

Acute Transient Stress Induced Adrenal Hypertrophy and Adrenal Medullary Hyperactivity

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ABSTRACT

Objectives: Adrenal gland hypertrophy can be related to acute stress with abnormal adrenal function tests. It may not always need treatment.

Material and methods: An acute presentation of adrenal gland hypertrophy following an abdominal emergency, with subsequent hypoadrenalism was investigated.

Results: Adrenal medullary and cortical function fully recovered without treatment.

Conclusions: We postulate that the adrenal glands became enlarged and hypertrophied during an acute stress event, possibly caused by acute adrenal medullary hypersecretion and subsequent cortical hyposcretion. A wait and watch policy should be followed if no other clinical symptoms and signs of adrenal disease are present. CT scan remains an important diagnostic tool.

LEARNING POINTS

- Abnormal adrenocortical function tests and hypertrophy can occur in an acutely ill patient as a stress response.
- It is possible to have both hypersecretion and hyposcretion of adrenal hormones.
- A wait and watch policy should be followed if no other clinical symptoms and signs of adrenal disease are present in an acutely ill patient.

KEYWORDS

Adrenal gland hypertrophy, transient adrenal insufficiency, reversible adrenal dysfunction, transient adrenal medullary hyperactivity, stress related hypertension.

INTRODUCTION

The adrenal medulla and cortex participate in the response to acute stress^[1]. Transient adrenocortical insufficiency may occur with abdominal emergencies associated with hypotension^[2]. Adrenal medullary responses are an important component of the stress response, but transient hypertension due to stress-related increased catecholamines has not been documented. Acute stress-related adrenal gland hypertrophy has been shown in animal studies^[3]. We report a case of hypertension of acute onset due to transient acute stress-related adrenal hypertrophy and transient adrenal medullary hypersecretion followed by transient adrenal insufficiency.

CASE REPORT

A 69-year-old man was admitted with a 1-week history of generalized abdominal pain. On examination he had a distended abdomen with

absent bowel sounds. An urgent CT scan of the abdomen showed free gas within the abdomen indicating hollow visceral perforation. The patient underwent an emergency laparotomy and Hartmann's procedure for sigmoid colon perforation on the same day. He developed post-operative sepsis. On auscultation, sounds were dull in the right lower side of his chest, and a chest x-ray showed right lower lobe collapse. He was treated with antibiotics for 14 days. A repeat CT scan showed right lower lobe pneumonia and bilateral hypertrophy of the adrenal glands. An episode of hypertension was treated with labetalol. The patient was seen by the endocrinology team who reviewed his 24 h urinary metanephrine levels. He had very high 24 h urine normetadrenaline levels of 5,566 nmol/24 h, 4,337 nmol/24 h and 4,693 nmol/24 h (normal range 0–3,000) on three separate occasions. His systolic blood pressure (BP) decreased without any oral antihypertensives to about 150–160 mmHg later on in the admission. He was deemed safe for discharge, as high urinary catecholamine was thought to be due to a stress response, as was enlargement of the adrenal glands.

The patient was seen in the endocrinology outpatient clinic 6 weeks later and was found to have a BP of 120/88. He weighed about 57 kg, having lost about 15 kg in weight after surgery, but was beginning to regain his appetite. An MRI of the adrenal glands showed a reduction in their size and the lesion on the right adrenal gland appeared smaller. The patient's 24 h urinary normetadrenaline levels had returned to normal (1,342 nmol), but had a suboptimal response to the short Synacthen test (SST) (cortisol level at 0 min was 316 nmol/l and at 30 min was 380 nmol/l). Adrenocorticotropic hormone (ACTH) was 134 ng/l (normal range 0–46) suggesting primary adrenal cortical deficiency. He did not have any symptoms of hypoadrenalism.

The SST was repeated after 3 months along with MRI of the adrenal glands; it was thought that adrenal insult at the time of surgery had caused the abnormal test results which would be temporary. Nevertheless, patient was advised about adrenal underactivity and given leaflets about endogenous steroid deficiency-related sick day rules. His repeat MRI scan 3 months later showed a decrease in the size of the right adrenal gland, the previously noted high signal changes in the right adrenal nodule had almost completely disappeared and the lesion showed a rather low and homogenous signal. The left adrenal gland was normal. His repeat SST also showed improvement, with baseline cortisol of 331 nmol/l, rising to 481 nmol/l after stimulation.

DISCUSSION

The adrenal gland is an essential stress-responsive organ that is part of both the hypothalamic-pituitary-adrenal axis and the sympatho-adrenomedullary system^[4]. Corticotrophin releasing hormone levels are increased in the hypothalamus as a stress response, which causes production of ACTH from the pituitary gland, which in turn causes increased production of cortisol from the adrenal glands. Several animal studies have shown hypertrophy of specific regions of the adrenal gland after exposure to chronic stress. Toxicology studies have demonstrated that hypertrophy can arise from an acute stress response^[5].

Our patient had raised urinary normetadrenaline levels, which was probably stress related as they returned to normal after a few months, as did his blood pressure. This may have been acute stress-related adrenal medullary hypersecretion with hypertension as an initial response and subsequent transient sepsis-related adrenal cortical hyposecretion causing a suboptimal response to short Synacthen. The SST improved in a repeat test and the patient did not require steroid replacement. The raised normetadrenaline levels and elevated blood pressure settled without treatment.

This remains an unusual case since the bilateral adrenal gland enlargement was not due to haemorrhage but hypertrophy, which presumably caused the raised normetadrenaline levels initially with subsequent transient cortical insufficiency, both resolving without treatment.

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