CARDIO-HEPATIC SYNDROME – AN UNDER-REALISED ENTITY?

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Heart failure affects many organs other than the heart due to the chronic generalised deficiency of required metabolites in most of the body systems. Liver is no exception; but rather more vulnerable for such injury. There is a resurgence of interest in this aspect in last few decades. A term 'Cardio hepatic syndrome' is coined and is being increasingly used (akin to cardio-renal syndrome) in the recent past.

Pathogenesis of liver affection in heart failure:

The blood supply of the liver is complex and that makes it unusually vulnerable to acute circulatory disturbances. The degree and pattern of liver injury depend on the proportion of venous congestion and reduced perfusion. The former abnormality is called 'nutmeg' and results in elevations of alkaline phosphatase (AP), g-gluamyl transferase (GGT) and direct and indirect bilirubin. Reduced perfusion leads to acute centrilobular hepatocellular damage and necrosis and reflects by elevated serum amino tranasferases [1, 2].Acute cardiogenic liver injury (ACLI), Shock liver, Ischemic hepatitis and Hypoxic hepatitis are modern terms used to denote the same pathogenic process. However such changes can also be seen in respiratory failure and septic shock. Most believe that ACLI is a more accurate descriptive term [3].

Clinical spectrum

Though detection of abnormal biochemical valves on routine testing is a common phenomenon symptoms referable to liver injury after the hemodynamic stability is obtained include- apathy, weakness, confusion, tremors, jaundice, coma or bleeding diathesis. The symptoms may peak around third day and fade off in 8 to 10 days' time [3].

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Biochemical abnormalities:

Elevated total bilirubin is the most common abnormal liver biochemical abnormality occurring in about 70% of HF patients; a fact established many decades back [4].Abnormal liver biochemistry in acute and chronic heart failure is authentically documented in a few recent trials. In SURVIVE trial, a large study of ADHF nearly half of the subjects showed elevated transaminases, AP or both [5].Data from the CHARM trial documented ALT elevation in 3.1%, low albumin in 18.3% and elevation of total bilirubin in 13% of the 2679 patients of chronic heart failure [6].Anaemia and raised natriuretic peptides are often associated with abnormal Liver parameters [7].

Prognostic implications:

The abnormal biochemical parameters like elevated bilirubin, AP, ALT and AST are utilised as predictors of poor prognosis in chronic heart failure. On proper decongestive therapy these parameters tend to normalise over days to weeks [7, 8].

In this issue the original article on "Should We Confine The Cardio Hepatic Syndrome only In Congestive Cardiac Failure Patients?" by B. Rajendra et al published their results of as simultaneous analysis of liver abnormalities and cardiac biomarkers in 15178 patients admitted to ICCU [majority of them being patients of heart failure or acute coronary syndrome). Among them after excluding those with primary liver disease, abnormal LFT was a frequent occurrence. SGPT elevation was the earliest abnormality seen in about 15.5% of the patients. While in 25% no obvious aetiology could be found, about half of them were using drugs which had the potential for liver damage. The authors believe that the spectrum of cardio hepatic syndrome beyond the liver abnormalities in heart failure is not fully defined and have rightly concluded that the syndrome deserves further study.

Take –Home Message:

It is not clear at present if a 'Cardio-hepatic syndrome' exists as a distinct entity, in lines of Cardio-renal syndrome. The biochemical abnormalities reflecting liver injury are not uncommon and are consequent to the hemodynamic effects like passive congestion and reduced perfusion. Certainly this group of patients deserve a relook and further research.

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