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Pathophysiology of Ascites Formation in Cirrhosis of the Liver

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Summary

Current concepts of the pathophysiology of ascites formation in cirrhosis of the liver have become more complex. Traditionally, the initiating event of renal sodium and water retention in cirrhosis was considered to be ascites formation ("underfilling" hypothesis) or primary renal dysfunction ("overflow" hypothesis). Changes in systemic, splanchnic and renal hemodynamics, as well as of volume regulating hormones observed in cirrhosis are compatible with a decrease in effective blood volume as suggested by the "underfilling" hypothesis. These changes, however, have been shown to precede ascites formation. This observation, together with the demonstration of an increase in total blood volume in cirrhosis prompted the "overflow" hypothesis. However, many studies are incompatible with this concept and, in addition, the agent causing primary renal sodium retention in cirrhosis still remains to be defined. The recently proposed "vasodilation" hypothesis reconciles the most salient features of both theories, proposing peripheral arterial vasodilation as the initiating event of decreased effective blood volume and renal sodium retention. Further studies are needed to elucidate the temporal relationship and more precisely define the character of hemodynamic, humoral and renal changes in cirrhosis of the liver.

Key words

Ascites formation – Liver cirrhosis – Underfilling – Overflow – "Vasodilation" hypothesis – Hemodynamics – Volume regulating hormones – Sodium retention

The regulation of sodium and water homeostasis has been a puzzling subject of investigation for many years (1). In particular the concepts of pathophysiology of volume regulation and ascites formation in cirrhosis of the liver have become more and more complex (2–8). Several hypotheses have been elaborated in an attempt to explain ascites formation in liver cirrhosis: the "underfilling", the "overflow" and, most recently, the "vasodilation" hypothesis (9). These concepts will be discussed in this review, incorporating hormonal and hemodynamic changes in cirrhosis of the liver.

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The "underfilling" hypothesis

The classical "underfilling" theory (10) proposed that ascites formation decreases centrally "effective" blood volume (EBV), thus inducing hormonal changes, resulting in renal sodium retention (Fig. 1): the cirrhotic liver produces less albumin, thus reducing plasma oncotic pressure. Hepatic venous outflow obstruction increases hydrostatic pressure in the portal circulation. These changes in Starling forces increase extravasation in the splanchnic circulation. Once the ability of the lymphatic system to return lymph to the circulation is exceeded, protein-rich fluid spills from the hepatic sinusoids into the peritoneal cavity and ascites becomes overt (11, 12). Ascites formation, as well as the formation of arteriovenous shunts (13) and reduction of peripheral vascular resistance (14), reduces non-splanchnic plasma volume. This reduction in "effective" blood volume activates sodium-retaining hormonal systems such as the renin-angiotensin-aldosterone system or the sympathetic nervous system, resulting in renal sodium retention. Owing to the above-mentioned pathophysiological changes the retained fluid cannot replenish the reduced EBV, but contributes to ascites formation, thus creating a vicious circle.

Some problems with this concept have been caused by the inability to clearly define and determine the "effective" blood volume. EBV has been introduced as an operational definition (15) of that part of the blood volume with an effect on receptors of the volume regulatory systems. To date it has not been unanimously agreed whether changes in this effective volume concern the arterial blood volume, the central blood volume, or both. In an attempt to quantitate EBV, it has been defined as total blood volume minus the volume of the portal circulation (16), or as the total intrathoracic blood volume (17). It has been shown that, despite an increase in total blood volume of up to 30% (17–20), EBV was reduced. Such changes were most marked in patients with ascites, while cirrhotic patients without ascites exhibited smaller increases in total blood volume and decreases in EBV as compared with controls.

Since changes in total blood volume primarily influence the low-pressure system (for review see 21), an influence on the atrial volume receptors (22, 23) might be anticipated. However, recent data (17, 20) suggest normal vascular filling of the cardiac compartment, but diminished filling of the aorta in cirrhosis. Thus, baroreceptors of the high-pressure system (24, 25) might be of pre-eminent importance in sensing reductions in EBV.

A number of hemodynamic and hormonal changes, as well as effects of maneuvers replenishing EBV are compatible with the "underfilling" hypothesis:

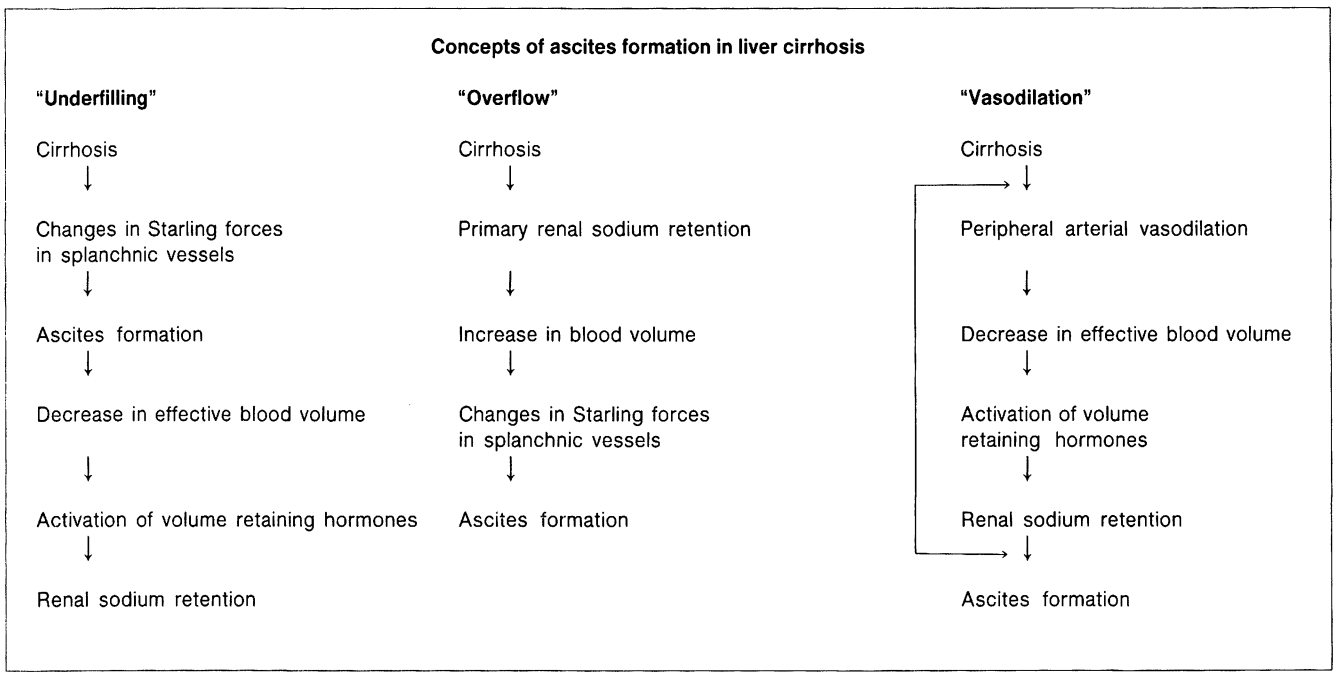

Fig. 1 Concepts of ascites formation in liver cirrhosis

Systemic hemodynamics: compared with controls, patients without ascites, and more so patients with ascites, exhibit decreased arterial blood pressure, increased heart rate and increased cardiac output. As mentioned before, EBV is reduced despite an increase in total blood volume. Peripheral vascular resistance is decreased (17, 26–28).

Splanchnic hemodynamics: portal venous pressure, splanchnic inflow, and azygos blood flow are increased in cirrhosis, and even more so in patients with ascites, whereas hepatic blood flow is reduced (17, 26–28).

Renal hemodynamics: renal blood flow, renal perfusion pressure and glomerular filtration rate are decreased at increased renal arteriovenous shunts (29–31).

Hormonal changes: with increasing decompensation, cirrhotic patients exhibit an activation of the renin-angiotensin-aldosterone system (28, 32, 33) and of the sympathetic nervous system, as indicated by plasma norepinephrine concentration (33, 34). Stimulation of sympathetic nervous system activity could contribute to sodium retention by changing the pattern of renal perfusion towards juxtamedullary nephrons (35), by direct effects on the tubular epithelium (36) or by stimulating renal renin secretion (37).

Natriuretic hormones: the existence of a "third factor" (38, 39) has long been proposed. This substance, presumably a peptide with digoxin-like immunoreactivity (40), has not been clearly characterized and quantified so far, and the concept of a deficiency of this natriuretic hormone in patients with cirrhosis (41) has not yet been confirmed. Recently, concentrations of digoxin-like immunoreactive substances have even been found to be increased in plasma and urine of patients with chronic liver disease (42). The first well-defined natriuretic hormone, the atrial natriuretic factor (ANF), does not show reduced plasma concentrations in cirrhosis (for review see 2). However, plasma concentrations of ANF relative to plasma renin activity or to plasma aldosterone concentration are reduced in cirrhosis, and ANF release upon volume stimulation seems to be blunted in patients with ascites (32). Renal response to endogenously released ANF (32) or to exogenously administered ANF (43, 44) would seem to be reduced in patients with ascites, possibly due to changes in renal ANF receptors (45).

Water retention might be due to non-osmotic stimulation of vasopressin (46). Interpretation of the importance of vasopressin is hampered by methodological problems: plasma concentrations of healthy subjects or of patients with cirrhosis following a water load, were often found to be below the sensitivity limit of radioimmunological detection (46, 47). Several observations indicate a minor role for vasopressin in the volume retention of cirrhosis: basal plasma concentrations in cirrhosis were not increased (for review see 47); in non-ascitic patients, water excretion was reduced despite normal vasopressin concentrations (48); following central volume expansion by water immersion (47) or peritoneovenous shunting (49), massive diuresis ensued despite unchanged or only minimally reduced plasma vasopressin concentrations.

The "underfilling" hypothesis suggests that ascites formation precedes renal sodium retention. The above-mentioned hormonal and hemodynamic changes and the well known fact of sodium retention in non-ascitic patients are not compatible with this concept, and were the main reason for the search for alternative concepts.

The "overflow" theory

Lieberman observed normal or increased total blood volume in patients with cirrhosis and ascites, which was

no different from patients with spontaneous diuresis (16). He therefore proposed the "overflow" hypothesis of ascites formation (50). This concept presumes a primary renal salt- and water-retention in cirrhosis, resulting in an increase in plasma volume; owing to the changes in Starling forces in the portal circulation, ascites secondarily becomes overt as an "overflow" from the intravascular space (Fig. 1)

This concept of ascites formation was supported by the observation that renal sodium retention precedes ascites formation in a dog model of cirrhosis (51, 52). The significance of these data was, however, questioned on account of the possible nephrotoxicity of nitrosamine, which was used to induce cirrhosis, the short interval between sodium retention and ascites formation, and the rather low level of sodium retention (53).

The idea of primary renal dysfunction in cirrhosis was supported by the observation of ascites in patients with compensated cirrhosis following administration of mineralocorticoids and thus lacking mineralocorticoid "escape" (54). This finding, however, would also be compatible with increased renal tubular sensitivity to mineralocorticoids, a decreased release of natriuretic factors, or a decreased sensitivity to these factors in cirrhosis.

A problem with the "overflow" theory is the as yet unresolved question as to why the kidney should retain sodium in liver disease. Since splanchnic blood volume was found to be increased in cirrhosis, some hepatorenal reflex has been hypothesized to cause renal functional impairment, but has not been clearly demonstrated so far.

Moreover, a number of observations are hardly compatible with the "overflow" theory: decreased mean arterial blood pressure, an increased heart rate, and activation of the renin-angiotensin-aldosterone (26, 32, 33) and of the sympathetic nervous system (33, 34) have been observed in cirrhotic patients without ascites.

Since neither the "underfilling" nor the "overflow" concept can fully explain ascites formation in cirrhosis, a new concept has recently been introduced, the "vasodilation" hypothesis (9).

The "peripheral arterial vasodilation" hypothesis

The vasodilation hypothesis supports the "underfilling" theory with the concept of peripheral arterial vasodilation as the initiating event (1, 9) (Fig. 1): peripheral arterial vasodilation initiates a moderate decrease in effective arterial blood volume, and, due to reduced cardiac preload, an increase in cardiac output. In a compensatory reaction the body increases the plasma concentration of renin, aldosterone, norepinephrine and vasopressin, resulting in moderate renal vasoconstriction with sodium and water retention. The resulting expansion of plasma volume then reduces the activation of sodium retaining hormonal systems to a normal level; this might explain plasma concentrations of renin, aldosterone, norepinephrine and vasopressin within the normal range, as observed in some patients without ascites (54–56). With increasing severity of cirrhosis, peripheral vasodilation and the subsequent decrease in EBV, even marked activation of sodium and volume retaining hormones cannot achieve replenishment of effective blood volume. These changes, together with a decreased plasma oncotic pressure by hypoalbuminemia, induce ascites formation. The "vasodilation" hypothesis, which is compatible with most observations on renal, hemodynamic and hormonal changes in cirrhosis, is supported by some recent studies (34, 57–59): water excretion was significantly augmented in hyponatremic patients with cirrhosis and ascites by head-out water immersion, inducing an increase in central blood volume (58). Increase in peripheral vascular resistance by infusion of norepinephrine in the same patients induced a comparable diuresis. A combination of both maneuvers significantly increased mean arterial pressure as well as renal perfusion pressure and peripheral vascular resistance, resulting in normalization of natriuresis and diuresis.

The importance of a decrease in distal tubular sodium delivery, caused by hemodynamic and hormonal changes, for sodium retention in cirrhosis has been demonstrated in another recently reported study (34): spironolactone treatment increased urinary sodium excretion as well as fractional sodium excretion more markedly in a group of patients with ascites than in a group of patients without ascites. In these same patients water immersion augmented natriuresis more markedly than immersion without spironolactone pretreatment. The effects of these maneuvers, as well as changes in hemodynamic and hormonal parameters were more pronounced in the patients with ascites than in those without ascites.

A close chronological relationship between a decrease in arterial pressure and the onset of hyperaldosteronism and sodium retention in the development of cirrhosis has recently been demonstrated in cirrhotic rats (59), lending further support to the "vasodilation" hypothesis.

A number of factors may be responsible for the vasodilation in cirrhosis: arteriovenous shunts in the splanchnic circulation, but also in the skin and lungs may contribute to arterial underfilling and to the ensuing sodium retention (60–62). Several bioactive compounds have been thought to be involved in the peripheral arterial, mainly splanchnic, vasodilation and in the renal vasoconstriction in cirrhosis (for review see 5, 63, 64): endotoxins and platelet activating factor (65–57), kinines (68–70), endogenous opioid peptides (71), false neurotransmitters (72), leukotrienes (73), prostaglandins (74, 75), substance P (76) and vasoactive intestinal peptide (77, 78). Furthermore, increased circulating levels of norepinephrine might induce downregulation of beta-adrenergic receptors (79), thus causing desensitization to sympathetic nervous system activity (80, 81).

The return of the renin-aldosterone and of the sympathetic nervous system activation to normal levels in early cirrhosis, as postulated by the "vasodilation" theory, as well as the temporal relationship of hemodynamic and hormonal changes to sodium retention and ascites formation, remains to be more clearly defined. Longitudinal studies may further elucidate this least well defined part of the "vasodilation" theory.

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