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Letter to the Editor

Embolization of thyroid arteries in a patient with compressive intrathoracic goiter ineligible to surgery or radioiodine therapy

Embolisation des artères thyroïdiennes chez un patient présentant un goitre plongeant compressif mais des contre-indications à un traitement chirurgical ou par radio-iode

1. Clinical case

A 65-year-old patient with ischemic cardiopathy underwent, in 2010, a thoracic CT scanner for routine follow-up of an aortic dilatation. The images unmasked a multinodular intrathoracic goiter, reaching both spine and aortic arch, displacing the trachea and the esophagus. The patient was referred to an ENT surgeon, who judged that surgical removal would require manubriectomy because of the voluminous intrathoracic thyroid extension.

The referring endocrinologist found subclinical hyperthyroidism with low Thyreostimulating hormone (TSH) (0.23 mU/L), and normal free thyroxin (fT4) (16 pmol/L). The hormonal dysregulation was attributed to iodine-containing contrast media since other current etiologies of hyperthyroidism were negative. The diagnosis was confirmed through iode^{123} scintigraphy that showed homogenous 3% fixation 2 hours after injection. Urinary iodine concentration was 252 μmol/24 h (N < 300). The patient spontaneously recovered from his subclinical hyperthyroidism, with two relapses when CT scanners were repeated in 2012 and 2014 to monitor the aortic dilatation (the cardiologist judged that magnetic resonance imaging could not be used as an adequate replacement).

Thyroid ultrasound showed a multinodular goiter: volume of the right lobe was 16 cm^3 with 2 nodules; volume of the left lobe was 63 cm^3, underestimated because of the intrathoracic part that could not be measured using ultrasound, despite the use of a microconvex probe; in the left lobe were a nodule and a nodular conglomerate made of isoechoic grouped nodules (measuring about 16 cm^3), scored TIRADS 3. The result of the fine needle aspiration of these nodules was benign. Radioiodine therapy was planned to reduce compression while avoiding hazardous surgery.

Unfortunately, repeated scintigraphies showed no improvement in iodine uptake which did not exceed 5.5% in 2012, while half of the volume of the gland revealed complete absence of uptake. An effective treatment would have needed a high radioiodine dose (41 mCi) with a low chance of success.

The patient became symptomatic in 2013 with grade 4 dyspnea and dysphagia. Worsening was confirmed through a new ultrasound evaluation: a 50% tracheal stenosis was diagnosed; right lobe volume was 20 cm^3; left lobe volume was at least 63 cm^3 with a nodular conglomerate of 48 cm^3. Magnetic resonance imaging (MRI) was performed: the right lobe was 26 cm^3, the extrathoracic left lobe 26 cm^3 and the intrathoracic left lobe 75 cm^3. Total goiter volume was estimated to be 127 cm^3 (normal total thyroid volume being under 20 cm^3).

Since radioiodine therapy was impossible and thyroidectomy would necessitate a thoracic surgery, embolization was proposed in order to reduce thyroid volume through superselective embolization of thyroid arteries.

Embolization was performed in 2014, using right femoral approach and 1 mL microspheres injection in both inferior thyroid arteries with no clinical side effect. In more details, selective catheterization of right and left common carotid artery was performed using a conventional 5-F catheter through a 5-F right femoral sheath after a 50 UI/kg i.v. heparin bolus was injected. Super selective catheterization of both inferior thyroidal artery using a 2.7-F microcatheter was subsequently performed through the 5-F diagnostic catheter. After angiographic control allowing to rule out potential anastomosis and confirm the thyroidal blush, free flow embolization of diluted Embospheres (Embospheres® 300–500 μm, Merit Medical, Salt Lake City, USA) was performed in order to achieve progressive total occlusion of the artery (Fig. 1). Care was taken to avoid reflux in the proximal external carotid artery in order to prevent any risk of cerebral embolization. At the end of the intervention, a closure device was used to allow early ambulation of the patient.

TSH was normal before intervention (0.48 mU/L) and very low after 2 weeks: 0.04 mU/L with a mild fT4 elevation (28 pmol/l, N < 25). After 3 weeks, TSH level remained low (0.04 mU/L) but fT4 spontaneously normalized (20 pmol/l). After 2 months, TSH was normal (0.60 mU/L) and remained normal after 6 months (0.86 mU/L) and 14 months (1.30 mU/L). The short period of hyperthyroidism remained asymptomatic and was attributed to thyroid tissue necrosis releasing thyroid hormones in the blood flow, or direct toxicity due to iodine-containing contrast media during the catheterization. Calcium level was normal before and after embolization.

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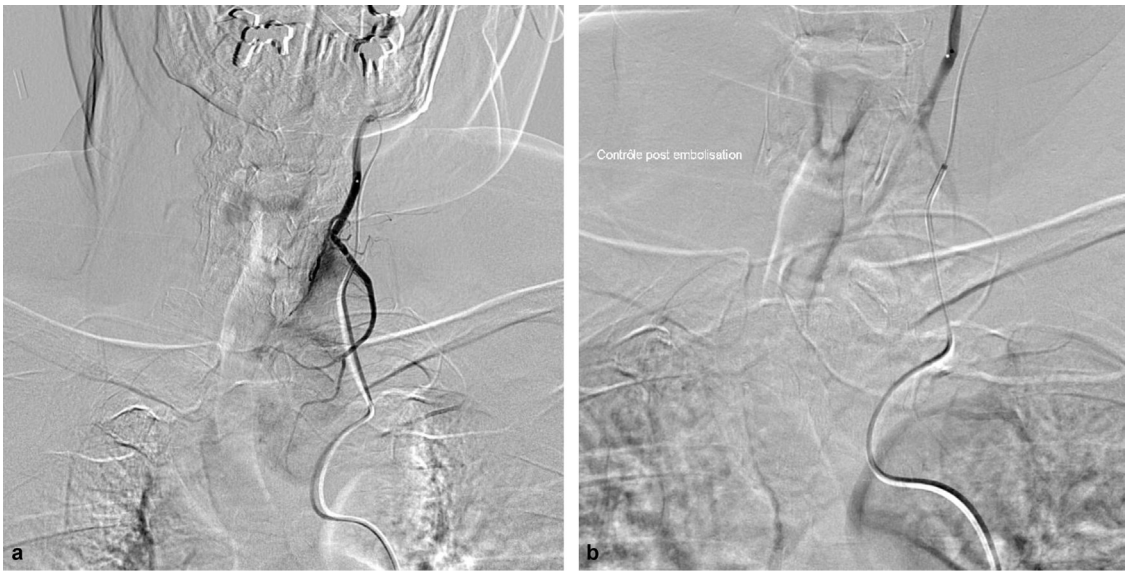

Fig. 1. a: selective angiography of the left superior thyroidal artery; b: angiographic control post-embolization with calibrated microparticules showing no residual thyroidal blush.

Anti-thyroid peroxidase, anti-thyroglobulin and anti-thyrotropin receptor antibodies remained negative. Compressive symptoms decreased.

Reduction of thyroid volume was assessed through MRI (Fig. 2) and ultrasound (Fig. 3). Three months after embolization, MRI showed a 39% diminution of thyroid volume (15 cm^3 for the right lobe, 63 cm^3 for the left lobe representing a 49 cm^3 total shrinking), stable up to 6 months. Ultrasound confirmed that the left nodular conglomerate decreased in volume from 48 cm^3 to 23 cm^3 (52% volume reduction) while the left lobe could be completely seen at the suprasternal notch, confirming the regression of the thyroid thoracic expansion. The tracheal stenosis was reduced from 50% to 25% (Table 1).

2. Discussion

Thyroid arteries embolization therapy is used in different thyroid pathologies when standard forms of treatment cannot be applied, because of side effects or contraindications: less than 200 cases are reported in the literature.

Apart from the initial Russian series published in 1994–1995 [1,2], the main series were published in Grave's disease [3,4] and other hyperthyroidism [5]. In these cases, embolization aimed to control hyperthyroidism, but a 30–50% [3] and 32% (13–76%) [5] glandular volume reduction was obtained.

Toxic goiters were also treated with selective embolization of thyroid arteries [6], but as a preliminary treatment to

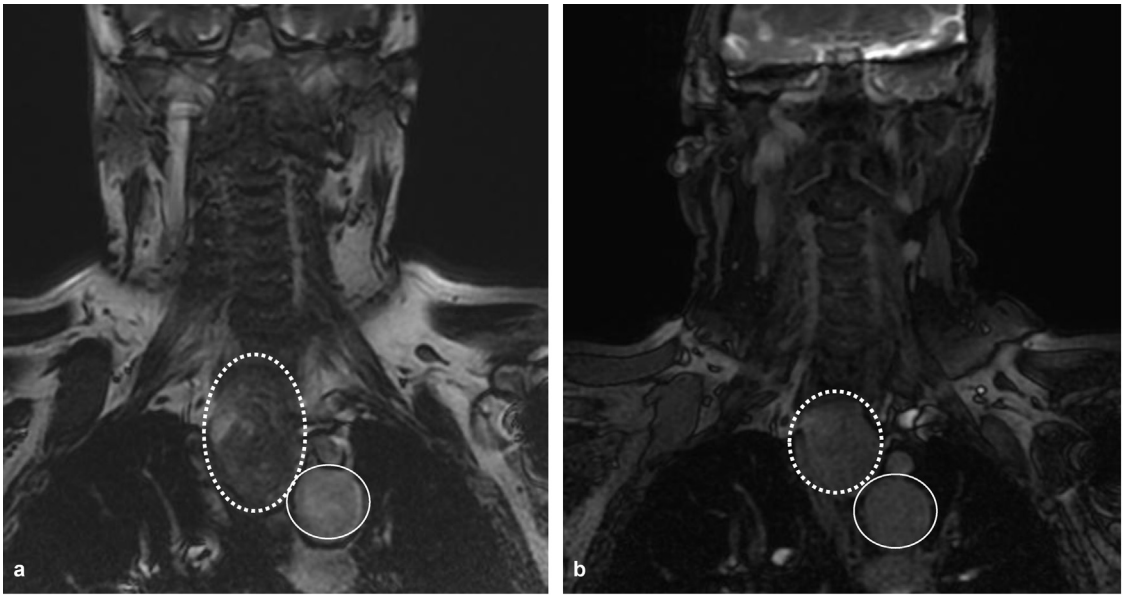

Fig. 2. a: coronal MRI pre-embolization. Large intrathoracic left thyroid lobe (dotted line) reaching the aorta (solid line); b: coronal MRI 6 months post-embolization. Marked reduction of the intrathoracic left lobe (dotted line) and of the contact with the aorta (solid line).