CLINICAL REPORT

Thyroid arterial embolization for the treatment of hyperthyroidism in a patient with thyrotoxic crisis

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Abstract

Purpose: We report a case of hyperthyroidism in a young woman caused by Graves' disease that was successfully treated with thyroid arterial embolization.

Clinical details: A 35 year-old woman with a history of thyrotoxic crises was admitted after the last thyroid crisis. Thyroid arterial embolization was used to treat the hyperthyroidism after it had been controlled. Immediately after embolization, the enlarged thyroid gland shrank and vascular murmurs disappeared. Serum thyroid hormones increased on day 3 following embolization but decreased gradually. Thyroid hormone returned to normal 2 months after embolization and remained normal at three years.

Conclusion: Thyroid arterial embolization is an effective means to treat refractory hyperthyroidism.

thyroidectomy.^{7,8} With the considerable progress in arterial embolization, a new therapy for GD hyperthyroidism has emerged.^{1-3,9} Arterial embolization is usually used to treat hyperfunction of parenchymatous organs while preserving normal organ function, similar to partial surgical resection. This approach involves embolization of most of the thyroid tissue to reduce thyroid hormone secretion, resulting in euthyroidism. After thyroid arterial embolization, vessels in the thyroid gland are occluded, the embolized tissue become ischemic, and aseptic necrosis and fibrosis will ensue, thus reducing thyroid function.^{1-3,9} In this report we describe a case of hyperthyroidism with thyrotoxic crisis treated by thyroid arterial embolization.

Although several effective therapies exist for hyperthyroidism of Graves' disease (GD) such as antithyroid drugs, radioiodine and surgery, there remain difficult cases not amenable to current treatment.¹⁻³ Even after long-term medication, hyperthyroidism may recur on withdrawal of antithyroid drugs. The use of radioactive iodine is associated with delayed onset of hypothyroidism.^{1, 3-6} Although surgery offers the advantage of quick control of hyperthyroidism, it may be complicated by recurrent laryngeal nerve injury or permanent hypoparathyroidism after near-total

Clinical report

A 35-year-old woman, who had had thyrotoxic crises in the past two years and had taken antithyroid drugs regularly, was admitted because of shortness of breath with concurrent loss of consciousness and urinary incontinence. Thyrotoxic crisis was diagnosed, and 30 drops sodium iodine, 500 mg hydrocortisone acetate and 4 mg propranolol were immediately infused intravenously. Methimazole therapy was 30 mg/6h, and other measures for cardiac failure were also started.

TABLE. Thyroid function before and after embolization.

	TT3(nmol/L)	TT4(nmol/L)	FT3(pmol/L)	FT4(pmol/L)	TSH(uIU/ml)
NR	1.08-3.1	77.2-154.4	2.50-9.82	10.0-25.0	0.25-4
TC	7.1	251	20.3	43	<0.01
PrE	2.29	90.52	3.19	20.22	<0.01
3d PoE	6.7	245	21.3	39	<0.01
2w PoE	4.5	209	15.7	30.6	0.08
1m PoE	0.56	36.07	2.13	17.48	2.34
2m PoE	2.1	112	7	16.8	0.31
1y PoE	2.0	106	6.8	16.2	0.5
3y PoE	1.06	66.31	3.83	15.87	3.94

TT4: total T4; TT3: total T3; FT4: free T4; FT3: free T3; TSH: thyroid-stimulating hormone; NR: normal range; TC: thyrotoxic crisis; PrE: pre-embolization; PoE: post-embolization; d: day; w: week; m: month; y: year.

On admission, temperature was 40°C, pulse rate 170 beat/min, and blood pressure 150/80mmHg. She was pale and comatose, with flushed and moist skin. Bilateral exophthalmos and a diffusely enlarged thyroid gland with an audible bruit were noted. The thyroid gland was III° enlarged, and thyroid function test revealed an aggravated hyperthyroid state in the patient (TC in the Table). Chest film and CT scan of her neck showed tracheal compression caused by thyromegaly. The narrowest part of the trachea was only 0.4 cm. (Fig.1). CT scan demonstrated the right thyroid lobe was 5mm×13 layers with the largest layer being 5.9 cm×3.8cm and the left thyroid lobe occupied 5mm×8 layers with the largest layer area being 4.8cm×1.8cm.

The patient responded well to medication. However, the thyrotoxic crisis occurred during the course of medication, the hyperthyroidism might be refractory to medication, and other approaches were considered. These included radioactive iodine, surgical ablation and thyroid arterial embolization. Thyroid arterial embolization was undergoing clinical trial in our hospital at the time. Patient consent for its use was given after discussion of the benefits and risks of treatment. The treatment plan was approved by the Institutional Review Board and the Ethics, Thyroid arterial embolization was performed after hyperthyroidism was controlled. Before embolization, a tracheal stent was placed to expand the stenosis (Fig.2). The stent was 5.0 cm in length and 2.0 cm in width,

with the upper end placed about 1.0 cm above the upper edge of the stricture, and the lower end 3.5 cm above the carina. One week later, thyroid arterial embolization was performed, by an experienced interventional radiologist under local anesthesia, using a SIEMENS BICOR PLUS/T.O.P model (1250mA). The thyroid major blood supply comes from two pairs of vessels: the left and right superior thyroid arteries that arise from the external carotid arteries, and the left and right inferior thyroid arteries originating from the subclavian arteries (Fig.3). After selective thyroid angiography, a 3F SP microcatheter (Boston Scientific, Fremont, CA, USA) was used to catheterize each of the bilateral superior and left inferior thyroid arteries (Fig.4). On catheterization of each artery, a mixture of polyvinyl alcohol, papaverine and a nonionic contrast agent (Omnipaque 300, Amershan Health, Shanghai, China) was slowly injected through the catheter under fluoroscopic control until blood flow ceased.

After embolization, vascular murmurs in the anterior neck region disappeared immediately, the enlarged thyroid gland shrank, and thyroid function gradually decreased (Table). However, at 1 month, thyroid function decreased with possible hypofunction which was considered to be caused by the antithyroid drug she was taking, the medicine was withdrawn. The tracheal stent was removed (Fig.5), and she was discharged home. The thyroid function returned to normal as shown at follow-up tests at 2 months, one

FIGURE 1. Chest film (A) and CT neck scan (B) before a stent was put within the trachea. The thyroid gland was greatly enlarged to compress the trachea with significant stenosis (arrows).

FIGURE 2. A stent was deployed within the stenotic trachea. A, B and C. A stent was deployed within the trachea at the position of the stenosis. D, E and F. After endotracheal stenting, CT scan demonstrated the stenosis had disappeared and the tracheal diameter had returned to normal.

FIGURE 3. Arteries of the thyroid gland. The superior thyroid artery originates from the external carotid artery while the inferior thyroid artery from the thyrocervical trunk of the subclavian artery. Some patients have a lowest thyroid artery.

year and two years after the embolization (Table).

Discussion

In this report, thyroid arterial embolization was used successfully to treat hyperthyroidism in a patient with thyrotoxic crisis. At day three after tembolization procedure, thyroid function temporarily increased (Table). Arterial embolization of the thyroid caused acute chemical thyroiditis and necrosis that released quantities of the existing hormone into the blood in a short time, resulting in a temporary increase of thyroid function.¹⁻³ Later, thyroid function gradually decreased to normal and remained in the normal range three years after embolization. However, shortly after the embolization procedure particularly at day three, special attention should be paid to prevention of additional thyrotoxic crisis caused by the release of the existing thyroid hormones.

Currently, thyroid arterial embolization has been applied to treat hyperthyroidism caused by GD.^{1-3,9,10} Zhao et al^{2,9} reported treatment of 41 patients with hyperthyroidism using thyroid arterial embolization

FIGURE 4. Right superior thyroid arterial embolization. A&B: Right superior thyroid artery angiography demonstrated significant staining (arrow) of the right thyroid lob. C. After embolization with polyvinyl alcohol, the gland abnormal staining disappeared.

FIGURE 5. Chest film. A. After a stent was deployed within the trachea, the stenosis disappeared. B. The stent had been withdrawn after thyroid arterial embolization, and the right inferior thyroid lobe was still enlarged to compress the trachea (arrow).

and, of 38 patients who had been followed up for 1-3 years, 27 became euthyroid (71.1%), four improved (10.5%) with reduced dosage of antithyroid drugs for maintenance and seven recurred (18.4%). Six patients who had long-term follow-up over three years were recurrent and re-embolized, and thyroid function test 6 months after embolization showed that all six patients were euthyroid. No side effects were found during follow-up. Xiao et al.¹ examined the use of thyroid arterial embolization as an option for 22 patients who suffered from GD, and 14 out of 16 patients (87.5%) who received interventional embolization alone became euthyroid and remained so for the follow-up of 27 months. Only two (12.5%) patients required a maintenance dose of antithyroid drug. Although clinical experience in using this technique is minimal, these studies demonstrate thyroid arterial embolization to be an effective, minimally invasive and safe method for patients with refractory hyperthyroidism caused by GD.

The mechanism of thyroid arterial embolization for the treatment of GD hyperthyroidism is to block most of the blood supply to the thyroid gland, thus leading to necrosis and later fibrosis of thyroid tissue which will decrease thyroid hormone secretion.^{1-3,9} The bilateral superior thyroid arteries account for over 70% of thyroid blood supply in the majority of patients,^{1-3,5} and embolization of bilateral superior thyroid arteries and one inferior artery will destroy 70-80% of the gland, achieving an effect similar to subtotal surgical thyroidectomy. Embolization of both the major parathyroid supplying arteries, bilateral inferior arteries, may cause hypo-parathyroidism.^{11,12} However, one unblocked inferior thyroid artery may be sufficient for blood supply to the glands and, in clinical practices, one inferior thyroid artery is usually left unembolized. This novel technique of thyroid arterial embolization can be applied in patients who are refractory to antithyroid drugs, who cannot tolerate or

choose not to accept current therapies (oral medication, radioactive iodine or surgery).

In conclusion, this report demonstrated that thyroid arterial embolization is effective in the treatment of refractory hyperthyroidism and is an alternative to current treatment modalities.

References

1. Xiao H., Zhuang W, Wang S, et al. Arterial embolization: a novel approach to thyroid ablative therapy for Graves' disease. *J Clin Endocrinol Metab* 2002;**87**:3583-9.
2. Zhao W, Gao BL, Jin CZ, et al. Long-Term Immunological Study in Graves' Disease Treated with Thyroid Arterial Embolization. *J Clin Immunol* 2008;**28**:456-63.
3. Zhao W, Gao, BL, Yang HY, et al. Thyroid arterial embolization to treat Graves' disease. *Acta Radiol* 200;**48**:186-92.
4. Bartalena L, Marcocci C, Bogazzi F, et al. Relation between therapy for hyperthyroidism and the course of Graves' ophthalmopathy. *N Engl J Med* 1998;**338**:73-8.
5. Cunnien AJ, Hay ID, Gorman CA, Offord KP, Scanlon PW. Radioiodine-induced hypothyroidism in Graves' disease: factors associated. *J Nucl Med* 1982;**23**:978-83.
6. Pearce EN, Braverman LE. Hyperthyroidism: advantages and disadvantages of medical therapy. *Surg Clin North Am* 2004;**84**:833-47.
7. Affleck BD, Swartz K, Brennan J. Surgical considerations and controversies in thyroid and parathyroid surgery. *Otolaryngol Clin North Am* 2003;**36**:159-87.
8. Boger MS, Perrier ND. Advantages and disadvantages of surgical therapy and optimal extent of thyroidectomy for the treatment of hyperthyroidism. *Surg Clin North Am* 2004;**84**:849-74.
9. Zhao W, Gao Bu-Lang, Yi Gen-Fa, et al. Apoptotic Study in Graves Disease Treated with Thyroid Arterial Embolization *Endocrine Journal* 2008;Nov 14. Advance publication on line, Viewed at: http://www.jstage.jst.go.jp/article/endocrj/advpub/0/0811070206/_pdf or http://www.jstage.jst.go.jp/article/endocrj/advpub/0/advpub_0811070206/_article.
10. Galkin EV, Grakov BS, Protopopov AV. First clinical experience of radio-endovascular functional thyroidectomy in the treatment of diffuse toxic goiter. *Vestn Rentgenol Radiol* 1994; 29-35.
11. Miller FR. Surgical anatomy of the thyroid and parathyroid glands. *Otolaryngol Clin North Am* 2003;**36**,1-7, vii.
12. Nobori, M, Saiki S, Tanaka N, Harihara Y, Shindo S, Fujimoto Y. Blood supply of the parathyroid gland from the superior thyroid artery. *Surgery* 1994;**115**,417-23.

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