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Hypoglycaemia and atrial fibrillation

Sir,

We would like to report a further case of hypoglycaemia-induced self remitting atrial fibrillation. Although 2 similar cases have been previously reported in the *Journal*,[1] we feel that the recent reports of sudden death in young insulin dependent diabetics[2] may make it important to re-emphasize this interesting observation.

A 27 year old male with Type I diabetes of 15 years duration presented to the Accident and Emergency Department following a severe hypoglycaemic reaction. He was taking three daily injections of a Human Velosulin (6 units) and Insulatard (12 units) and claimed good glycaemic control which was supported by a serum fructosamine of 2.95 mmol/l (normal range 1.9–2.7) on admission. He had however been admitted to hospital on two previous occasions following severe nocturnal hypoglycaemia. There was no past medical or family history of cardiac arrhythmias.

At 20.00 h on the day of admission he became weak and confused. His mother recognized his symptoms and recorded a blood glucose of 1.4 mmol/l (BM 1–44 stick read by meter) before giving the patient milk. In the Accident and Emergency Department (21.00 h) he was given a further dose of oral glucose and a subsequent Reflocheck of 14 mmol/l was recorded. Although asymptomatic he was in atrial fibrillation with ventricular rate 100–150 beats/min, confirmed on electrocardiograph. Physical examination was normal and within one hour the rhythm spontaneously reverted to sinus. Subsequent cardiological assessment (auscultation, resting electrocardiogram and echocardiogram), serum potassium and thyroid stimulating hormone levels were all normal.

Hypoglycaemia is common in insulin treated diabetics[3,4] but the incidence of associated cardiac arrhythmias remains unknown. This case supports the view that asymptomatic arrhythmias can be induced by hypoglycaemia and may suggest a possible mechanism for the phenomenon of sudden death in insulin dependent diabetics.

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Inappropriate secretion of vasopressin, hypopituitarism and corticosteroid therapy

Sir,

A recent report describes five patients with hypopituitarism and severe hyponatraemia due to the syndrome of inappropriate antidiuretic hormone secretion (SIADH) that improved within a few days after the institution of hydrocortisone therapy, whereas the infusion of normotonic or hypertonic saline had been found to be less effective.[1]

We have observed the association between hyponatraemia due to SIADH and hypopituitarism in a 60 year old previously healthy women who presented with lethargy, weakness and complete absence of axillary and pubic hair. Laboratory features revealed severe hyponatraemia (111 mmol/l), low plasma/urine osmolality ratio (230/298 mosmol/kg) and an increased urinary sodium excretion (50 mmol/l). Chest X-ray, serum creatinine and arterial blood gases were normal. She was treated by restricting fluid intake and with intravenous administration of 5% saline solution that restored serum sodium concentration and plasma osmolality within the first week of admission. A clinical diagnosis of panhypopituitarism was confirmed by means of the LHRH-TRH test as well as both acute and continuous ACTH stimulation performed after a stay of 7 days, at which time hormonal replacement was instituted.

Oelkers emphasize that a response to corticosteroids should be the hallmark of SIADH secondary to hypopituitarism. Although hydrocortisone can restore sodium excretion to normal by inhibiting vasopressin directly[3] there is also evidence that it has mineralocorticoid-like properties and produces renal tubular sodium reabsorption in the absence of ADH.[4] On the other hand, we re-established normal serum sodium levels after conventional therapy without corticosteroids. Probably plasma osmolality improvement itself after saline infusion may