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Liver Function Tests: An Updated Review Article for Clinical Pathologists

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Abstract:

Background: The liver, a vital organ for detoxification, metabolism, and protein synthesis, requires precise evaluation to identify potential dysfunctions or injuries. Liver function tests (LFTs) assess hepatocellular damage, cholestasis, and synthetic capacity through a range of biomarkers like ALT, AST, ALP, GGT, and bilirubin.

Aim: This review provides a comprehensive overview of liver function tests, their interpretation, associated etiologies, and clinical implications for pathologists.

Methods: Data were collected from peer-reviewed studies focusing on LFTs, their diagnostic accuracy, and patterns of elevation. The review categorizes LFT abnormalities into hepatocellular, cholestatic, mixed injury, and isolated hyperbilirubinemia patterns while elucidating the pathophysiology and serological markers involved.

Results: Hepatocellular patterns predominantly indicate elevated aminotransferases linked to viral hepatitis, steatohepatitis, or autoimmune conditions. Cholestatic patterns, defined by increased ALP and GGT, suggest bile duct obstruction or hepatobiliary disorders. Mixed injury and isolated hyperbilirubinemia provide additional diagnostic insights. Advanced serological tests like AFP and CA19-9 aid in identifying malignancies, while synthetic function tests assess albumin production and prothrombin time. Specimen handling and storage play crucial roles in ensuring test accuracy.

Conclusion: Liver function tests remain indispensable in diagnosing hepatic dysfunctions. However, accurate interpretation requires an integrated understanding of biochemical patterns and clinical contexts. Advances in diagnostic tools and biomarker analysis further enhance the precision of hepatological assessments.

Keywords: liver function tests, hepatocellular injury, cholestasis, biomarkers, diagnostic pathology, ALT, AST, bilirubin, synthetic function tests, serological markers.

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Introduction:

The liver, situated in the right upper quadrant beneath the diaphragm, performs essential physiological functions, including detoxification of metabolites, protein synthesis, and the production of digestive enzymes [1]. Additionally, it plays a vital role in metabolism, the regulation of red blood cells (RBCs), and the synthesis and storage of glucose. Evaluation of liver function typically involves various tests, such as alanine aminotransferase (ALT), aspartate aminotransferase (AST), alkaline phosphatase (ALP), gamma-glutamyl transferase (GGT), 5'-nucleotidase, total bilirubin, conjugated (direct) bilirubin, unconjugated (indirect) bilirubin, prothrombin time (PT), the international normalized ratio (INR), lactate dehydrogenase, total protein, globulins, and albumin. These assessments aid in identifying hepatic injury, with specific elevation patterns providing insights into differential diagnoses [2]. Although commonly referred to as "liver function tests," this term is somewhat inaccurate since many of these tests do not directly measure liver functionality but rather indicate the site of hepatic damage. For instance, elevations in ALT and AST disproportionate to ALP and bilirubin suggest hepatocellular disease, whereas an increase in ALP and bilirubin with relatively normal ALT and AST levels indicates a cholestatic pattern. Mixed injury patterns involve elevations in both ALP and AST/ALT levels, while isolated hyperbilirubinemia is characterized by elevated bilirubin with normal ALP and AST/ALT levels [3] [4]. The liver's actual functional capacity can be assessed through its ability to produce albumin and vitamin K-dependent clotting factors.

Etiology and Epidemiology

Elevated liver function tests are observed in approximately 8% of the general population, with many elevations being transient and resolving spontaneously in asymptomatic patients. Studies indicate that up to 30% of these elevations normalize within three weeks, underscoring the need for careful interpretation to avoid unnecessary diagnostic procedures [5]. The classification of AST and/or ALT elevation ranges from borderline (less than 2 times the upper limit of normal, ULN) to massive elevations (exceeding 10,000 IU/L). The magnitude of elevation often correlates with the underlying cause of hepatocellular injury, with mild elevations (2 to 5 times ULN) and moderate elevations (5 to 15 times ULN) serving as common markers of liver dysfunction [6].

Differential Diagnosis of Elevated Liver Function Tests

Hepatocellular Pattern

Elevated aminotransferases that are disproportionately higher than alkaline phosphatase typically indicate a hepatocellular pattern of liver injury. When ALT predominates, potential causes include acute or chronic viral hepatitis, steatohepatitis, acute Budd-Chiari syndrome, ischemic hepatitis, autoimmune hepatitis, hemochromatosis, medication-induced injury, alpha1-antitrypsin deficiency, Wilson's disease, and celiac disease. Conversely, an AST-predominant pattern may be associated with alcohol-related liver injury, steatohepatitis, cirrhosis, or non-hepatic conditions such as hemolysis, myopathy, thyroid disorders, or strenuous exercise.

Cholestatic Pattern

A cholestatic pattern, defined by elevated alkaline phosphatase levels along with gamma-glutamyl transferase (GGT) and bilirubin, suggests bile duct obstruction or other hepatobiliary conditions. Hepatobiliary causes include primary biliary cirrhosis, primary sclerosing cholangitis, medication-induced liver injury, infiltrative liver diseases (e.g., sarcoidosis, amyloidosis, lymphoma), cystic fibrosis, hepatic metastasis, or cholestasis of pregnancy. Non-hepatic causes may involve bone disease, chronic renal failure, lymphoma or malignancies, congestive heart failure, growth phases in childhood, infections, or inflammation. This diagnostic framework aids clinicians in identifying the underlying etiology of liver test abnormalities and tailoring management strategies accordingly.

Pathophysiology

Hepatocellular Labs

Aminotransferases, encompassing AST and ALT, are fundamental markers of hepatocellular injury. These enzymes contribute to gluconeogenesis by catalyzing the transfer of amino groups from aspartic acid or alanine to ketoglutaric acid, resulting in the production of oxaloacetic acid and pyruvic acid, respectively. AST exists in both cytosolic and mitochondrial isoenzymes and is distributed across several tissues, including the liver, cardiac muscle, skeletal muscle, kidneys, brain, pancreas, lungs, leukocytes, and red cells. However, AST is less sensitive and specific to hepatic injury compared to ALT, and its elevation can arise from nonhepatic causes. Notably, AST activity in neonates and infants is approximately double that in adults, gradually decreasing to adult levels by about six months [7]. ALT, predominantly a cytosolic enzyme, is found in high concentrations in the liver. With a half-life of approximately 47 ± 10 hours, ALT is typically higher than AST in most liver diseases where enzyme activity primarily originates from the hepatocyte cytosol. Hepatocellular damage, even without cell death, triggers the release of these enzymes into circulation. Furthermore, ALT and AST levels tend to be higher in males than females and exhibit a positive correlation with body mass index (BMI), reflecting obesity-related changes [8][9].

Cholestasis Labs

Alkaline phosphatase (ALP), a zinc metalloenzyme, is concentrated in the microvilli of the bile canaliculus and various other tissues, including bone, intestines, and the placenta. The enzyme family comprises four isozymes: placental ALP (PLALP), germ cell ALP (GCALP), intestinal ALP (IALP), and tissue-nonspecific ALP (TNALP). PLALP and GCALP demonstrate the highest heat stability, while the bone component of TNALP is least stable. In healthy individuals, PLALP and GCALP constitute less than 1% of serum ALP activity [10]. Benign transient hyperphosphatasemia, characterized by significantly elevated plasma ALP, is a condition commonly observed in infants but can also affect adults and pregnant women. This benign state, often linked to concurrent infections, typically resolves within 6–8 weeks. Its biochemical signature includes variant isoenzymes detected through polyacrylamide gel electrophoresis [11].

Gamma-glutamyltransferase (GGT), a glycoprotein, is found on cell membranes with high secretory or absorptive activity. It catalyzes the transfer of gamma-glutamyl groups and is more specific for biliary disease compared to ALP since it is absent in bone. Elevated GGT levels, more pronounced than ALP in obstructive liver conditions, also correlate with bile duct damage in pediatric populations and certain idiopathic cholestasis cases. The enzyme levels remain high in infants with biliary atresia who are breastfed, as human breast milk contains significant GGT levels postpartum [12]. A relationship exists between plasma GGT levels and obesity, with values increasing by 50% in individuals with a BMI exceeding 30 kg/m^2. Elevated GGT levels also appear in steatosis, diabetes mellitus, non-alcoholic steatohepatitis, and conditions causing liver fibrosis or cirrhosis, including alcoholic cirrhosis, primary biliary cholangitis (PBC), and Wilson's disease. Moreover, space-occupying lesions, malignancies, and granulomatous conditions such as sarcoidosis and tuberculosis are associated with raised GGT levels. Similarly, 5′-nucleotidase (5′NT), localized in the canalicular and sinusoidal plasma membranes, serves as an indicator of hepatobiliary disease and differentiates ALP elevations from bone-related causes [13]. Bilirubin, derived from heme catabolism, primarily originates from hemoglobin breakdown. Unconjugated bilirubin, transported to the liver bound to albumin, is water-insoluble and cannot be excreted in urine. Upon conjugation in the liver, bilirubin becomes water-soluble, allowing its excretion through bile and urine [14].

Synthetic Function Tests

Albumin, synthesized by hepatic parenchymal cells, plays a critical role in maintaining colloidal osmotic pressure. Its synthesis rate depends on plasma albumin concentration, dietary protein intake, and feedback regulation. Despite liver disease, plasma albumin levels are maintained with only 10% of normal hepatocyte mass. However, reduced synthesis due to hepatic dysfunction or loss through malnutrition, nephrotic syndrome, or malabsorption results in low serum albumin levels [15][16]. Prothrombin time (PT) evaluates the liver's synthetic function by measuring the conversion rate of prothrombin to thrombin, a process dependent on coagulation factors synthesized by the liver. Delayed PT in the absence of liver dysfunction may indicate conditions such as warfarin therapy or vitamin K deficiency [17].

Serological Tests

Serological tests are crucial in diagnosing autoimmune liver diseases by identifying specific autoantibodies. In autoimmune hepatitis type 1 (AIH-1), the presence of anti-nuclear antibodies (ANA) and smooth muscle antibodies (SMA) is a key diagnostic marker. Conversely, autoimmune hepatitis type 2 (AIH-2) is characterized by anti-liver kidney microsomal type 1 (anti-LKM1) and anti-liver cytosol type 1 (anti-LC1) antibodies. The detection of antimitochondrial antibodies (AMA) serves as a hallmark for diagnosing primary biliary cirrhosis (PBC), a chronic progressive liver condition. Additionally, atypical perinuclear anti-neutrophil cytoplasmic antibodies (p-ANCA) are commonly linked to sclerosing cholangitis and its variants. These serological markers not only facilitate early and accurate diagnosis but also aid in distinguishing between overlapping liver conditions, enhancing clinical decision-making. By recognizing these distinct autoantibody patterns, clinicians can implement tailored therapeutic strategies and improve patient outcomes in managing autoimmune liver diseases [18][19].

Secondary Biochemical Liver Tests

Alpha-fetoprotein (AFP) serves as a crucial tumor marker for the detection and monitoring of primary hepatocellular malignancies, including hepatoblastoma and hepatocellular carcinoma (HCC). This elevation in alpha-fetoprotein is attributable to its production by hepatoblasts, particularly during liver regeneration, as observed in chronic viral hepatitis cases [20]. Carbohydrate-deficient transferrin is recognized for its high specificity in detecting excessive alcohol consumption, which can lead to liver damage. The carbohydrate antigen CA19-9, on the other hand, is instrumental in tracking the progression of primary sclerosing cholangitis (PSC), an autoimmune condition that frequently evolves into bile duct tumors or cholangiocarcinoma [21]. Additionally, serum ferritin measurements can aid in identifying hemochromatosis; however, as ferritin acts as a positive acute-phase reactant, its levels may rise in various illnesses and during acute hepatic failure due to hepatocyte damage [22].

Specimen Requirements and Procedure

The preferred specimen for liver tests is serum. Given the potential presence of infectious agents such as HIV or hepatitis B virus, all plasma or serum specimens must be handled with standard precautions. It is critical to send specimens to the laboratory for prompt processing. Separated serum or plasma should not remain at temperatures of +15°C to +30°C for more than 8 hours. For assays that cannot be completed within 8 hours, storage at +2°C to +8°C is recommended. If further delays are anticipated beyond 48 hours, samples should be frozen at −15°C to −20°C, ensuring that frozen samples are thawed only once to avoid analyte deterioration caused by repeated freeze-thaw cycles [23].

Testing Procedures

Liver function tests are performed using semi-automatic or fully automated analyzers, which operate based on photometric principles to measure analyte concentrations in liquid solutions. Photometry involves analyzing the absorption of light in the ultraviolet (UV), visible (VIS), and infrared (IR) spectrums. The analyzers incorporate specialized light sources and detectors, such as photodiodes, photoresistors, or photomultipliers, to detect transmitted light and convert it into electrical signals proportional to the analyte's concentration. These signals are processed according to Beer–Lambert's law, which establishes a direct relationship between the absorbance of light and the concentration of a substance in a sample. A test-specific calibration function ensures accurate and precise measurements by correlating the absorbance values with the analyte concentration. This advanced photometric approach provides reliable data essential for assessing liver function, aiding in diagnosing and monitoring liver diseases [24].

Interfering Factors

Several factors, including hemolysis, icterus, and lipemia, can compromise specimen integrity, leading to inaccurate laboratory test results and potentially erroneous clinical decisions. Hemoglobin's absorbance spectrum, peaking around 415 nm and spanning 320 to 580 nm, can interfere with colorimetric assays measuring within these ranges. This is particularly relevant for tests such as those measuring iron, lipase, albumin, and γ-glutamyl transferase. Similarly, icterus affects assays through spectrophotometric and chemical interferences, as bilirubin absorbs light between 400 and 540 nm, with distinct effects depending on whether the bilirubin is conjugated or unconjugated. Lipemia introduces challenges through light scattering, differential analyte partitioning between phases, and interactions with assay reagents, often affecting results for assays using shorter wavelengths. For example, assays measuring changes in NAD(P)H at 340 nm are particularly susceptible to lipemia interference. Moreover, certain medications, including metronidazole, may disrupt alanine aminotransferase (ALT) methods due to their absorbance near 340 nm. Variations in ALT levels have also been linked to diurnal rhythms, with fluctuations up to 45% observed between morning and afternoon, as well as factors such as body mass index (40–50% higher in individuals with higher BMI) and physical activity (20% lower in active individuals) [28]. Other influencing factors include dietary intake, which can transiently elevate ALT by up to 30 U/L, particularly in individuals with blood groups B and O, where increases may persist for up to 12 hours. Smoking has been associated with elevated placental-like alkaline phosphatase (PLALP) levels, which normalize within one to two months of cessation. Elevated alkaline phosphatase (ALP) levels are also seen during growth phases in children and adolescents due to heightened osteoblastic activity, as well as during the third trimester of pregnancy due to the placental isoenzyme. Any unexplained elevations in ALP should prompt a repeat fasting sample to confirm the findings. Drug-induced liver injury, whether predictable or idiosyncratic, remains a significant concern, with amoxicillin/clavulanate being the most commonly implicated agent, occurring at an incidence of 19 cases per 100,000 individuals [25][26][27].

Results, Reporting, and Critical Findings

The interpretation of liver function test (LFT) results should align with findings from a comprehensive patient history and physical examination. Essential aspects to consider include the patient's age and medical history, such as the presence of diabetes, obesity, hyperlipidemia, inflammatory bowel disease, celiac sprue, thyroid disorders, autoimmune hepatitis, acquired muscle disorders, alcohol use disorder, exposure to medications or toxins, and a familial history of genetic liver conditions like Wilson disease, alpha-1-antitrypsin deficiency, and hereditary hemochromatosis [30]. A systematic review of the patient's symptoms is vital to identify indications of chronic liver disease, which may manifest as jaundice, ascites, peripheral edema, hepatosplenomegaly, gynecomastia, testicular hypotrophy, muscle wasting, encephalopathy, pruritus, or gastrointestinal bleeding. Additional diagnostic tests that can aid in identifying the etiology of elevated transaminase levels include hepatitis panels, fasting lipid profiles, hemoglobin A1C levels, fasting glucose measurements, complete blood counts with platelet levels, comprehensive metabolic panels, iron studies, hepatitis C antibody, and hepatitis B surface antigen testing. Reference intervals for LFTs often vary among laboratories and can differ based on sex and body mass index. Consequently, it is essential to interpret a patient’s test results in the context of the specific reference values established by the laboratory conducting the tests. Laboratories are advised to determine their own reference ranges based on their methodologies.

- **Alanine transaminase (ALT):** 4 to 36 IU/L
- **Aspartate transaminase (AST):** 5 to 30 IU/L
- **Alkaline phosphatase (ALP):** 30 to 120 IU/L
- **Gamma-glutamyltransferase (GGT):** 6 to 50 IU/L
- **Bilirubin (total):** 2 to 17 μmol/L
- **Direct bilirubin:** 0 to 6 μmol/L
- **Prothrombin time:** 10.9 to 12.5 seconds
- **Albumin:** 35 to 50 g/L
- **Total protein:** 60 to 80 g/L
- **Lactate dehydrogenase (LDH):** 50 to 150 IU/L

Clinical Significance

The assessment of liver function test (LFT) results provides critical insights into differential diagnoses, as various pathological processes exhibit distinct patterns of liver enzyme abnormalities. Confirmation of abnormalities through repeated testing necessitates further investigation.

Alcohol

In individuals with alcohol use disorder, the aspartate aminotransferase (AST) to alanine aminotransferase (ALT) ratio often exceeds 2:1, serving as a biochemical hallmark of alcoholic liver disease. This elevation reflects increased AST activity relative to ALT, as AST is more abundantly released from mitochondrial damage caused by chronic alcohol consumption [31]. Additionally, gamma-glutamyl transferase (GGT) levels are frequently elevated in individuals with excessive alcohol intake. Elevated GGT, when observed alongside increased AST, can provide further evidence of alcohol misuse; however, GGT lacks specificity as a standalone marker for alcohol-induced liver damage, as its levels can also rise due to other liver and biliary disorders or the use of certain medications [32]. These parameters, used in conjunction with clinical history and other diagnostic findings, are essential in differentiating alcohol-related liver damage from other liver pathologies.

Medications

Numerous medications are known to induce hepatotoxicity. Commonly implicated drugs include NSAIDs, antibiotics, statins, anti-seizure medications, and treatments for tuberculosis. Acute hepatocellular injury can occur due to drugs such as acetaminophen, allopurinol, NSAIDs, alcohol, isoniazid, pyrazinamide, rifampin, statins, ketoconazole, tetracyclines, valproic acid, phenytoin, fluoxetine, risperidone, valacyclovir, and ritonavir [25]. Drugs such as anabolic steroids, tricyclic antidepressants, alcohol, azithromycin, amoxicillin, nafcillin, rifampin, and trimethoprim-sulfamethoxazole may cause acute cholestasis, while long-term use can lead to chronic hepatocellular or cholestatic damage [26]. Methotrexate, widely used for inflammatory arthritis, can cause both transient LFT elevation and permanent liver damage, such as fibrosis and cirrhosis, particularly at high cumulative doses. Chronic alcohol intake, methyldopa, and ergot alkaloids may also contribute to liver damage, with oral contraceptives potentially causing hepatic venous outflow obstruction (Budd-Chiari syndrome) [33]. Additionally, herbal remedies are increasingly recognized for their hepatotoxic potential [34].

Viral Hepatitis

Viral infections are significant contributors to hepatitis and elevated liver function tests (LFTs). Hepatitis B, C, and D are primarily associated with chronic hepatitis, often leading to long-term liver damage such as cirrhosis and liver cancer. These viruses directly affect liver cells, triggering inflammatory responses that result in sustained LFT elevation. Hepatitis A and E, on the other hand, generally cause acute forms of hepatitis that are resolved on their own in most cases, although they can still cause transient LFT elevations during the acute phase of infection [35]. In addition to the hepatitis viruses, other viral infections can also induce hepatitis and lead to abnormal LFTs. Human immunodeficiency virus (HIV) has been shown to cause liver dysfunction, especially in individuals with co-infections such as hepatitis B or C. Epstein-Barr virus (EBV) and cytomegalovirus (CMV) are other examples of viruses that can cause viral hepatitis, particularly in immunocompromised individuals. These infections can cause elevated LFTs as part of their clinical presentation, even though they are not typically associated with chronic liver disease [36]. Consequently, viral hepatitis requires careful diagnosis and monitoring, as LFT results alone may not distinguish between different types of viral infections, highlighting the importance of comprehensive clinical evaluation and appropriate serologic testing [36].

Autoimmune Hepatitis

Autoimmune hepatitis is a chronic inflammatory condition that often causes cirrhosis. It predominantly affects young women, with a female-to-male ratio of 4:1. Patients frequently present with elevated LFTs in the absence of an apparent cause [37]. Autoantibody positivity, including antinuclear antibodies, anti-smooth muscle antibodies, anti-liver/kidney microsomal antibodies, and antibodies against liver antigens, aids in diagnosis [38].

Hepatic Steatosis and Nonalcoholic Steatohepatitis

Nonalcoholic steatohepatitis, a form of fatty liver disease, has emerged as a significant cause of chronic liver disease and hepatocellular carcinoma. It commonly affects overweight individuals with type II diabetes or dyslipidemia who lack significant alcohol consumption [39]. The AST and ALT levels are typically elevated in a 1:1 ratio, with other LFT parameters remaining normal [40].

Hemochromatosis

Hemochromatosis involves excessive iron accumulation in parenchymal organs, resulting in organ toxicity. It is the most prevalent autosomal recessive disorder and a leading cause of severe iron overload. Symptoms include diabetes, liver dysfunction, and hyperpigmentation [41]. Elevated serum ferritin levels may suggest hemochromatosis, though transferrin saturation exceeding 45% provides greater diagnostic reliability. HFE mutations, such as C282Y and H63D, are essential for confirming hereditary hemochromatosis. Secondary forms may arise from excessive iron intake [42].

Wilson Disease

Wilson disease, a rare autosomal recessive disorder of copper metabolism, leads to copper deposition in the liver, brain, and other tissues, which can be fatal if untreated. A reduced serum ceruloplasmin level is observed in up to 85% of cases [43]. While Kayser-Fleischer rings may serve as a clinical clue, they are not universally present. Abnormal 24-hour urinary copper excretion exceeding 100 micrograms strongly supports the diagnosis, with liver biopsy serving as the definitive test [44].

Alpha-1 Antitrypsin Deficiency

Alpha-1 antitrypsin deficiency (AATD) is a relatively common yet underdiagnosed genetic disorder [45]. It predisposes individuals to obstructive pulmonary disease and liver conditions such as cirrhosis and hepatocellular carcinoma, affecting both children and adults. Early-onset panacinar emphysema is a hallmark feature, particularly among Caucasians [46].

Celiac Disease

Celiac disease, a gluten sensitivity disorder, is associated with mild elevations in liver transaminases. Patients with persistent LFT abnormalities should undergo screening, which includes testing for tissue transglutaminase IgA and serum IgA levels or tissue transglutaminase IgA and anti-deamidated gliadin peptide IgG [47][48].

Thyroid Disorders

Abnormal LFTs can also be linked to thyroid dysfunction, including hypothyroidism and hyperthyroidism, which may manifest as hepatocellular or cholestatic injury, particularly in severe cases such as myxedema or thyrotoxicosis. Screening involves assessing thyroid-stimulating hormone levels, with additional testing of free T4 and free/total T3 as required [49].

Quality Control and Laboratory Safety:

Regulations governing non-waived tests mandate laboratories to analyze at least two levels of control materials within a 24-hour period as a minimum standard. Laboratories may choose to perform quality control (QC) testing more frequently if necessary to ensure reliable results. QC samples should also be tested after calibrating or conducting maintenance on analyzers to confirm proper method performance. In situations where manufacturers recommend less frequent QC testing than regulatory requirements, such as once per month, laboratories may implement an Individualized Quality Control Plan (IQCP). This plan involves a comprehensive risk assessment to identify potential error sources across all testing phases and outlines a QC strategy to mitigate these risks. The Westgard multi-rules are often utilized to evaluate QC runs. If a violation of any rule occurs, laboratories must promptly undertake corrective and preventive actions before resuming patient testing [23].

Enhancing Healthcare Team Outcomes:

Liver function tests (LFTs) are among the most frequently ordered diagnostic tools in clinical practice. While mild, isolated elevations in LFTs may reflect normal biological variations and not warrant extensive or costly diagnostic evaluations, clinicians should remain vigilant regarding potential conditions that may cause abnormal LFT results. A detailed patient history and thorough physical examination are essential to narrowing down differential diagnoses. Particular attention should be given to the patient's drug and medication history, as this information is critical. The nursing team plays a pivotal role in medication reconciliation, while pharmacists can assist in identifying agents that may pose a risk of hepatotoxicity. In some cases, referral to a hepatologist or other specialists may be required. Adopting an interprofessional team approach ensures accurate diagnosis and appropriate management. This model of collaborative patient care, supported by meticulous record-keeping and open communication across healthcare disciplines, leads to improved clinical outcomes and enhanced patient care [50].

Conclusion:

Liver function tests (LFTs) serve as cornerstone diagnostic tools in assessing hepatic health. While commonly termed "function tests," many parameters measure cellular injury or enzyme activity rather than direct liver function. Hepatocellular injury, marked by elevated ALT and AST, is frequently linked to conditions like viral hepatitis, autoimmune diseases, and metabolic disorders. Cholestatic patterns involving ALP and GGT signify bile duct-related dysfunctions or hepatobiliary diseases. Mixed injury patterns and isolated hyperbilirubinemia provide critical diagnostic nuances. Synthetic function tests, including albumin and prothrombin time measurements, reflect the liver's capacity to maintain homeostasis. Additionally, advanced serological markers like AFP and CA19-9 expand diagnostic horizons, particularly for hepatocellular carcinoma and sclerosing cholangitis. However, effective interpretation necessitates considering patient history, clinical presentation, and the magnitude of biochemical deviations. Practical considerations, such as prompt specimen processing and optimal storage conditions, are integral to obtaining reliable results. The evolving landscape of diagnostic technology, incorporating automated systems and biomarker discovery promises enhanced precision in liver disease diagnosis. Despite their widespread utility, LFTs are not standalone indicators. Collaborative approaches, integrating imaging and clinical assessments, remain vital for comprehensive hepatological evaluations. Future research should focus on refining diagnostic criteria, identifying novel biomarkers, and improving predictive models for liver dysfunction. Such advancements will undoubtedly fortify the clinical pathologist's ability to deliver timely and accurate diagnoses, ultimately improving patient outcomes in hepatic care.

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اختبارات وظائف الكبد: مقال مراجعة محدث لعلماء الأمراض السريرية

الملخص:

الخلفية :الكبد، العضو الحيوي للتخلص من السموم، الأيض، وتخليق البروتينات، يحتاج إلى تقييم دقيق لتحديد أي اختلالات أو إصابات محتملة. تقيم اختبارات وظائف الكبد (LFTs) تلف الخلايا الكبدية، الركود الصفراوي، والقدرة التخليقية من خلال مجموعة من المؤشرات الحيوية مثل ALT و ASTو ALPو GGT والبيليروبين.

الهدف :تقدم هذه المراجعة نظرة شاملة لاختبارات وظائف الكبد، وتفسيرها، والأسباب المرتبطة بها، والآثار السريرية لعلماء الأمراض.

الطرق :تم جمع البيانات من دراسات محكمة تركز على اختبارات وظائف الكبد، دقتها التشخيصية، وأنماط الارتفاع. تصنف المراجعة الشذوذات في اختبارات وظائف الكبد إلى أنماط تلف الخلايا الكبدية، الركود الصفراوي، الإصابة المختلطة، وفرط البيليروبينيميا المعزول، مع توضيح الفيزيولوجيا المرضية والعلامات المصلية المعنية.

النتائج :تشير أنماط تلف الخلايا الكبدية بشكل رئيسي إلى ارتفاع الأميونترانسفيراز المرتبط بالتهاب الكبد الفيروسي، التهاب الكبد الدهني، أو الحالات المناعية الذاتية. تشير أنماط الركود الصفراوي، التي تتميز بزيادة ALP وGGT ، إلى انسداد القنوات الصفراوية أو اضطرابات الكبد والمرارة. تقدم الإصابة المختلطة وفرط البيليروبينيميا المعزول رؤى تشخيصية إضافية. تساعد الاختبارات المصلية المتقدمة مثل AFP و CA19-9 في التعرف على الأورام، بينما تقيم اختبارات الوظائف التخليقية إنتاج الألبومين ووقت البروثرومبين. تلعب معالجة العينات وتخزينها دورًا حاسمًا في ضمان دقة الاختبار.

الخلاصة :تظل اختبارات وظائف الكبد أمرًا لا غنى عنه في تشخيص اضطرابات الكبد. ومع ذلك، يتطلب التفسير الدقيق فهماً متكاملاً للأنماط الكيميائية الحيوية والسياقات السريرية. تعزز التقدمات في الأدوات التشخيصية وتحليل المؤشرات الحيوية دقة التقييمات الكبدية.

الكلمات المفتاحية :اختبارات وظائف الكبد، تلف الخلايا الكبدية، الركود الصفراوي، المؤشرات الحيوية، علم الأمراض التشخيصي، ALT، AST، البيليروبين، اختبارات الوظائف التخليقية، العلامات المصلية.