Hyponatraemia

Hyponatraemia may be either due to a deficiency of sodium or an excess of water. The determination of the hydration status of a patient is therefore the most important criterion in the evaluation of an hyponatraemic patient. Medical textbooks frequently cite reduced skin turgor, dry mouth and sunken eyeballs as signs of dehydration but these are all signs of severe dehydration. The use of haemodynamic variables eg pulse, arterial blood pressure and jugular (central) venous pressure give much earlier warning of fluid loss. The rate of urine flow is also a valuable sign but may be misleading in patients with renal disease or in states of osmotic diuresis.

Urine sodium is used as a critical step in this approach to hyponatraemia. However, sodium excretion as an obligate cation may occur even in hyponatraemic patients who are dehydrated with normal renal function. Examples include patients who become alkalotic with persistent vomiting and significant bicarbonaturia; or in patients with keto-acidosis who excrete large quantities of urinary keto-acids which have an effect as both osmotic diuretics and as anions requiring an obligate cation eg diabetic or alcoholic keto-acidosis or starvation.

Increased sodium excretion is only reliably seen as an acute presentation of adrenal failure. In chronic cases, the reduced filtered load of sodium may lead to a new steady state with a lower urinary sodium concentration. Moreover, patients with pituitary disease and secondary adrenal failure may not be dehydrated due to the presence of concurrent SIADH.

Increased urinary sodium may occur in patients following sub arachnoid haemorrhage or aneurysm surgery. This cerebral salt wasting may be due to excessive secretion of B type natriuretic peptide.

Acute vs chronic hyponatraemia

It is important to differentiate between acute and chronic hyponatraemia since the former carries a mortality of up to 50%. Chronic hyponatraemia carries a better outcome but symptomatic cases have a mortality of 10-15%. Acute hyponatraemia may be defined as Na < 120 mmol/L developing over less than 48 hours or a fall in Na of greater than 0.5 mmol/hr.

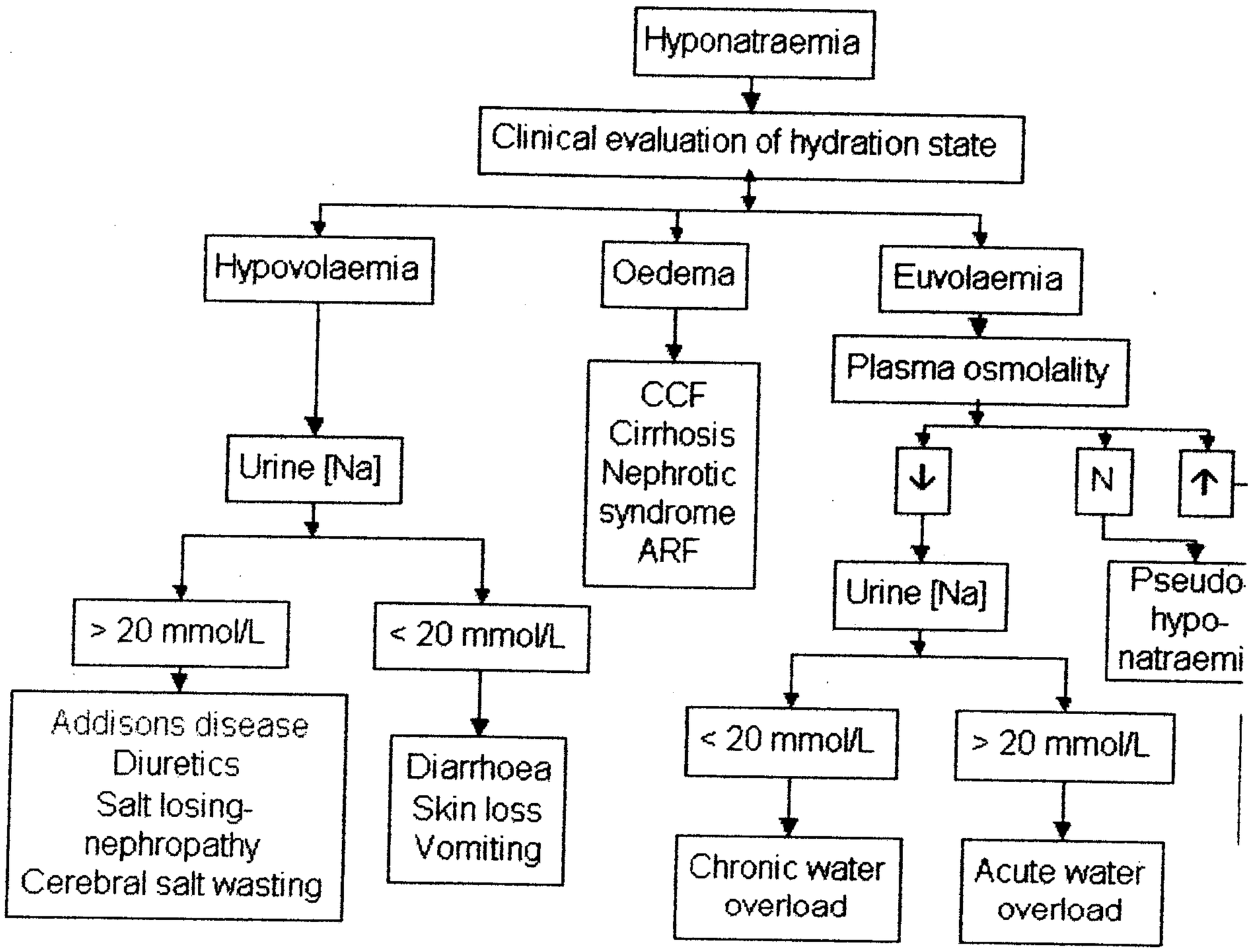
A recent analysis of hyponatraemia demonstrated a prevalence of 0.14% over a 6 month period in a district general hospital. After the exclusion of factitious causes, the majority of cases were due to chest infection, diuretics, cardiac failure, post-operative, other cases were related to malignancy and serotonin re-uptake inhibitors.

SIADH

The diagnosis of SIADH is by exclusion and is based on the evaluation of a patients hydration state, their general medical condition and presence of other diseases, drug therapy and the exclusion of endocrine causes of abnormalities of water homeostasis eg thyroid, pituitary and adrenal function. SIADH is characterised by plasma hypotonicity with concentrated urine with high sodium excretion. Measurement of ADH is rarely required.

Cerebral salt wasting

Cerebral salt wasting is characterised by dehydration, hyponatraemia, massive urinary sodium loss and typically complicates cases of hypothalamic damage. Patients with subarachnoid haemorrhage have raised concentrations of the brain natriuretic peptide which appears to be raised in proportion to the intracranial pressure. This is associated with a suppression of the normal salt and water homeostatic mechanism: both plasma ADH and the renin-aldosterone axis are suppressed. The diagnosis should be considered in all cases of cerebral injury since plasma volume loss > 10% occurs in 50% of patients with subarachnoid haemorrhage.



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