Treatment of Glypressin Induced Hypoglycaemia with Desmopressin Acetate (DDAVP)

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Abstract
Severe hyponatraemia is an important cause of mortality and morbidity both independently and subsequent to rapid correction when osmotic demyelination may occur\(^1\). We report a case of severe iatrogenic hyponatraemia secondary to Glypressin. Treatment with intramuscular Desmopressin acetate (DDAVP) was necessary to deliver controlled normalisation of the serum sodium, which was not possible with hypertonic saline and fluid restriction alone.

Case History
A 45-year-old lady with Primary Biliary Cirrhosis was admitted with lethargy, she was anaemic and her haemoglobin 3.4g/dl. Bleeding oesophageal varices were diagnosed at endoscopy and she was commenced on intravenous esomeprazole, 6 hourly glypressin and ciprofloxacin prophylaxis. She became haemodynamically unstable and over 4 days received a total of 12.5L crystalloid fluid and 5 units of blood. Her serum sodium fell precipitously on the 5\(^{th}\) day of her admission to 120mmol/L, then 116mmol/L later that day. The following day she had a generalised seizure and was transferred to Intensive Care. Glypressin was discontinued and hypertonic saline commenced aiming to increase the serum sodium by 10mmol/L over the next 24 hours. After stopping the Glypressin, which had been administered 6 hourly for 5 days, she developed a marked diuresis with a urine output of 440mls/hr. Her serum Sodium rose dramatically from 109 to 115mmol/L within 2 hours placing her at high risk of osmotic demyelination. In order to counterbalance the sudden loss of renal V2 receptor stimulation, an agent that could be used to partially activate this system in a measured response was sought. DDAVP was administered at a low dose (1mcg) intramuscularly and fluid intake restricted to 1L/24hours. Her urine output at 1 hour fell to 220mls and after a second dose fell to 60mls/hr. Necessity for repeated dosing was based on a urine output of >120mls/hr for 2 consecutive hours at any time. A 3\(^{rd}\) dose was required at 12 hours based on these principals and her urine output remained controlled at 60mls/hr giving a total urine output of 1314mls in 24 hours. Serum Sodium levelled at 119mmol/L. No further doses of DDAVP were necessary. Serum Sodium normalised slowly over the next 3 days and no features of osmotic demyelination developed.

Discussion
Arginine vasopressin stimulates the renal V2 receptors leading to pure water retention. Physiological causes for activation include hypovolaemia and haemorrhage. Glypressin predominantly targets the splanchic circulation but has a cross-over effect on these receptors. Prolonged stimulation leads to suppression of AVP through homeostatic control mechanisms. The low AVP levels subsequent to removal of a prolonged stimulus results in aquaeuresis and uncontrolled rise in serum Sodium putting the patient at risk of osmotic demyelination. DDAVP is used to target this mechanism in Diabetes Insipidus. Here it has been used to provide mild activation before homeostatic controls could reset. Rat studies over a decade ago demonstrated that this approach could work\(^2\). However there is only one similar case reporting humans\(^3\). The case presented here demonstrated DDAVP as a safe and effective agent in the treatment of hyponatraemia secondary to over activation of the AVP system.

References