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CASE REPORTS

Heat oedema: a clinical study

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Summary

Transient generalized oedema was observed in a patient upon exposure to a hot climate. Oedema disappeared in 5 days. Clinical study during oedema revealed decreased urine volume, high urine osmolality, high urine sodium concentration, increased blood volume, hyponatraemia and hypo-osmolality. The total solute excretion was unchanged. The endogenous creatinine and para-aminohippurate clearances were normal. The findings were suggestive of increased antidiuretic hormone activity, and heat might be responsible.

Introduction

Well recognized clinical disorders due to heat exposure usually include prickly heat, syncope, heat cramp, heat exhaustion and heat stroke. Heat oedema is uncommon and less known. It is transient oedema developing during the early days of exposure to heat which disappears as acclimatization occurs. Although much has been studied in heat acclimatization (Bass *et al.*, 1955; Ladell, 1957; Rowell *et al.*, 1967; Senay, Mitchell and Wyndham, 1976), very little is known about heat oedema. Cutaneous vasodilatation is believed to be responsible for its development (Maxwell and Kleeman, 1972). The author now reports a clinical study with attention to renal function which was made in a patient with heat oedema in an attempt to define the mechanism of this clinical entity.

Case report

The patient was a 32-year-old army officer who became acclimatized to the cool climate after having long worked in a mountain camp up in the northern part of Thailand. He experienced several episodes of oedema of the lower extremities and the feeling of bloating with weight gain of 3–5 kg whenever he came to Bangkok in the summer where the temperature was about 15–20°C higher. Oedema usually disappeared within 3–5 days without any treatment, restriction of activity or changes in diet or fluid intake. There was no history of renal disease, heart or liver problem.

The patient was seen in the hospital clinic with a BP of 134/84 mmHg and pulse rate of 80/min. Physical examination was essentially normal except for oedema of the lower extremities. Blood chemistry revealed a blood urea nitrogen (BUN) of 2·85 mmol/l, serum creatinine 88·4 μmol/l, serum albumin 42 g/l, globulin 28 g/l, serum Na 133 mmol/l, K 4·0 mmol/l, Cl 94 mmol/l, CO_2 content 26 mmol/l and plasma osmolality (Posm) 272 mOsm/kg. Hb was 11·9 g/dl; WBC was 6550 with normal differential. Urinalysis showed a specific gravity of 1·038 with normal urinary sediment. The patient was followed-up daily in the clinic without any treatment or restriction of activity. The pattern and amount of fluid intake did not differ from usual averaging 2500 ml/24 hr. Oedema disappeared in 5 days after the gradual increase in urine flow.

Clinical study and results

The patient was studied on the second day of oedema on an out-patient basis with normal activity to simulate normal life. Blood volume was determined on the second day of oedema and on the fifth day using ^{131}I-labelled tagged albumin by a standard technique (Veall and Vetter, 1958). Daily urine collection was made for the measurement of volume, creatinine, osmolality (U_{osm}) and sodium concentration (U_{Na}). Para-aminohippurate clearance (C_{PAH}) was performed on the third and fifth days by a standard method (Smith, 1957). Serum creatinine, haematocrit, plasma osmolality, body weight and plasma protein were obtained daily. Creatinine clearance and urinary sodium excretion were calculated. Blood volume, blood chemistry and urine electrolyte were again obtained 10 days later.

The results are shown in Table 1. On the second day of oedema the urine volume was 820 ml with U_{osm} of 862 mOsm/kg and U_{Na} of 282 mmol/l. Blood volume was increased. The urine volume gradually increased with decrement of urine osmolality and U_{Na} until on the fifth day the urine volume was 3350 ml, U_{osm} was 234 and U_{Na} was 79 mmol/l. The haematocrit rose from 37 to 42%. Daily urine sodium excretion and C_{osm} were relatively unchanged. Urinary sodium excretion ranged from 206·8 to 264·7 mmol/24 hr and C_{osm} varied from 1·80 to 1·89 ml/min during the 4 days of study. Blood volume was 70·2 and 71·4 ml/kg on the fifth

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TABLE 1. Laboratory data

Day	Body weight kg	Blood volume ml/kg	Urine volume ml/24 hr	Uosm mosmol/kg	Posm mosmol/kg	Cosm* ml/min	UNa mmo/l	Na excretion mmol/24 hr	Ccr** ml/min	CPAH ml/min	Haematocrit %
2	62·2	81·7	820	862	272	1·81	282	231·2	112·4	–	37
3	60·7		1800	402	279	1·80	120	216·0	106·2	612	38
4	59·5		2200	356	287	1·89	94	206·8	110·4	–	40
5	58·1	7·02	3350	234	290	1·89	79	264·7	120·6	598	42
15	58·0	71·4	1500	517	291	1·85	150	225·0	118·7	–	42

*, osmolal clearance; **, creatinine clearance

and fifteenth days respectively. The values fell within the normal range. Creatinine and PAH clearances were within normal limits throughout the period of study.

Discussion

Normal physiological response to heat exposure includes vasodilatation, salt and water retention and expansion of plasma volume mediated through the stimulation of aldosterone and antidiuretic hormone (ADH) secretion (Bass *et al.*, 1955; Fletcher *et al.*, 1961; Hellman *et al.*, 1956). In a recent study of body fluid compartments following heat exposure, the increase in plasma volume has been ascribed to the transfer of interstitial protein and water to intravascular compartments (Senay *et al.*, 1976). There was no change in total body water, and oedema was not observed.

Although vasodilatation is believed to be responsible for the development of heat oedema (Maxwell and Kleeman, 1972), the precise mechanism of oedema is yet unclear. In this patient, transient oedema was observed upon exposure to a hot climate. Hypervolaemia was observed, being in accord with normal physiological response. This was associated with hyponatraemia, hypo-osmolality, decreased urine volume, high urine osmolality and high urine sodium concentration. There was, however, no change in total urine solute excretion as indicated by daily urine sodium excretion and osmolal clearance which remained relatively unchanged throughout the period of study (Table 1). The findings were consistent with increased tubular reabsorption of water which could be due either to increased ADH activity or increased tubular sensitivity to ADH. There is no known evidence that heat has any effect on tubular response to ADH. Although the plasma level of ADH was not determined it is likely that the patient has increased ADH activity. This increase in ADH activity could be primarily induced in response to relative hypovolaemia secondary to vasodilatation during the early stage of heat acclimatization (Bass *et al.*, 1955). However, in the presence of hypervolaemia, hyponatraemia, decreased plasma osmolality and normal renal function, this continued increased ADH activity as observed on the second day was indeed inappropriate and could not be explained by volume and osmolar factors. Some other factors could be involved. The data fit the criteria given for inappropriate secretion of ADH although the duration was short. Since temperature change is among non-osmotic factors affecting the release of ADH from hypothalamus, it is possible that prolonged acclimatization to a cool climate could suppress ADH release and sudden exposure to a hot climate might cause a rebound rise in ADH (Hellman and Weiner, 1953; Maxwell and Kleeman, 1972), and it appears that this effect overcomes the ADH-lowering effect of hypo-osmolality and hypervolaemia. The change is temporary, requiring a few days for adaption. Perhaps this mechanism may explain delayed response to water load and hyponatraemia in febrile diseases, including malaria (Miller *et al.*, 1967).

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