EDITORIAL

The Syndrome of Inappropriate Secretion of Antidiuretic Hormone

The etiology and pathogenesis of hyponatremia are usually readily detectable from the clinical setting in which this condition is encountered. Too little sodium from insufficient intake or excessive loss and too much water from excessive intake or impaired water diuresis account for almost all cases of hyponatremia. The underlying cause of these factors becomes apparent as the patient is studied.

However, hyponatremia may occasionally occur in which the mechanism for its production is obscure. This variety of hyponatremia has been called idiopathic, unexplained and asymptomatic. It may be found in such widely diversified conditions as central nervous system diseases, where it has been referred to as cerebral salt wasting, myxedema and acute porphyria. However, such hyponatremia should be of special interest to the chest physician since it is most frequently encountered in bronchogenic carcinoma and advanced pulmonary tuberculosis in which the term pulmonary salt wasting has been used.

The most logical explanation for this type of hyponatremia is that it is related to a sustained inappropriate secretion of antidiuretic hormone (ADH). The main features of this inappropriate ADH syndrome are: (1) hyponatremia and serum hypotonicity; (2) increased extracellular fluid volume; (3) urinary excretion of significant quantities of sodium despite hyponatremia resulting in hypertonic urine; (4) absence of dehydration, hypotension or edema; (5) no significant renal, hepatic, adrenal or cardiac disease; (6) normal or low blood urea nitrogen; (7) poor response of hyponatremia to hypertonic saline; (8) improvement in both hyponatremia and renal loss of sodium by fluid restriction.

In the usual forms of dilutional hyponatremia seen in congestive heart failure, cirrhosis and nephrotic syndromes, there is retention of both sodium and water by the kidneys. This syndrome under discussion is a special type of dilutional hyponatremia characterized by retention of water, but not salt. The stimulus provided in an unknown fashion by the underlying disorder (meningitis, head trauma, encephalitis, cerebral vascular accident, brain tumors, pituitary tumors, bronchogenic carcinoma, pulmonary tuberculosis, etc.) provokes an increased secretion of ADH resulting in retention of water, thereby diluting the extracellular fluid and causing lowered osmotic concentration of electrolytes. The normal and therefore the appropriate response of the osmoreceptor in the posterior pituitary to changes in the osmotic concentration of electrolytes such as sodium in the plasma is such that with lowered osmotic concentrations, ADH is suppressed producing a loss of free water by the kidney in an attempt to restore the plasma osmolality to normal. However, in patients with this syndrome, ADH secretion continues despite lowered osmotic pressure and is therefore inappropriate. In addition to the hypo-osmolality, the water retention results in an expansion of the extracellular fluid including an increased plasma volume. The appropriate response to increased plasma volume is stimulation of volume receptors which inhibit ADH secretion establishing loss of free water. Again, in these patients with expanded plasma volume, ADH secretion continues inappropriately.

An increase in plasma volume normally, as well as in patients with the syndrome of inappropriate secretion of ADH, brings about an increase in excretion of sodium. This response is mediated through the adrenal sodium-retaining hormone, aldosterone. A decrease in plasma volume gives rise to the secretion of aldosterone and sodium retention while an increase in plasma volume by inhibiting aldosterone secretion provokes
an increase in sodium excretion. A further consequence of the increased plasma volume is an increased glomerular filtration rate producing the filtration of more sodium. Thus, increased sodium in the urine in these patients is due to the suppression of aldosterone, as well as an increased glomerular filtration rate. The hyponatremia is a consequence of plasma dilution and sodium loss.

The laboratory and clinical data in the syndrome of inappropriate secretion of ADH indicate that the hyponatremia is different from that seen in true sodium-depleted states. In the latter situation, there is contracted blood volume with dehydration, hypotension, high hematocrit and high normal or elevated blood urea nitrogen. The more common causes of dilutional hyponatremia such as congestive heart failure, nephrotic syndrome and cirrhosis of the liver in which salt and water are retained can be eliminated on clinical grounds by the frequently accompanying edema, as well as low urine sodium concentration.

The diagnosis of inappropriate secretion of ADH is presumptively established when, in the absence of renal insufficiency, severe hepatic disease, congestive heart failure or other reasons for increased ADH activity, consistently hypertonic urine is associated with hyponatremia. Of both diagnostic and therapeutic significance is the correction of the electrolyte abnormalities when fluids are restricted below daily water loss. Such a negative water balance decreases the plasma volume resulting in a rise in sodium concentration.

The discovery of this syndrome is of more than academic interest since the continued intake of inordinate amounts of fluids could result in water intoxication because of impaired ability to excrete water.

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SELECTIVE BRONCHIAL ARTERIOGRAPHY
Anatomic evaluation of the bronchial (systemic) circulation is possible by selective bronchial arteriography. The findings in 22 patients are reported. Abnormal tumor vessels and enlarged bronchial arteries were found in some patients with primary and metastatic pulmonary neoplasms. The main value of this procedure may be in the differentiation of malignant and inflammatory disease and in the assessment of surgical operability of malignant disease. Moreover, this technic facilitates anatomic, physiologic and pathologic studies of the bronchial circulation.


OXYGEN INSUFFICIENCY IN PATIENTS WITH HYPERTENSIVE VASCULAR DISEASE
Functional investigation of the external respiration disclosed oxygen insufficiency in patients suffering from hypertensive vascular disease of the first, second and third stages without circulatory disorders. Upon an analysis of the data of ventilation and gas exchange, the author found not only evident, but also latent forms of oxygen insufficiency. Of 110 patients, 50 cases (45.6 per cent) oxygen insufficiency was revealed without any signs of circulatory disorders.