SIR, Dr Binymin is clearly not aware of the challenging 200-word limit placed on Clinical Vignettes. We thank him for his interest, and for giving us an opportunity to expand on the details. The patient gave a good history for secondary Raynaud’s phenomenon. We do not possess a capillaroscope in Derby, but the nail folds did not demonstrate any abnormalities using an ophthalmoscope (poor man’s capillaroscope).

Dr Binymin is correct to say that pituitary insufficiency is a biochemical diagnosis and does not rest on the pituitary anatomy. We disagree however that in this case further dynamic testing was necessary in order to make a firm diagnosis of hypopituitarism. At the time of diagnosis of her hypopituitarism she had marked hypothyramia with sodium at 122 mmol/l. Endocrine testing revealed abnormalities of all her anterior pituitary hormonal axes (normal ranges are given in brackets). She had secondary thyroid deficiency with a TSH 2.37 mu/l (0.3–5.5), Free T4 6.0 pmol/l (11.0–23.0) and Free T3 1.5 pmol/l (3.5–6.5). These results are not compatible with primary hypothyroidism as suggested by Dr Binymin. She had gonadotrophin deficiency with an LH 0.11u/l (1–17), FSH 0.71u/l (2.5–10.2) and a previous test showing a low oestradiol of 33 pmol/l (95–800). She had cortisol deficiency with a short synacthen test at 9am that showed a basal cortisol of <187 nmol/l (150–600). This is the typical response seen in hypopituitarism with a low basal and a delayed and inadequate response. ACTH was not measured because of a problem with sample collection. She had an undetectable growth hormone (<0.13 mu/l) and an undetectable IGF1 [<1 mu/l (114–492)], although dynamic testing would be needed to formally document growth hormone deficiency.

Given her clinical state and marked hypoaetraemia, it was necessary to start steroid treatment immediately after sending off these initial samples. The clear abnormalities of all the anterior pituitary hormonal axes can only be explained by a central cause and made further dynamic testing unnecessary for the diagnosis of hypopituitarism in this case. We thank Dr Binymin once again for converting our Clinical Vignette into a fuller case report.

The authors have declared no conflicts of interest.

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Maria AL-Deiri, Jonathan JB, Chris MD. An unusual association with Raynaud’s phenomenon: reply

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An unusual association with Raynaud’s phenomenon: reply

SIR, We read with interest the report by AL-Deiri et al. [1]. There is little doubt that the clinical scenario suggests an association of possible Raynaud’s phenomenon (RP) and pituitary insufficiency. It again raises the question whether the association of hypothyroidism and RP is a direct hormonal influence or an indirect immune process. We would like to comment on certain aspects in the reporting of this case.

A 36-year-old lady presenting for the first time with RP should always raise suspicion of a possible secondary cause. The diagnosis of RP is often dependent on patient account and description of the event. Patients often cannot differentiate a cold hand from biphasic colour change. A normal immune profile does not exclude any rheumatological cause for RP. An abnormal nailfold capillaroscope [2] (not done in this case), however, would have suggested an autoimmune rheumatic disorder that might be brought to light by thyroxin deficiency [3]. This is a fundamental issue in this case as we need to prove a causal relation between a reversible RP and hormone deficiency, and in the mean time demonstrate the absence of structural vascular damage related to other causes such an unrelated immune process.

The abnormal short synacthen test indicates adrenal insufficiency and is not a discriminatory test between a primary adrenal or pituitary disorder. The association of primary adrenal and primary thyroid insufficiency is well established. A long synacthen test would be mandatory to confirm a possible pituitary insufficiency.

Atrophy of the pituitary gland on the MR scan is not diagnostic of any specific disorder unless pituitary hormone deficiency is confirmed using provocation test rather than spot check of pituitary hormone levels as reported in this case. Hormonal studies should be performed in pairs of target gland and their respective stimulatory pituitary hormone for proper interpretation. It would have been more appropriate to present the ACTH and cortisol levels, TSH with free T3 and T4, LH/FSH and oestrogen levels simultaneously as well as GH with provocation tests [4].

Although the history and the clinical picture leaves little doubt about the diagnosis of the case reported, more attention should have been given to the standards for work up of RP and panhypopituitarism which would have strengthened the validity of the case.

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