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Review Article

Whispering of the Liver and other Body Organs

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Abstract:

Abstract:

The most important organ and second- largest organ is the liver. The liver performs a wide range of tasks for maintaining health and homeostasis and also breaks down a variety of drugs. liver illnesses might have a negative impact on health. Our aim is to show the new generations that organ deserves respect and awareness of importance and demonstrates the crosstalk between the liver and other body organs.

Key words: liver; disease; alcohol; renal; heart

Introduction

The most important organ and second- largest organ is the liver. The liver performs a wide range of tasks for maintaining health and homeostasis and also breaks down a variety of drugs. liver illnesses might have a negative impact on health [1]

Viewpoints:

Beginning with Oral Manifestations: Alcohol-related liver disease, nutritional deficiencies cause glossitis, atrophic tongue, HCV, IBD/PSC cause fissured tongue, alcohol-related disease, HCV cause Parotid gland enlargement/sialadenitis, Coagulopathy causes petechiae, telangiectasia, hematoma, gingival bleeding, reduced wound healing, biliary atresia, malnutrition cause discolorations of teeth, enamel hypoplasia, delayed eruption of teeth, HCV, PBC, IBD/PSC cause Xerostomia Hyposalivation, Cytopenia, immunocompromised cause Periodontal disease, hyposalivation/xerostomia alcohol-related liver disease cause tooth decay (caries), alcohol-related disease gastric reflux cause erosion, HCV PBC cause oral lichen planus lichenoid lesions, HCV causes Leukoplakia, IBD/PSC, PBC causes Mucosal ulcers, immunocompromised causes Candidiasis, angular cheilitis[2] Patients with liver cirrhosis exhibit several features of gut dysfunction. Gastric emptying and small bowel transit are prolonged. This may be related to disturbances in postprandial glucose, insulin, and ghrelin levels, which, in turn, appear to be associated to insulin resistance, a common finding in cirrhosis. Also, bacterial overgrowth and intestinal barrier dysfunction- especially with portal hypertension- which is related to bacterial translocation and permeation of intestinal bacterial products, e.g., endotoxin and bacterial DNA[3]

Gut microbiota imbalances, or dysbiosis, are implicated in liver conditions such as viral hepatitis, colorectal cancer metastasis to the liver, and drug-induced liver injury. Dysregulation of the gut-liver axis contributes to the development and progression of various liver diseases. Chronic hepatic inflammation, fibrosis, and portal hypertension disrupt the intestinal barrier, leading to systemic inflammation and complications including multi-organ failure or even death. Human clinical trials are emerging. For example, increased intestinal permeability is closely linked to liver disease. High-fat diets and alcohol intake can weaken the

intestinal barrier, allowing bacterial toxins to reach the liver and trigger inflammation. Over time, this persistent inflammation contributes to conditions like fatty liver disease. Also, specific gut microbiota patterns are being linked to improved responses to hepatic cancer immunotherapies in humans[4] Many systemic conditions affect both the liver and the kidneys. Certain liver diseases are also common in patients with chronic renal disease, especially viral hepatitis, either because the renal disease occurs as a complication of viral hepatitis, or the viral hepatitis is acquired as a result of dialysis. Renal tubular dysfunction is also frequently observed with cholestasis. However, liver complications of renal diseases are extremely uncommon as nephrogenic ascites and nephrogenic hepatic dysfunction. Nephrogenic ascites can mimic liver cirrhosis with ascites, and it improves with renal transplantation. Nephrogenic hepatic dysfunction is a manifestation of renal cell carcinoma, which settles with the removal of the renal cell carcinoma. Viral hepatitis should be treated, if possible, before renal transplant. If cirrhosis is present, renal transplant alone is contraindicated; combined liver and kidney transplantation is indicated in patients with end-stage renal disease and advanced cirrhosis[5] HRS-1, also called HRS-AKI constitutes a form of AKI unique to the state of cirrhosis and portal hypertension. Although HRS-1 is a condition primarily characterized by marked renal vasoconstriction and kidney hypoperfusion, other pathogenic processes, such as acute tubular injury and renal vein congestion, can overlap and further complicate the course of HRS-1. ALF can lead to AKI through mechanisms that involve systemic inflammation, direct drug toxicity, or bile acid-induced tubulopathy. Moreover, the growing prevalence of NASH is changing the spectrum of chronic kidney disease in cirrhosis[6] The liver makes toxic substances in the body harmless. When the liver is damaged, these poisons build up in the bloodstream and affect the function of the nervous system. The result may be HE that can occur suddenly and you may become ill very quickly. Causes of HE include: Hepatitis (uncommon to occur this way) Blockage of blood supply to the liver, poisoning by toxins or medicines, constipation, upper gastrointestinal bleeding. In some cases, HE is a short-term problem that can be corrected. It may also occur as part of a long-term (chronic) problem from liver disease that gets worse over time[7] The liver affects the skin in several ways: Detoxification: If the liver is inefficient, toxins can build up, leading to skin issues like acne and inflammation, also may contribute to oxidative stress, accelerating premature ageing and impairing the skin's radiance and elasticity. Metabolism of Nutrients: The liver processes vitamins (A, D, E, K), minerals, and fatty acids necessary for skin health, deficiencies in these nutrients can result in dryness and decreased elasticity, vitamin A deficiency can impair cell turnover, causing flaky skin. Production of Proteins and Lipids: The liver produces albumin, which maintains blood osmotic pressure and prevents fluid retention or puffiness. It also makes cholesterol and triglycerides, essential for the lipid barrier of the skin. A compromised lipid barrier can lead to dehydration, loss of firmness, and increased sensitivity. Hormone Regulation: The liver metabolises and regulates hormones like estrogen. Hormonal imbalances can trigger conditions like melasma, acne, or sagging skin. Effective hormone metabolism supports even skin tone and texture.

Antioxidant Production: The liver produces glutathione, a powerful antioxidant. Adequate glutathione levels protect against premature ageing, supporting a more youthful appearance.

Habits harm liver and kin: Toxins from cigarettes, Alcohol dehydrates the skin, Sleep deprivation accelerates ageing, High sugar diet, Sedentary lifestyle, Overuse of certain supplements or medications, Dehydration reduces the liver's ability to detoxify; also reduces skin's elasticity and skin hydration, Stress, pumps out cortisol causing liver damage and skin produces excessive sebum which worsen conditions like acne, seborrheic dermatitis, and rosacea. Cortisol breaks down collagen and elastin, which accelerates premature ageing. [8] A variety of chest manifestations are seen in patients with chronic liver diseases, namely hepatopulmonary syndrome, portopulmonary hypertension, intrathoracic portosystemic collaterals, hepatic hydrothorax, infections, drug-induced changes, manifestations of hepatocellular carcinoma, gynecomastia, acute respiratory distress syndrome, autoimmune changes, aspiration pneumonitis and changes due to α1-antitrypsin deficiency[9] The heart causes liver diseases as congestive hepatopathy, cardiogenic ischemic hepatitis and the liver causes heart diseases as cirrhotic cardiomyopathy, hyperdynamic circulation, systolic and diastolic dysfunction, Stress-Induced cardiomyopathy.

Cardiac disorders in NAFLD: coronary artery disease, and cardiac arrhythmias. Moreover, the coronary microvascular and endothelial function shown to be impaired in patients with NAFLD, they have left ventricular diastolic dysfunction causing heart failure. Again, NAFLD has been associated with morphological and valvular heart abnormalities. Hepatic steatosis is associated with aortic-valve sclerosis and mitral annular calcification, both known to enhance the risk of cardiac arrhythmias and heart failure. Cardiac rhythm disturbances are also linked to NAFLD as atrial fibrillation, QT prolongation, and ventricular arrhythmias leading to sudden cardiac death[10] The liver is the central organ in the growth hormone/insulin-like growth factor (GH-IGF) axis, and the acid-labile subunit (ALS) is one important component of that system in humans. The ALS is linked to the far end of the GH-IGF axis and is an integral constituent in the formation of the 150-kDa IGF/insulin-like growth factor binding protein-3 (IGFBP-3) complex that transports 95%of the IGF-I in the circulation and determines its bioavailability to tissues.

The complex (IGF-I, IGFBP-3, ALS) has an important function: to prevent the hypoglycemic effect of IGFs, prolong their biological half-life, and prevent cross-endothelial transport of IGFs. The liver predominantly secretes ALS, and contribution from extrahepatic tissue may exist. The binding proteins such as ALS, produced in tissues other than the liver, could account for a paracrine effect or local need as compared with the endocrine function of the liver. In liver cirrhosis, ALS serum levels are decreased and participate in the extreme alterations that occur in the GH-IGF axis in this state. Liver cirrhosis is characterized as one of the GH-resistant states, whereby GH serum levels are high in the presence of low IGF serum levels. Other prominent alterations include a paradoxical increase in serum GH levels in response to thyrotropin-releasing hormone, reduced number of liver GH receptors, a parallel decrease in serum GH-binding protein (GHBP) and reduced serum levels of IGF-I and its binding proteins. liver cirrhosis may influence the GH-IGF axis profile by several mechanisms, the most important being the nutritional state; portal hypertension, causing shunting of blood away from the liver; decreased hepatic functional reserve, causing deficiency in serum IGF-I and GHBP; and metabolic disturbances, mainly those associated with glucose metabolism. These changes may have significant therapeutic and diagnostic-prognostic aspects. The classical Child-Turcotte classification is not optimal in predicting the prognosis of patients. It seems that the classical groups have further subgroups of patients that differ in prognosis. Example, patients with liver cirrhosis

may run a disease course that is affected mainly by the level of portal hypertension. These patients tend to develop ascites at an early stage and bleed from varices. It is essential to differentiate the effect of portal hypertension from that of low residual liver function. The binding proteins, produced mainly by the liver, correlated very well with the hepatic functional reserve. It was suggested that using a GH-IGF-I stimulation test was comparable to the classical Child-Turcotte classification, has important survival predictive value. Also, comparable to other provocative tests, but with the advantages of not using pharmacological materials and no patient discomfort due to blood testing. The ALS can also be looked at as a prognostic factor[11] Thyroid diseases must be excluded in transaminase elevation. Drugs such as propylthiouracil, may induce liver damage and other drugs such as amiodarone, carbamazepine, and several chemotherapeutic agents can lead to both thyroid and liver abnormalities. Liver diseases such as hepatitis, hepatocellular carcinoma, and cirrhosis may cause altered levels of thyroid hormones, and alcoholic liver disease, both due to the noxious substance ethanol as well as to the hepatic damage it causes, may be responsible for altered thyroid function. Both excess and insufficiency of adrenal function may result in altered liver function, and adrenocortical dysfunction may be present in patients with cirrhosis. Again, alcohol may be associated with pseudo-Cushing syndrome. While oestrogens are related to cholestatic liver damage, androgens are the culprit of adenomas and hepatocellular carcinoma, among others. Chronic liver disease, on the other hand, has profound effects on sex hormone metabolism, inducing feminization in men and infertility and amenorrhoea in women. Lastly, metabolic syndrome, links the liver damage ranging from steatosis to cirrhosis, to the endocrine features of the syndrome, including insulin resistance, central obesity, and hyperlipidaemia[12] Hepatobiliary injury is frequently associated with significant morbidity and mortality in patients with haematologic-oncologic disorders with reported cases of acute liver failure and cirrhosis development. For this reason and owing to the growing use of haematopoietic stem cell transplantation (HSCT), the diagnosis and management of liver complications is important. The differential diagnosis is broad and may initially be classified according to the pattern and severity of serum liver test abnormalities. Frequent hepatobiliary conditions include drug-induced liver injury, neoplastic infiltration of the liver, sepsis-associated liver injury, sinusoidal obstruction syndrome (SOS), graft vs. host disease (GVHD), haemophagocytic lymphohistiocytosis (HLH), viral hepatitis, ischaemic hepatitis, Budd-Chiari syndrome and splanchnic thrombosis, and nodular regenerative hyperplasia (NRH), among others. SOS typically manifests post-HSCT with hepatomegaly, abdominal pain, ascites, weight gain, peripheral oedema, transfusion-refractory thrombocytopenia, and jaundice. GVHD, on the other hand, may have a different presentation in the acute (<100 days) vs. chronic (>100 days) setting. Acute GVHD occurs in up to 62% of patients post-allogenic HSCT. While the liver is infrequently involved compared to other organs in acute GVHD, GVHD with liver involvement is associated with higher HSCT-related mortality. The hepatic manifestation often presents as cholestatic disease alongside cutaneous and gastrointestinal symptoms. Nodular regenerative hyperplasia NRH can develop in some patients with myelo- or lympho-proliferative disorders or those who have received high-dose chemotherapy. NRH can lead to non-cirrhotic portal hypertension and its complications, and may even be an indication for transplantation. HLH, conversely, is a multisystem condition characterized by inflammatory response with high mortality. Patients present with jaundice, persistent high-grade fever, pancytopenia, splenomegaly, haemophagocytes in the bone marrow and biochemical abnormalities as hyperferritinaemia, but it may also present as acute liver failure. Primary and secondary hepatic involvement is also possible in haematologic malignancies, including lymphomas, myeloid sarcoma(chloroma), multiple myeloma, and Castleman disease. Suggestive findings of a haematologic neoplasm as the underlying cause of liver lesions include young age, abnormal bone marrow biopsy findings, unexplained fever, and night sweats. In allogeneic HSCT recipients, viraemic donors can transmit viral hepatitis B and C to recipients. In patients with chronic hepatitis B virus infection or previous exposure to the virus, the disease may reactivate after chemotherapy or HSCT[13] Various chronic liver diseases inevitably end up with cirrhosis of the liver, and this comes with a whole range of haematological complications such as anaemia, thrombocytopenia, coagulopathy, leukopenia, and haemolytic disorders, which are known to contribute to morbidity and mortality in cirrhotic patients significantly[14] Autoimmune hepatitis causes defective tear secretion and stability dry eye syndrome peripheral ulcerative keratitis uveitis, Vitamin A deficiency causes Night blindness retinal dystrophies photoreceptor cell death keratomalacia corneal ulceration corneal xerosis, Chronic alcohol use/cirrhosis causes Bitot spots, corneal necrosis, bilateral ocular pain, photophobia, and decreased visual acuity. Galactosemia causes Infantile cataracts. GSD type V- McArdle disease causes Pattern dystrophy of retinal pigment epithelium. Biliary Disorders cause Visual impairment, refractive errors, and optic nerve damage. Zellweger Syndrome Causes corneal opacification, cataracts, glaucoma, pigmentary retinopathy, and optic atrophy. Mucopolysaccharidoses causes amblyopia, strabismus, and large refraction defects, corneal clouding, retinopathy, optic neuropathy, glaucoma, and optic nerve abnormalities. Niemann-pick disease Causes Cherry-red macula, corneal opacifications. HCV Causes Retinal vasculitis, keratoconjunctivitis sicca, dry eye syndrome, keratitis, scleritis, and retinopathies, Mooren's ulcer, Sjogren's syndrome[15]

Conclusion and recommendations:

The liver has many functions and as Dr. Fleming said " Nature did that. I only discovered it by accident." [16] It may be injured during the course of many systemic diseases. The mechanisms of injury can be broadly divided into four pathways: vascular, toxic, immune, and hormonal. Hormonal effects are principally involved when overnutrition leads to hyperinsulinemia followed by hepatocellular necrosis[17] So, in the past The liver, considered the centre of life and was examined by well trained religious experts[18] As Imhotep was considered a god of medicine in the past[19] we should be pioneers in protecting the liver and elucidating that it deserves respect and awareness of importance by the new generations in the present.

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