Does hyperthyroidism cause coronary vasospasms?

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Summary

We report a 62-year-old women with atypical chest pain who had catheter-induced vasospasms during which she experienced the same type of chest pain she had before hospital admission. In addition, she was found to have hyperthyroidism and was discharged on methamizole and a beta blocker. Within a few weeks, TSH, fT3, and fT4 normalised, and she did not have recurrent chest pain. Summarising our case report and the existing literature, it appears likely that hyperthyroidism causes coronary vasospasms at least in a subset of patients and therefore, we believe that hyperthyroidism should be ruled out in patients with chest pain that is thought to be secondary to vasospasms.

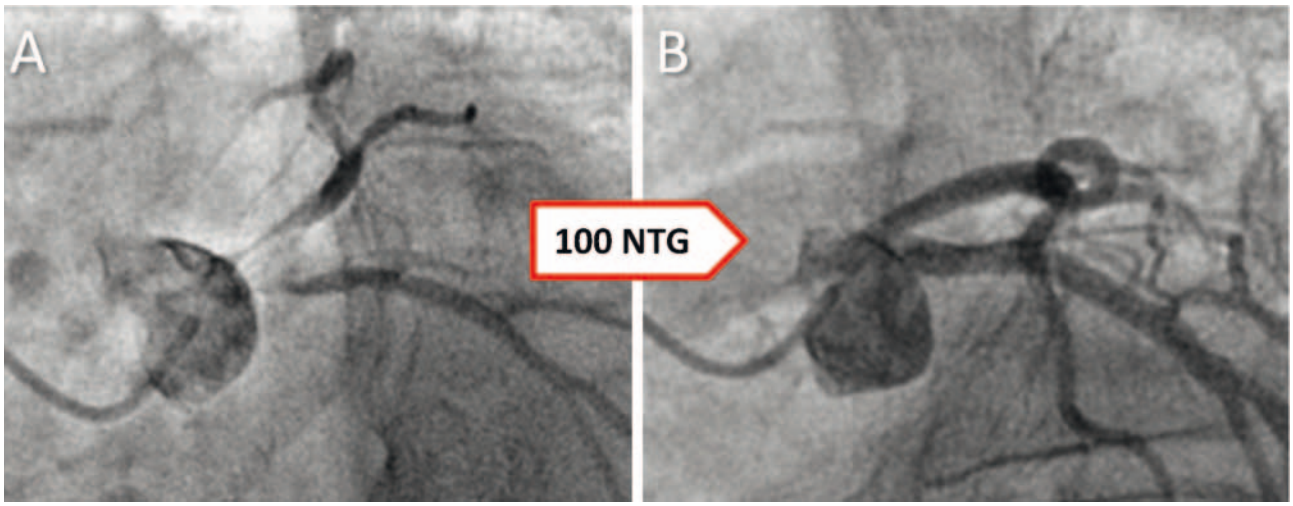
Key words: hyperthyroidism; thyrotoxicosis; vasospasm; coronary spasm

Case report

This 62-year-old previously healthy woman had a one month history of recurrent chest pain at rest. Typically, her chest pain occurred during both day and night, lasted about 30 minutes, and was not related to exercise. After a prolonged episode of chest pain, she was admitted to hospital. Her ECG was normal with a heart rate of 85/min, Troponin was not elevated and echocardiography was normal with an ejection fraction of 75%. There was no left-ventricular hypertrophy. The patient was transferred to St. Paul's Hospital for coronary angiography during which she had severe catheter-induced vasospasms of her ostial left anterior descending (LAD) and circumflex (CX) coronary artery (fig. 1A). During these spasms, she experienced the same type of chest pain she had before hospital admission. The patient also had transient ECG changes. The

Figure 1

Catheter-induced vasospasms. Catheter-induced spasms of the left anterior descending (LAD) and the circumflex (CX) coronary artery (A) which resolved after (non-selective) intracoronary injection of 100 µg nitroglycerin (NTG, B). Note that this patient had separate ostia of the LAD and CX.



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spasms resolved after administration of 100 µg of intracoronary nitroglycerin, her ECG normalised, and she became free of pain (fig. 1B). No spasm occurred during intubation of the right coronary artery ostium. After the coronary angiography, the patient was noted to have a short episode of atrial fibrillation on telemetry. Blood work revealed hyperthyroidism with a TSH <0.02 mU/l (normal values 0.27–4.20), fT4 44 pmol/l (10–22), fT3 6.9 pmol/l (3.5–6.5), white blood cell count 14.2 G/l (4.0–11.0). Other routine blood work was normal. The patient did have a palpable thyroid gland, was also experiencing heat intolerance and anxiety, diaphoresis, generalised tremors and recent onset diarrhoea. She also had a history of 10 kg weight loss but there was no exophthalmos and the patient did not have arterial hypertension. Further work-up revealed TSH receptor autoantibodies and the patient was diagnosed with coronary vasospasm and Grave's disease. She was discharged on metamizole and a beta blocker, but continued having episodes of chest pain. Within a few weeks, TSH, fT3, and fT4 normalised, and the patient did not have recurrent chest pain during a follow-up period of six months.

Does hyperthyroidism induce coronary vasospasms?

Triiodthyronine, the active form of thyroid hormone, among other effects, increases heart rate, causes atrial fibrillation (in about 5–15% of cases, more frequently in men), increases cardiac contractility, and decreases systemic vascular resistance by dilating the resistance arterioles of the peripheral circulation [1, 2]. Because increased heart rate and increased contractility lead to increased oxygen demand, hyperthyroidism can induce angina in patients with coronary artery disease. Since hyperthyroidism overall causes vasodilation which is thought to be mainly due to excessive endothelial nitric oxide (NO) production, it appears unlikely that hyperthyroidism induces coronary vasospasms [1, 3]. In fact, there is no good explanation for this phenomenon. Some studies have suggested that different arteries may react in different ways to hyperthyroidism. In particular, medium-sized muscular arteries react with potentiated vasoconstrictor response to nor-epinephrine in patients with hyperthyroidism [3, 4]. Furthermore, this exaggerated response was corrected when euthyroidism was restored by medical therapy [3]. This hypersensitive reaction offers a possible explanation why hyperthyroidism may induce coronary vasospasms in medium-sized muscular arteries such as the coronaries despite an overall vasodilative effect.

The first case with angiographic confirmation of coronary vasospasms in a patient with hyperthyroidism was reported in 1979 [5]. Since then, about 35 cases of patients with hyperthyroidism and coronary vasospasms have been published [6]. Most of these patients were women between 45 and 65 years of age, and the most frequent diagnosis was Grave's disease. Also, symptoms improved in most patients after TSH had normalised. Nevertheless, one has to keep in mind that both coronary vasospasms and hyperthyroidism are frequent diagnoses. Since only a few case reports have been published linking the two together, coincidence cannot be ruled out. The patient we described had new onset of chest pain likely due to coronary vasospasms and also a new diagnosis of hyperthyroidism. Her chest pain subsided after her TSH had normalised suggesting an association between the two diagnoses.

Summarising our case report and the existing literature, it appears likely that hyperthyroidism causes coronary vasospasms at least in a subset of patients and therefore, we believe that hyperthyroidism should be ruled out in patients with chest pain that is thought to be secondary to vasospasms.

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