A case of Addison’s disease. High clinical suspicion should guide diagnosis and caution should be used when reviewing initial laboratory investigations.

M Sutton-Smith, M Ravelo, D Lipscomb, N Haya

The Bending & Sheppard Centre for Diabetes and Endocrinology, East Sussex Healthcare NHS Trust Eastbourne District General Hospital, Kings Drive, Eastbourne, East Sussex, BN21 2UD

Case history

A 69-year-old lady with a history of autoimmune diabetes mellitus and primary autoimmune hypothyroidism presented to the Specialist Diabetes Clinic with a significant inexplicable variation in her capillary blood glucose. She had been undergoing investigations with the Gastroenterologists for nausea, vomiting and weight loss. Despite extensive investigations including: oesophageal-gastro-duodenoscopy, CT- thorax, abdomen, pelvis scanning and gastric emptying studies no cause for her symptoms had been found.

Her drug therapy included Ranitidine, Omeprazole, Metformin, Levothyroxine, Folic acid, Simvastatin, Lantus and Novorapid. Given her clinical presentation and autoimmune diagnoses, Metformin was stopped and she was investigated for adrenal insufficiency. Her basal serum cortisol was 410 nmol/L (normal range > 400nmol/L). Her short Synacthen test results were 421 nmol/L at 30 minutes and 433 nmol/L at 60 minutes. Basal ACTH 1100 ng/L (normal range 5-46), plasma renin 28 nmol/L/h (normal range 0.5-3.1), and plasma aldosterone 110 pmol/L (normal range 100-800). Anti-adrenal antibody titre was positive at 1:10 dilution.

Laboratory investigations leading to a diagnosis of Addison’s disease

<table>
<thead>
<tr>
<th>Hormone</th>
<th>Lab Values</th>
<th>Reference Range</th>
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<tbody>
<tr>
<td>Cortisol (nmol/L)</td>
<td>410</td>
<td>&gt; 400</td>
</tr>
<tr>
<td>Basal ACTH (ng/L)</td>
<td>1100</td>
<td>5-46</td>
</tr>
<tr>
<td>Plasma Renin (nmol/L/h)</td>
<td>28</td>
<td>0.5-3.1</td>
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<td>Plasma Aldosterone (pmol/L)</td>
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Baseline serum cortisol level was perceived to be adequate; however response to ACTH was stunted. The ACTH, aldosterone and renin levels together with positive anti-adrenal antibody titre are consistent with a diagnosis of primary autoimmune adrenal insufficiency (Addison’s disease). She was immediately started on hydrocortisone replacement with explanation about steroid sick day rules, mineralocorticoid was added and she was reviewed in clinic 3 weeks later. Her glycaemic profile improved, hypoglycaemic episodes were abolished and gastroenterological symptoms completely resolved.

Conclusions

This case highlights the importance of retaining a high degree of clinical suspicion in diagnosing adrenal insufficiency despite a “normal” basal Cortisol and how ACTH, aldosterone, renin and anti-adrenal antibody testing may aid diagnosis in such cases.

References