

FEATURE

CHRISTMAS 2011: DIAGNOSIS

Manchester United induced addisonian crisis

Akbar Choudhry and colleagues describe a diagnostic challenge

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Towards the end of 2010 a 58 year old woman reported a significant deterioration in her exercise tolerance. Previously she had enjoyed excellent health and participated in high intensity training for extended periods. Within three months even leisurely walks on flat terrain resulted in severe fatigue and intermittent chest discomfort. She was referred for cardiological assessment, which included dobutamine stress echocardiography and coronary angiography. Reversible ischaemia in the territory of the left anterior descending artery was managed medically but with poor symptomatic response. A multitude of new symptoms, including poor concentration, poor quality of sleep, visual blurring, and progressing fatigue, together with a persistent hyponatraemia (serum sodium level 120-125 mmol/L, normal range 135-145 mmol/L) led to endocrinology referral.

A diagnosis of autoimmune mediated Addison's disease was established on the basis of an absent serum cortisol response to synacthen stimulation, increased plasma adrenocorticotropic hormone level (509 ng/L, normal range 0-46 ng/L), undetectable plasma aldosterone with increased plasma renin activity (18.8 nmol/h, normal range 0.3-2.2 nmol/h), and positive antibodies to the adrenal cortex.

Fever pitch

Addison's disease is a difficult condition to diagnose; 60% of affected people are seen by two or more clinicians before the diagnosis is even considered.¹ The main presenting symptoms include non-specific fatigue, weakness, lethargy, and low mood,² symptoms that are intermittently described by the "healthy" general population and reported in many chronic conditions.

"I know this sounds ridiculous doctor, but . . ."

Once the diagnosis had been established, we could review and explain the patient's diverse symptoms. The most stunning was the symptom complex described by the patient while watching

Manchester United play. All clinicians are familiar with the refrain "I know this sounds ridiculous doctor, but . . ." and the following story emerged.

Before replacement treatment was initiated, the patient would experience episodes of anxiety, palpitations, panic, light headedness, and a sense of impending doom towards the end of high profile football matches at Old Trafford, Manchester United's home ground. This was particularly noticeable during the clashes against Manchester City and Chelsea in the early months of 2011, where the outcome of the match was in question until the last minute. On these occasions she considered leaving the stadium because she felt so unwell. Symptoms worsened as the second half progressed during the big matches but were barely noticeable when the opposition was from the lower reaches of the league. Fortunately, her usual seat was situated in the lower tier of the north stand (recently renamed the Sir Alex Ferguson stand) and thus the exertional component of her symptoms was rarely noticed at matches.

We have not been able to find any studies measuring the serum cortisol response during premier league football matches, but we postulate an increase mediated by stimulation of the hypothalamic-pituitary axis. There may even be a dose-response relation depending on the intensity of the action or significance of the encounter.³ We believe that our patient was having difficulty mounting an appropriate physiological cortisol response during the big games and therefore we present this as the first description of Manchester United induced addisonian crisis.

The initiation of replacement treatment coincided with the start of the 2011-12 football season, and our patient managed to attend all games at Old Trafford in August and September without any adverse effects. All games were won by a large margin (with an aggregate score of 14-3 in favour of the home team) so glucocorticoid function was barely tested.

However, Manchester United's form dipped over the following weeks, and the 6-1 defeat by local rivals Manchester City in

October was their heaviest on home ground for some 56 years.↓
On holiday at the time, our patient was spared this collapse. By this time we had fine tuned her replacement therapy (on the basis of a cortisol day curve): two of her daily doses are taken at 3:30 pm and 8:30 pm; mid-way through afternoon and evening matches, respectively.

We are pleased to report that she has remained symptom free during the recent contests against Sunderland, when a slender lead for the home team was defended during a tense second half, and FC Basel, who were able to turn a two goal deficit into a 3-2 lead, only for the home team to equalise in the final minute. To quote our patient, "Last season I would've been in a terrible state."

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Patient consent obtained.

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Figure



Hydrocortisone would have helped that crash. Manchester United go down 6-1 to their local rivals

[Image: Laurence Griffiths/Getty Images]