–46 mmHg with a heart rate between 69 and 102 bpm.

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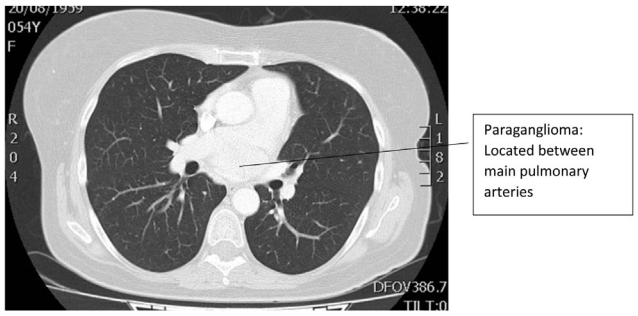

Fig. 1. Thoracic CT scan with contrast. Pulmonary window.

Full body metaiodobenzylguanidine scintigraphy was performed. There was intense fixation on the mediastinal mass that obstructs output of the left main bronchus. There were no other locations suggestive of metastasis (Fig. 2).

Fibrobronchoscopy revealed an extrinsic compression in the anterior and posterior side of the carina with revascularization in the anterior mucosa without any signs of infiltration being performed. Pulmonary function tests have a result of FVC: 115%; VEMS: 120% and the Doppler echocardiography showed normal values and limits to rule out pulmonary hypertension.

Prior to surgery, catheterization of both the subclavian arteries, showed hypertrophy of both thyrocervical trunks with vascular branches of the pathological aspect (Fig. 3). An embolization of the right thyrocervical trunk with particles of 500–1200 microns and 2 coils was performed to reduce intraoperative bleeding. During the embolization of the left thyrocervical trunk, the patient reported episodes of headache and loss of vision so the embolization was stopped.

The patient progressively recovered vision and a brain CT was performed. There was no evidence of acute or subacute brain injury.

2.1. Surgical and anaesthetic management

The night before the intervention, 10 mg of diazepam (oral) and premedication with 2 mg of midazolam (intravenous) were administered before entry to the operating-room. At anaesthetic induction, the patient was preoxygenated for 5 min with FiO_2 100%. 150 μg of fentanyl, 150 mg of propofol and 50 mg of rocuronium were administered. By direct laryngoscopy, orotracheal intubation was performed. There was no episode of haemodynamic instability during induction.

Mechanical ventilation in volume control with the following parameters: Tidal volume: 450 ml; FR: 12 rpm, PEEP: 5 cmH_20; FiO_2: 50%. Peak airway pressure: 22 cmH_2O and plateau pressure: 18 cmH_2O. The patient maintained 100% saturation after anaesthetic induction.

We proceeded to invasively monitor blood pressure by catheterization of the left radial artery and haemodynamic monitoring using the advanced ProAQT® monitor Pulsion Medical Systems (Fig. 4).

Two peripheral venous catheters 18G and a central via jugular right were used due to the surgical risk and likely intraoperative drug delivery. The initial parameters were: IC 2.7 L/min/m^2, VVS: 7%, VPP: 8%, RVS: 1100 dyn s cm5/m^2, SVI: 40 ml/m^2.

The right radial artery was also catheterized due to the possibility of doing an antegrade cerebral perfusion if the surgeon needed to clamp the supra-aortic trunk during the cardiopulmonary bypass.

During maintenance of anaesthesia, we used intravenous therapy with propofol at doses 2–3 mg/kg/h, rocuronium 0.3 mg/kg/h and remifentanil 0, 1–0, 2 μg/kg/min.

The anaesthetic depth was monitored using the bispectral index (BIS) which maintained values between 35 and 55 (Fig. 4).

At the beginning of surgery the patient remained haemodynamically stable, maintaining values of IC: 3.5–4.5 L/min/m^2, HR: 70 bpm; PA 100/60 mmHg; VVS: 10%; RVS: 1000 dyns cm^5/m^2.

To control arrhythmias and possible hypertensive crisis during surgery, drugs such as urapidil, esmolol, phentolamine and nitroglycerin were prepared.

As the surgery advanced, manipulation of cardiac structures was required. The tumour was placed around the aortic arch (shaped like an hourglass) and the pulmonary artery and dorsal trachea were compressed. Hypotension episodes in relation to the mobilization of cardiac structures were recovered with administration of phenylephrine dose 100–200 μg and momentary detention of the manipulation of the heart. Because of the haemodynamic instability it was decided to initiate extracorporeal circulatory support without cardiac arrest.

Cerebral oximetry monitoring was performed by the NIRS (Non-invasive Near Infrared Spectroscopy) SOMANETICS INVOS 5100c® monitor. During the period of haemodynamic instability, oximetry presents values between 45/50 that were recovered after reaching haemodynamic stabilization (Fig. 5).

The estimated blood losses were 1200 ml. It transfused four red blood cell concentrates, one concentrate of plasma and 1 g of

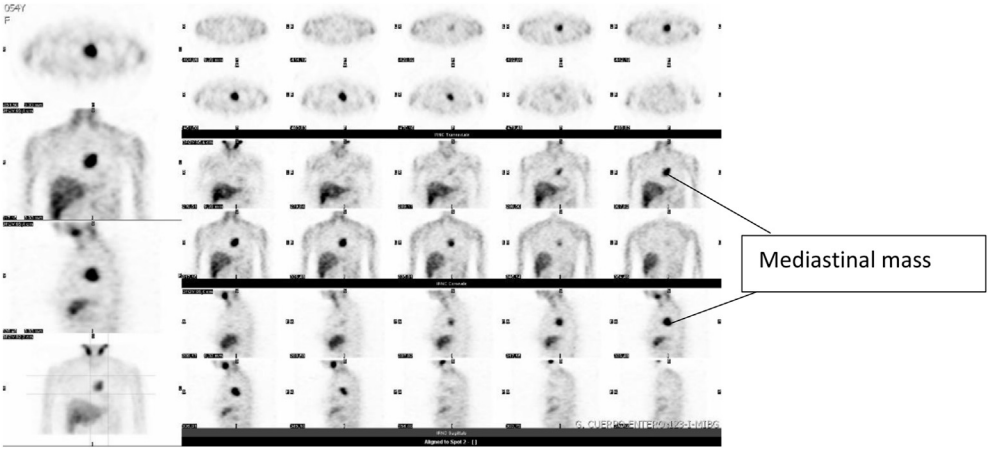

Fig. 2. Scintigraphy with 123-metaiodobenzylguanidine.