Our audit showed poor compliance to the standards of DVT assessment and prophylaxis. We recommend that patients admitted to medical units in the future should be routinely assessed for DVT prophylaxis on admission and that this should be offered to eligible patients. This pattern is currently being practiced successfully at many hospitals in Wales. We are planning to conduct a second audit six months after the implication of the recommendations.

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Fig 1. Audit scoring proforma.

Contraindications

Active bleeding Cerebrovascular accident Clotting disorders Active gastric/duodenal ulcer Chronic liver disease Thrombocytopenia

Age >40	1
Weight >80 kg	1
Major surgery <6 months	1
Severe varicose veins/leg ulcers	1
Recent immobilisation	1

Anticipated bed rest rest >72 hours	2
Severe COPD	2
Active IBD	2
Severe infection eg pneumonia	2
Post partum <1 month	2

2
3
3
3
3

Score: If score is five or more, consider prophylactic treatment with enoxaparin 40 mg subcutaneous daily until patient fully mobile (14 days maximum). Nurse recommendation for enoxaparin: yes/no

COPD = chronic obstructive pulmonary disease; DVT = deep vein thrombosis; HRT = hormone replacement therapy; IBD = inflammatory bowel disease; OCP = oral contraceptives; PE = pulmonary embolism.

lesson of the month

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Complicated hypothyroidism?

A negative investigation does not necessarily indicate the absence of pathology. The pre-test clinical probability, and test sensitivity and specificity must be borne in mind when interpreting the result of any investigation.

Lesson

A 53-year-old man seen in the endocrine clinic with 'resistant' hypothyroidism had initially presented to his general practitioner 15 months earlier with fatigue, reduced exercise capacity, and myalgia, with a clear diagnosis of primary autoimmune (Hashimoto's) hypothyroidism: free T4 7.4 pmol/l (normal range (NR) 11–24), thyroid-stimulating hormone (TSH) 61 mIU/l (NR 0.35–4.5), thyroid peroxidase antibodies positive. Treatment with thyroxine 150 micrograms

daily was commenced. Over the following year, he remained symptomatic, with ongoing biochemical hypothyroidism despite stepwise increments in thyroxine dose. Serum TSH eventually normalised on thyroxine 400 mcg/day. Prescription records did not suggest concordance issues. He was referred for an endocrine opinion.

In addition to fatigue, he reported longstanding problems with loose bowel movements exacerbated by stress, alcohol and fatty foods. There was no history of weight loss, past medical history or past family history. He had a normal body mass index, no goitre, no outward manifestations of systemic disease, and no detectable intra-abdominal pathology. Investigations suggested small bowel malabsorption (Table 1).

Coeliac disease was suspected, but no antiendomysial antibodies (EmA) were detected. The duodenum appeared non-specifically abnormal on endoscopic inspection. Histological examination demonstrated marked villous atrophy of the duodenum with an intraepithelial lymphocytosis, consistent with a diagnosis of coeliac disease. Serological testing for tissue

	Result	Normal range
Haemoglobin	12.6 g/dL	13–18
MCV	80 f/I	80–96
Free T4	11.2 pmol/l	11–24
Free T3	3.9 pmol/l	3.9–6.8
TSH	12.9 mIU/I	0.35-4.5
Calcium (corrected)	2.16 mmol/l	2.1-2.55
Vitamin D	8.9 mcg/ml	7–40
Parathyroid hormone	12.9 pmol/l	1.6–6.9
Ferritin	15 ng/ml	30-400
B12	445 pg/ml	161–200
Folate	>20 mcg/ml	2.2-17.5

 $\ensuremath{\mathsf{MCV}}$ = mean corpuscular volume; TSH = thyroid-stimulating hormone.

transglutaminase (TTG) antibodies was positive. His gastro-intestinal symptoms abated on a gluten-free diet, and serum TSH normalised on thyroxine 200 mcg/day.

Comment

Management of primary hypothyroidism is usually straightforward: levothyroxine 125–175 mcg/day will generally bring serum TSH to the lower end of the normal range. Failure to respond to higher doses (commonly) suggests concordance issues, malabsorption of thyroxine in the small intestine, or additional pathology. Primary hypothyroidism is usually autoimmune, and thus may be associated with coeliac disease, pernicious anaemia, Addison's disease, vitiligo and type 1 diabetes mellitus. This can have implications for management, eg deterioration in glycaemic control in a patient with type 1 diabetes should prompt investigations to exclude a thyroid disorder, hypoadrenalism and coeliac disease. Thyroxine resistance in primary

hypothyroidism due to coeliac-related malabsorption may be sub-clinical. 1,2

Coeliac disease is frequently diagnosed on the combination of clinical features, and positive serological testing for EmA and TTG antibodies, without duodenal biopsy in every patient. Enzymelinked immunoassays for TTG antibodies are highly sensitive and specific, and are now becoming the first-line test for coeliac disease.³ In clinical practice, however, a negative test does not exclude a diagnosis, and the pre-test probability should be considered: few investigations have 100% specificity and sensitivity. A recent study in patients with biopsy-proven coeliac disease found 94% sensitivity and 99% specificity for TTG, and 89% sensitivity and 98% specificity for EmA testing.⁴ In the case described here, the clinical suspicion of coeliac disease was high, yet the antibody tests gave conflicting results. Because of the importance of establishing a definitive diagnosis before embarking on lifelong treatment, the patient underwent duodenal biopsy, confirming the diagnosis.

Coeliac disease should be considered in all patients requiring unusually high doses of thyroxine. Serological testing is a convenient first-line investigation to screen for this condition.

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