INTRODUCTION

Chronic hepatitis and cirrhosis are stages in the progression of many liver diseases of different aetiology , *Chronic hepatitis* is a histological diagnosis, defined as chronic hepatic inflammation persisting for more than 6 months. The histological features were previously divided into persistent, lobular and active chronic hepatitis, in ascending order of severity , chronic hepatitis is now described by aetiology, grade and stage. The grade is a measure of the severity of the inflammatory process and the stage refers to the degree of fibrosis. Some causes, including viral, autoimmune, alcohol, drugs and metabolic disorders, are specifically amenable to treatment **(Travis etal.,2005).**

Cirrhosis is also a histological diagnosis that is the end stage of the process of hepatic damage. It is defined as disruption of normal hepatic architecture by fibrosis with nodular regeneration. Fibrosis implies irreversible liver damage, but progression to decompensated liver disease or, in some circumstances, hepatoma, can often be delayed by treatment **(Travis etal.,2005).**

(Table 1) Diagnosis of specific causes of chronic liver disease (Travis etal.,2005).

Viral hepatitis		
	B	HBsAg and HBeAg positive. Viral DNA by PCR rarely indicated. Orcein stain positive on liver biopsy.
	C	HCV antibody by ELISA, confirmed by RIBA testing. HCV RNA by PCR is indicated for monitoring interferon therapy. Lymphoid follicles characteristic on biopsy.
Alcohol		History, random alcohol γGT, raised MCV, elevated IgA. Fatty infiltration, megamitochondria, Mallory's hyaline on biopsy.
Autoimmune		Antismooth muscle antibody titre >1:80, antinuclear antibody >1:80. Elevated IgG titre, predominance (8:1), associated thyroiditis.
Metabolic		
	Haemocbromatosis	Serum ferritin >1000 μg/l (also in alcoholics or chronic inflammation) Fe/TIBC ratio >80%, HLA A3 positive. Perl's stain positive on biopsy.
	Wilson's	Serum caeruloplasmin <0.2 g/l. Increased urinary copper (>0.1 mg/ 24 hourse). Increased liver copper.
	α1-antitrypsin deficiency	serum α, α antirrypsin <0.2 g/l, Pizz Serum α1 antitryspin <0.2 g/l, PiZZ phenotype on electrophoresis. PAS positive globules on biopsy.
Cholestasis		
	Primary biliary cirrbosis	Antimitochondrial antibody titre>1:250, M2 antigen specific. Elevated serum IgM. Bile duct, proliferation, lymphoid aggregates and granulomas on biopsy.
	PSC	ERCP. Sigmoidoscopy abd biopsy (80% associated with ulcerative colitis).
	Drugs	History. Amiodarone, methotrexate, nitrofuration, α-methyldopa, etc. Wide variety of feature on biopsy
Hepatic venous obstruction		
	Budd-chtari syndrome	Prothrombotic states (tumour, polycythaemia, antiphospholipid syndrome). Dropper ultrasound or hepatic venography.
	Veno-occlusive Disease	Biopsy shows occlusion and hyaline necrosis of small hepatic veins.

	Constructive pericarditis	Clinical signs, echocardiogram.
Cryptogenic		All other causes excluded (15-30%)

Rheumatic manifestations in patients suffering from hepatitis are most commonly due to viral infections from hepatitis B (HBV) or C (HCV) virus , although the overall number of patients who develop rheumatic syndromes is relatively small, nevertheless their appearance is usually associated with a number of diagnostic and therapeutic challenges **(Vassilopoulos et al., 2009).**

Hepatitis B (HBV) and hepatitis C (HCV) virus infections and their complications are major health concern throughout the world, rheumatological manifestations of these infections are also frequent and include arthralgia, myalgia, arthritis, vasculitis and sicca syndrome **(Aydeniz et al., 2009) .**

Simple arthalgias are common in HCV patients (20-50%), but true inflammatory arthritis is rather uncommon occurring in less than 5% of patients , the appearance of arthritis in the setting of chronic HCV infection could be related to the virus itself either directly (HCV- associated arthritis) or

indirectly (mixed cryoglobulinemia syndrome), to a co-existent rheumatic disease [rheumatoid arthritis (RA), systemic lupus erythematosus (SLE), Sjogren's syndrome] or, more rarely, it could be induced by the antiviral therapy (IFN-a) **(Buskila D, 2009).**

Autoimmune hepatitis is a chronic hepatitis of unknown etiology characterized by immunologic and autoimmunologic features, generally including the presence of circulating autoantibodies and a high serum globulin concentration **(Krawitt , 2006) .**

Circulating autoantibodies are often detected in patients with chronic HCV infection. Antinuclear antibodies (ANA), rheumatoid factor (RF), and anti-smooth muscle antibodies are the most frequently found, and other auto- antibodies (such as anti-dsDNA, anti-extractable nuclear antigens [anti-ENA], antimitochondrial antibodies [AMA], or anti-liver-kidney microsomes [anti-LKM-1]) are infrequent **(Casals et al., 2005).**