BEYOND ALCOHOL INTOXICATION

Persistent Severe Hypertension in the Emergency Department

Unmasking Decompensated Alcohol-Related Liver Disease and Suspected Pseudo-Cushing’s Syndrome

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BACKGROUND

Alcohol-related presentations are common in the Emergency Department (ED). Hypertension may be attributed to intoxication, withdrawal, pain or anxiety. When blood pressure remains severely elevated on repeated measurements, clinicians should reassess the patient and consider concurrent disease.

This case illustrates how reassessment of an apparently alcohol-related presentation led to recognition of decompensated alcohol-related liver disease (ArLD) and investigation of possible hypercortisolism.

CASE PRESENTATION

A 36-year-old man attended the ED with one month of progressive abdominal distension, increasing lethargy, nausea, reduced functional capacity and a non-thunderclap headache. He denied vomiting and visual disturbance. He reported substantial alcohol consumption and had known hypertension, for which he was not taking regular medication. He was initially seen by the Alcohol Liaison Team.

His blood pressure remained markedly elevated despite repeat observations: 191/138, 192/128, 173/127 and 174/135 mmHg. He was tachycardic but otherwise haemodynamically stable. Examination demonstrated abdominal distension with moderate ascites and peripheral oedema, without hepatic encephalopathy.

INVESTIGATIONS

Blood tests showed sodium 130 mmol/L, potassium 3.1 mmol/L, albumin 29 g/L, bilirubin 41 µmol/L, AST 218 IU/L, alkaline phosphatase 414–483 IU/L and GGT 1,634 U/L. CRP was 46 mg/L. ECG showed sinus tachycardia without acute ischaemic changes.

ENDOCRINE ASSESSMENT

Progressive central abdominal enlargement, abdominal striae, proximal muscle weakness and difficulty rising from a seated position raised concern for Cushing’s syndrome. A 24-hour urinary free cortisol result was elevated at 206 nmol/24 hours (reference range 0–129). However, chronic alcohol excess can cause a pseudo-Cushing’s state with overlapping clinical and biochemical findings.

Endocrinology arranged further outpatient assessment, including reassessment after sustained alcohol abstinence. The available findings did not establish endogenous Cushing’s syndrome; the endocrine diagnosis remained provisional at discharge.

LIVER DIAGNOSIS

Abdominal ultrasonography demonstrated hepatomegaly, diffuse fatty infiltration and moderate ascites, without focal liver lesions. Gastroenterology diagnosed decompensated ArLD (Child–Pugh B). Diagnostic ascitic fluid analysis excluded spontaneous bacterial peritonitis, allowing antibiotics to be discontinued.

TREATMENT AND OUTCOME

Persistent severe hypertension was treated with amlodipine and losartan, with gradual improvement to 138/86 mmHg. Hypokalaemia was corrected, and intravenous thiamine was followed by oral supplementation. Ascites was managed medically with spironolactone and furosemide; therapeutic drainage was not required.

At discharge, plans included gastroenterology and hepatology follow-up, liver multidisciplinary team discussion, upper gastrointestinal endoscopy for variceal screening, endocrine review and support for alcohol cessation.

DISCUSSION

The key diagnostic challenge was avoiding premature closure. Alcohol use could have contributed to the initial presentation, but it did not adequately account for persistent severe hypertension together with ascites, oedema, electrolyte abnormalities and abnormal liver biochemistry. Repeat observations and a full examination prompted investigation beyond the initial explanation.

The cortisol result required equally careful interpretation. Hypercortisolism was a reasonable concern given the clinical findings, but a single elevated urinary value during active chronic alcohol use cannot distinguish true Cushing’s syndrome from a pseudo-Cushing’s state. Specialist follow-up and reassessment after abstinence were therefore essential.

Collaboration among Emergency Medicine, Gastroenterology, Endocrinology and Alcohol Liaison Services allowed the liver disease to be recognised and treated while arranging a safe pathway for unresolved endocrine questions.

CONCLUSION

Persistent severe hypertension in a patient with alcohol-related concerns deserves repeated assessment and interpretation in the context of the whole presentation. In this case, diagnostic vigilance revealed decompensated ArLD and prompted appropriate investigation of possible cortisol excess.