Diagnosing hyperplastic oral candidiasis

Sir,
Recently, De Giorgi V et al.\(^1\) published an interesting clinical picture titled ‘Hyperplastic oral candidiasis of the tongue’. I read this article with great interest and compliment the authors for their clinical report. However, I would like to highlight several important issues.

We should analyze carefully when the authors state, ‘Clinically, it is nearly impossible to differentiate such lesions from a squamous cell carcinoma or a verrucous form of oral leukoplakia, except for the fact that they disappear after appropriate antifungal therapy’.\(^1\) Despite being an uncommon subtype (or manifestation) of oral candidiasis when compared to pseudomembranous and erythematous types, the careful clinical examination might be helpful to distinguish the lesions, avoiding surgical procedures.

First, once the tongue intermittently contacts the palate, a ‘kissing’ lesion may be seen on the palate surface opposing the tongue lesion.\(^2\) It is more often reported in erythematous candidiasis, but can be observed in hyperplastic lesions either. Thus, it is important to mention if this relation was clinically verified or if the patient wore complete dentures.

Second, the authors did not mention if there was anything relevant in the medical records of the patient. Once the oral candidiasis is an opportunistic infection, this information is relevant for the establishment of the final diagnosis.

Third, the occurrence of oral cancer in the dorsum of the tongue, especially in the median line is rare. Moreover, a one-year malignant lesion would not present such indolent behavior. Therefore, the presumptive diagnosis of hyperplastic candidiasis or another benign condition would be first hypothesized.

Fourth, despite some authors suggest that hyperplastic candidiasis is a premalignant condition,\(^3\) this issue remains controversial. If the biopsied candidiasis with epithelial dysplasia was developed due to the carcinogen potential of the fungus, or if the dysplasia was already established and the infection by the fungus came later, is an unsolved question. However, the World Health Organization does not consider the hyperplastic candidiasis a premalignant condition.\(^4\)

Finally, I believe that a careful clinical examination is essential to lead to a less invasive and effective treatment, reduction of the costs and improvement of the patient’s quality of life.

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doi:10.1093/qjmed/hcs124
Advance Access Publication 30 June 2012

Palpitations in a young patient: are we always sure about the diagnosis?

Sir,
Palpitations remain one of the commonest presentations in patients in an acute medical setup. It is an established symptom in patients who are older and have evidence of structural heart damage consequent to previous ischaemic heart disease, heart failure or defects involving the cardiac muscles or valves. It is also very commonly encountered in
people with infections, metabolic derangements or often due to idiopathic reasons when a definitive cause is not established after multiple investigations. The situation becomes slightly more tricky and challenging when patients are young and present to the emergency department with recurrent attacks of palpitations for no apparent cause. Clinicians have to keep an open mind in observing these patients and investigate them appropriately to get to the bottom of the real problem. We present an interesting case of a young patient with palpitations who posed a real diagnostic challenge.

A 25-year-old female veterinary nurse was admitted to the acute medical unit with symptoms of palpitations which were present for 2 months. Her symptoms were intermittent, short lasting, self resolving and was not related to any exertion. She also had accompanying breathlessness during these episodes and complained of mild tiredness. There was no history of weight loss, change in appetite, sweating, tremors, bowel or menstrual abnormalities, mood swings, weather preferences or pedal swelling. She was under some stress relating to her family and personal circumstances. Her past medical history included Crohn's disease and probable bulimia. She was a non-smoker and did not have any addiction to alcohol. She had a few relatives who had thyroid problems but was unsure about their biochemical status.

Enquiring further into her history, we learned that she was recently reviewed in the cardiology outpatient department following a referral from her general practitioner (GP) to whom she presented earlier with similar complaints of palpitation. She went on to have an echocardiogram, ECG and two samples (24 h collection) of urine for catecholamine’s which were all within normal limits. Interestingly, a thyroid function was not performed only showed an uptake of 0.1% and was interpreted as negative. Her symptoms were florid and she was commenced on carbimazole 30 mg once daily with a β blocker (propranolol) which she took for a few days but discontinuing later after she felt symptomatically better. She was followed up again in the next few weeks but her clinical and biochemical picture remained identical to her previous visits. The carbimazole dose was increased to 60 mg and she was called back again in 6 weeks time with repeat thyroid function test.

Her next visit did not yield any change in her symptoms and further investigations were arranged. Her TFT’s remained deranged with a TSH of <0.01 mU/l and her free hormones showed an FT4 of >77 pmol/l and an FT3 of >46 pmol/l. Her serum thyroglobulin was low normal (5 ng/ml) and a thyroid ultrasound showed normal size and echo texture of the gland. Her corrected calcium at that point was marginally better (2.6 mmol/l) but her serum parathormone was within normal limits. A technetium thyroid uptake scan was organized which only showed an uptake of 0.1% and was interpreted as negative. Her symptoms were florid at that point and a decision was made to admit her electively to hospital to improve her medically. The aim was to enable her to take her carbimazole tablets under medical supervision and observe for any differences in her clinical or biochemical profile.

Her only major clinical symptom at the point was palpitation although there were accompanying symptoms of weight loss, tiredness and mild breathlessness. Her thyroid biochemistry never settled but we felt there was a fair degree of disparity between her clinical illness and the biochemical picture. We arranged for a faecal thyroxine estimation which showed an elevated concentration of thyroxine (T4). This led us to the belief that her symptoms were made and she was commenced on a professional diagnosis of 'idiopathic sinus tachycardia' related to her stress and anxiety state but a proviso was marginally better (2.6 mmol/l) but her serum parathormone was within normal limits. A technetium thyroid uptake scan was organized which only showed an uptake of 0.1% and was interpreted as negative. Her symptoms were florid at that point and a decision was made to admit her electively to hospital to improve her medically. The aim was to enable her to take her carbimazole tablets under medical supervision and observe for any differences in her clinical or biochemical profile.

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and her thyroid biochemistry were not likely due to endogenous or intrinsic thyroid disease but due to surreptitious use of the product from outside. The points in favour of a diagnosis was that her TFT's never settled with treatment (TSH persistently <0.01 mU/l and FT4 > 77 pmol/l and FT3 > 46 pmol/l) and her serum thyroglobulin was low. Moreover, she also had a negative antibody status and her ultrasound and technetium uptake scan were all normal.

We decided to confront her with the possible diagnosis of ‘thyrotoxicosis factitia’ in our minds. She was shocked at the beginning but became tearful and submissive after a brief period into the interview. She confirmed that she was using some herbal supplements for losing weight for the past few months but did not disclose the source from where she used to receive them. She agreed not to take those supplements further and a psychiatry counseling was arranged. Her carbimazole was stopped but her β blocker was continued for symptomatic improvement. Unfortunately, she got preoccupied with the thoughts that her surreptitious use of herbal medicines which possibly contained thyroxine extracts was discovered and chose to self discharge from the hospital. She was given a few follow-up appointments in the endocrine clinic which she never attended.

This case strongly proves the strong and strange obsession of the young generation to lose weight and the extreme measures adopted to pursue this unrealistic goal. Our patient was a veterinary nurse, had a history of bulimia and had relatives who were possibly on thyroxine. She was well placed to have an access to either a veterinary thyroxine preparation or from her own relatives. We could not confirm or validate her intake of herbal medicines for losing weight as she did not disclose the name of the person who prescribed or dispensed it to her.

‘Factitious thyrotoxicosis’ is produced due to ingestion of excessive thyroid hormones from an exogenous source. This condition is commonly noted in people who have relatives who suffer from thyroid conditions and have access to thyroxine preparations due to their profession. Diagnosing this condition can be difficult and requires a high index of suspicion without which misdiagnoses and mismanagement are common.

This condition is not commonly encountered but treatment for weight loss, depression, infertility, menstrual abnormalities and attempted shrinkage of colloid goitre in patients with normal function are some of the reasons that are commonly attributable for the surreptitious use of thyroxine.1 Cosmetic creams containing thyroid or iodine preparations and weight reducing herbal medicines has been also considered to be common offenders.2 The diagnosis of factitious thyrotoxicosis should be suspected in any patient with apparent hyperthyroidism but lacking thyroid enlargement. Patients usually lack clinical signs of Graves’s disease or signs of acute or sub acute thyroiditis.2 It is predominantly encountered in young or middle age females with psychopathological conditions but should be also suspected in old people when thyrotoxicosis is inexplicable in origin.3

The systemic effects of prolonged exogenous thyroxine intake have been well reported. Hypercalcaemia in any form can feature due to the direct effects of thyroid hormones on the bone leading to increased osteoclastic activity.4 Reports have also confirmed occurrence of hypokalaemic periodic paralysis5 and acute myocardial infarction6 in patients with factitious thyrotoxicosis. Prolonged intake of thyroxine can also precipitate a thyroid storm which is often technically difficult to manage.7

Diagnosis is based on a clinical suspicion, a proper medication history and physical examination in different patient groups. Patients usually have thyrotoxic biochemistry and a low uptake of radioiodine. Colour flow doppler shows almost absent blood flow to the thyroid.8 Thyroid antibodies are negative and serum thyroglobulin is usually low or undetectable.9 Another important diagnostic tool is the estimation of 24 h urine iodine concentration and measurement of faecal thyroxine. Faecal T4 which are ~1 nmol/kg in normal healthy subjects and mildly increased in Graves’s disease is markedly elevated in people with thyrotoxicosis factitia.10

In general, patients often present to GP’s or to the acute medical units with non-specific and vague symptoms of palpitations and tiredness. They often tend to deny surreptitious use of thyroxine extracts from other sources and confirming a diagnosis then becomes very difficult. Physicians dealing with them should have an open mind while exploring differential diagnoses of palpitations in young people when the diagnosis is not apparent. Symptoms usually subside after discontinuation of thyroxine intake but a careful and sensitive enquiry in the early part of the disease often gives a clue to the diagnosis. Most of the patients should be referred to a psychiatrist as they have underlying psychopathological conditions that need to be explored by experts.

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doi:10.1093/qjmed/hcs116

Advance Access Publication 28 June 2012