Sodium Homeostasis in Cirrhosis

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Renal complications of cirrhosis have received renewed investigative attention in the past decade. Prominent among the renal complications of cirrhosis is the progressive impairment of renal sodium leading to the formation of ascites and peripheral edema.

Patients with Laennec's cirrhosis manifest a remarkable capacity for sodium chloride retention and they excrete urine which is free of sodium (Farnsworth, 1948; Eisenmenger et al., 1950; Faloon et al., 1949). Extracellular fluid accumulates as ascites and edema. Cirrhotic patients who are unable to excrete sodium will continue to gain weight and accumulate ascites and edema as long as the dietary sodium content exceeds the maximal urinary sodium excretion. Ascites and edema formation will cease when sodium intake is limited (Epstein, 1979).

The abnormality of renal sodium handling in cirrhosis is not a static and unalterable condition. The cirrhotic patients may undergo a spontaneous diuresis (Gabuzda, 1970). Cirrhotics who excrete more than 10 meq of sodium daily in urine and have normal GFR are more Jikely to undergo such spontaneous diuresis. Patients with reversible liver disease such as fatty liver of the alcoholic also tend to respond favourably when rested and fed (Epstein, 1979).

The pathogenetic events leading to the deranged sodium homeostasis of cirrhosis is simplified by a consideration of the afferent and effer-. ent events which take part ir this derangement.

Afferent events include the detector element responsible for the recognition of the degree of volume alterations as well as a consideration of the extracellular fluid translocations or interstitial fluid compartments which characterize advanced liver disease (Epstein, 1979).

Renal sodium retention accompanying cirrhosis is attributable primarily to enhanced tubular reabsorptioii rather than to alterations in the filtered 1oad of sodium. The mediators of the enhanced tubular reabsorption of sodium in cirrhosis and their re'ative participatic'n in sodium retention have not been &ucidated comp'etely. Several mechanisms have been suggested including (a) hyperaldosteronism, (b) alterations in intra renal blood flow distribution, (c) an increase in sympathetic nervous system activity, (d) alterations in the endogenous release of renal prostag- landins (e) changes in the Kallikrein Kinin system, (f) the possible role of humoral natriuretic factor and (g) vasoactive intestinal peptide.

To sum up, the renal sodium retention of advanced liver disease is a complex pathophysiologic phenomenon due to numerous causes. Knowledge concerning these abnormalities is still incomplete and many of the hormonal mechanism responsible are unknown. However measurement of hormones that affect renal hacrnodynamics and renal sodium handling provide a basis for a more complete understanding of the mechanism that promotes sodium retention in cirrhosis.

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