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An unusual case of orthopnea

Editor – Keelan *et al*[1] describe an interesting patient with bilateral phrenic nerve palsies, which they ascribe to cervical spondylosis. The phrenic nerve arises mostly from C4 with contributions from C3 and C5. It would be quite exceptional, if not anatomically impossible, for cervical spondylosis to affect only those fascicles destined for the phrenic nerves, without any clinical evidence of a myelopathy or other radicular signs. They cite eight reported cases, seven of which had a myelopathy and one that was unilateral. A much more likely diagnosis is neuralgic amyotrophy, which may be bilateral in up to 30% of patients[2] and may follow strenuous exercise (17%[2]), as in this patient who was lifting heavy iron tables the day before the onset of symptoms. Phrenic nerve palsy, both unilateral and bilateral is well described in neuralgic amyotrophy and maybe the presenting and only feature.[3–7] ■

Conflicts of interest

The author has no conflicts of interest to declare.

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Adrenal insufficiency – recognition and management

The observation that Addison's disease may present as an unexplained reduction in insulin requirement in an individual with diabetes mellitus[1] has, as its corollary, the observation that a requirement for an unusually large dose of hydrocortisone (over and above concurrent fludrocortisone therapy) to maintain an adequate blood pressure (BP) might signify that Addison's disease coexists with hitherto unrecognised thyrotoxicosis.[2] This was the case in a 74-year-old man who had initially presented with Addisonian crisis characterised by BP 70/40 mmHg and a pulse rate 130 bpm. The diagnosis of Addison's disease was subsequently validated by a positive synacthen test. Nevertheless, despite the fact that hydrocortisone was co-prescribed with fludrocortisone, he required as much as 80 mg/day of hydrocortisone to maintain a BP 120/80 mgHg. The fact that tachycardia also persisted raised the index of suspicion for thyrotoxicosis, a diagnosis that was duly validated by free thyroxine and tri-iodothyronine levels of 45.3 nmol/L (normal 10–30) and 5.9 nmol/L (normal 0.8–3), respectively. A flat response to the thyrotropin-releasing hormone test clinched the diagnosis. Following treatment with carbimazole he became euthyroid and his pulse rate fell to 68 bpm. It also subsequently became possible to reduce the dose of hydrocortisone to a level of 30 mg/day, which maintained him in good health.[2]

A comparable scenario was documented in a 42-year-old woman in whom the initial diagnosis was Addison's disease and in whom treatment with prednisolone 5 mg twice per day resulted in a 1.5-year period of relief of symptoms.

Subsequently, however, she experienced two episodes of Addisonian crisis 4 months apart. The maintenance dose of prednisolone was then increased to 10 mg in the morning and 5 mg in the evening, and this was co-prescribed with fludrocortisone 50 µg/day. Following identification of thyrotoxicosis as the precipitating cause of adrenal crisis, she was rendered euthyroid by means of carbimazole, followed by radioiodine. After she became euthyroid, she remained symptom free and gained 8 kg in weight while taking prednisolone 5 mg/day and fludrocortisone 50 µg/day.[3]

Although the association of Addison's disease and thyrotoxicosis is rare,2–5there should be a heightened index of suspicion for coexisting thyrotoxicosis when symptoms and signs of hypoadrenalism persist despite progressively increasing doses of replacement therapy[4] or when a previously well managed patient experiences Addisonian crisis without an obvious precipitating cause. ■

Conflicts of interest

The author has no conflicts of interest to declare.

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