Research & Reviews: Journal of Medical and Health Sciences

e-ISSN:2319-9865
p-ISSN:2322-0104

HIV Infections with Hepatitis

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Review Article

Received: 30/08/2016
Accepted: 25/08/2016
Published: 13/09/2016

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Keywords: Antiretroviral treatment, Hepatitis C, T lymphocyte, Virologic leap

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ABSTRACT

Exceedingly dynamic antiretroviral treatment (HAART) for the Human Immunodeficiency Virus (HIV) contaminated patient has introduced a period of declining deft diseases and prompted another attention on other driving reasons for horribleness, for example, end-stage liver illness (ESLD) optional to hepatitis B infection (HBV) and hepatitis C infection (HCV) disease. We will survey the collaborations amongst HIV and HBV and HCV co-diseases and in addition their study of disease transmission, pathogenesis, research facility assessment and late upgrades in treatment alternatives.

HEPATITIS B CO-INFECTION

HBV does not change the course of HIV sickness, but rather HIV alters the course of HBV [1,2]. Patients with interminable HBV contamination were 3.5 times more inclined to have liver illness than those with no HBV disease (P<0.02) [3]. HIV tainted people are more averse to clear intense HBV contamination suddenly and face a higher danger of liver-related demise than the individuals who are contaminated with just HIV [4]. The nearness of ceaseless HBV can likewise prompt an expanded danger of hepatotoxicity identified with the organization of HAART. People co-tainted with HIV and HBV, particularly those with low bunch of separation 4 (CD4) T lymphocyte nadir checks, are at expanded danger for liver-related mortality [5]. Administration of HBV in co-tainted patients is muddled by the distinctions in normal history, as well as, by the movement of a considerable lot of the medications dynamic against both infections and by the improvement of medication safe HIV and HBV variations.

The Study of Disease Transmission

HBV is a deoxyribonucleic corrosive (DNA) infection. In the United States, the predominance of endless carriage of hepatitis B surface antigen (HBsAg) is available in under 1% of the populace [6,7]. In HIV contaminated people, this predominance is roughly 10-20 folds higher [8,9]. In a US study inspecting 16,248 HIV-tainted patients, the commonness of ceaseless HBV was found in 8% of unvaccinated members [10]. A higher commonness was found in non-Hispanic men particularly in men who engage in sexual relations with men (MSM) and in the individuals who were matured 35-44 [9]. In the United States and Western Europe, HBV is commonly obtained amid sexual action in youthfulness or early adulthood. Then again, in Asia and sub-Saharan Africa, perinatal transmission is more basic with constant HBV disease present in 90% of uncovered newborn children [11]. HBV contamination in HIV tainted patients builds the danger of cirrhosis, ESLD and passing from liver illness, particularly in patients with a low CD4 cell check or attending liquor use [5].

Liver malady identified with contamination with HBV is a continuous reason for dismalness and mortality in those tainted with HIV. HIV and HBV contaminations offer comparable transmission examples and danger variables, for example, intravenous medication clients (IVDU) and sexually transmitted ailments (STD). In created nations, research center markers of earlier HBV disease are more normal in MSM and IVDU [10,12,13].

e-ISSN:2319-9865
p-ISSN:2322-0104

HBV Genotypes

Eight HBV genotypes have been distinguished as A through H [14,15]. The commonness of each HBV genotype relies on upon the topographical area. All known HBV genotypes have been found in the United States with the accompanying commonness:A (35%), B (22%), C (31%), D (10%) and E-G (2%) [16]. While HBV genotype An is the most widely recognized in co-tainted people, the non-A genotypes were connected with more propelled fibrosis [17]. Moreover, Genotypes B and C were connected with higher viral burdens than were sorts An and D [18].

PATHOGENESIS

There are numerous potential explanations behind expanded liver-related mortality in HIV and HBV co-contaminated people. In HIV contaminated people, a quickly dynamic type of liver sickness called fibrosing cholestatic hepatitis is seen and thought to be expected to a viral cytopathic impact [19]. Revill reported a novel - 1G transformation which was recognized in the HBV pre-center and covering center qualities. This transformation truncates the pre-center/center proteins. The mutant genome was the prevailing species in some HIV/HBV co-contaminated people and was more pervasive in HIV/HBV co-tainted people than HBV monoinfected people. The change was connected with higher HBV DNA fixations in HIV/HBV co-contaminated people. Changes in the HBV center and pre-center qualities might add to sickness pathogenesis in HIV/HBV co-contaminated people [20]. Preiss reported deficient HBV DNA (dDNA) that is converse translated from joined HBV pre-genomic errand person RNA (pgRNA) and has been distinguished in patients with interminable HBV. The major 2.2-kb graft pgRNA encoded a novel HBV quality item, the hepatitis B join protein (HBSP), by means of a cancellation and casing shift inside the polymerase. In spite of the fact that grafted RNA and HBSP expression have been connected with expanded HBV DNA levels and liver fibrosis, the part of dDNA in HBV related sickness is to a great extent unclear. Deficient DNA was distinguished in 90% of people with unending HBV. There was no huge contrast in the relative wealth of dDNA between the mono-tainted and HIV/HBV coinfected bunches [21].

Safe actuation because of expanded HIV incited microbial translocation might be a system for quickening liver malady. Coursing lipopolysaccharide (a marker of microbial translocation) was essentially expanded in incessantly HIV-contaminated people and in simian immunodeficiency infection (SIV) tainted rhesus macaques (P= 0.002). Compelling antiretroviral treatment appeared to decrease microbial translocation [22].

HAART impelled resistant rebuilding may switch the insusceptible response to HBV from a tolerant to a narrow minded stage, prompting either the complete control of HBV replication or all the more regularly to a compounding of unending hepatitis. Patients who suddenly recoup from HBV contamination mount overwhelming CD4 and bunch of separation 8 (CD8) T-cell reactions to different HBV epitopes [10,23]. Lascar reported reconstitution of HBV particular T cell reactions in HIV contaminated patients after a decrease in their HBV load. This possibility to recoup T cell reactions, which is basic for HBV control, gives backing to the expansion of hostile to HBV treatment in the treatment of HIV/HBV co-tainted patients [24].

Lab Assessment

Hepatitis B surface Antigen (HBsAg0) is the serologic sign of HBV disease and shows up in serum one to ten weeks after intense introduction to HBV. This is trailed by the recognition of IgM and afterward IgG hostile to Hepatitis B center immunizer (against HBc). Most grown-ups clear HBsAg and create hostile to Hepatitis B surface neutralizer (against HBs) reliable with insusceptibility. Tirelessness of HBsAg for over six months suggests ceaseless disease and is connected with the nearness of HBV DNA viremia.

Ceaseless hepatitis B ought to be assessed further by requesting hepatitis B e antigen (HBeAg) (which is the extracellular type of the hepatitis B center Antigen (HBcAg)) and hepatitis B e counter acting agent (against HBe). The HBeAg seropositive state demonstrates more grounded viral infectivity. Hepatitis D (HDV) is a little roundabout encompassed ribonucleic corrosive (RNA) infection that requires the nearness of HBV for replication. The nearness of HDV is connected with more intricacies and a higher mortality. HDV antibodies ought to be checked particularly in patients from Eastern Europe, the Mediterranean, and the Amazon bowl who have higher rates of this HBV DNA (measured in duplicates/ml or IU/ml) is valuable in observing remedial adequacy. Presently there is no particular rule for ultrasound (US) and alpha-fetoprotein (AFP) recurrence in HIV/HBV co-contaminated people. The American Association for the Study of Liver Diseases (AASLD) Practice Guideline in 2009 prescribed observation of HBV transporters at high danger of hepatocellular carcinoma (HCC) with US each 6-12 months and AFP alone when US is not accessible or expense is an issue [25]. In any case, AFP determination needs sufficient affectability and specificity for successful observation [26] thus reconnaissance with US at regular intervals is favored [27].

e-ISSN:2319-9865
p-ISSN:2322-0104

Constant HBV action is derived by rises of aminotransferases and by the nearness of viremia. The nearness of HBeAg shows the level of infectivity as well as is typically connected with more elevated amounts of HBV DNA and dynamic liver illness. Seroconversion from HBeAg to against HBe positive is generally connected with decreases in serum HBV DNA and abatement of liver malady. A few patients keep on having dynamic liver sickness and large amounts of HBV DNA in serum even after the loss of (HBeAg negative interminable hepatitis). These last patients may have HBV variations that cancel or abatement the generation of perceptible HBeAg in serum. HIV contaminated patients can have elevated amounts of HBV DNA with hepatic corruption and irritation when they are hostile to HBc positive yet HBsAg negative [28].

TREATMENT

There are two incessant HBV treatment modalities in particular interferon (IFN) and nucleoside reverse transcriptase inhibitors (NRTI). The last is the standard treatment for HIV/HBV co-disease. The double antiviral movement of NRTI requires cautious coordination and determination keeping in mind the end goal to evade choice of resistance transformations and harmfulness. Patients who need treatment for HBV contamination yet not HIV disease ought not to get HBV solutions that have movement against HIV. Rather, they ought to get specialists with HBV action alone.

HBV Treatment Start in HIV Patients

Treatment of HBV in HIV contaminated patients is like patients with HBV disease alone. In the patient with endless HBV disease, viral replication and/or evaluation of liver histopathology are useful in deciding the requirement for antiviral treatment. There is insufficient information in HIV/HBV co-disease to decide the suitable cut off worth for HBV DNA levels for treatment start however numerous specialists prescribe an edge >2000 IU/ml (>10,000 duplicates/ml), as is suggested in patients with HBV alone [29-36].

In HIV/HBV co-tainted, if HBe Ag is negative, HBV DNA<2,000 IU/ml with an ordinary alanine aminotransferase (ALT), then consider observing like clockwork. On the off chance that HBV DNA ≥ 2,000 IU/ml with an ordinary ALT, consider liver biopsy and if fibrosis is available then consider treatment. On the off chance that HBV DNA ≥ 2,000 IU/ml with an anomalous ALT, consider treatment [25,29].

In HIV/HBV co-contaminated patients, lamuvidine (3TC) and tenofovir (TDF) in blend with a third antiretroviral is the highest quality level for treating both diseases together. Emtricitabine (FTC), which is utilized as a part of blend with TDF (Truvada, brand name), has been connected with hepatitis flares and is not FDA endorsed for HBV treatment. Be that as it may, numerous clinicians support utilizing this on account of the movement against both HIV and HBV in one tablet. Right now, monotherapy with a nucleos(t)ide simple or PEG IFN is prescribed as the underlying treatment in solo tainted HBV patients. On the off chance that lone treating HBV however not HIV in a co-contaminated patient, Pegylated Interferon (PEG IFN), adefovir (ADV) or telbuvidine (LdT) might be utilized as monotherapy yet ADV has frail antiviral action [30] and LdT, similar to 3TC, has high rates of medication resistance. Imperviousness to 3TC is very much perceived in HIV strains and is encoded inside the tyrosine-methionine-aspartate-aspartate (YMDD) theme close to the reactant site of opposite transcriptase [37]. HBV imperviousness to 3TC was accounted for to be half following 2 years and 90% following 4 years of treatment in a review companion investigation of HIV/HBV co-contaminated people [38,39]. Against HBe seroconversion and HBeAg seronegativity were seen in 11% and 18.5% of cases, individually in 3TC treated patients. Variables connected with an expansion rate of 3TC resistance incorporate long treatment span, high pre-treatment serum HBV DNA levels and high leftover infection levels after start of treatment [34,35]. ADV is the main widely considered remedial option for 3TC safe HBV contamination in HIV/HBV co-disease. In vitro and clinical studies demonstrated that ADV is compelling in stifling wild sort and in addition 3TC safe HBV [40]. LdT is more intense than 3TC in lessening the HBV DNA levels following one year of treatment [41]. There were a few concerns in regards to action of LdT with HIV yet a late study demonstrated that LdT has no movement against HIV in vitro [42].

Entecavir (ETV), TDF, 3TC and FTC ought not be utilized as monotherapy for HBV as a part of a contaminated HIV quiet since they have against HIV action and can prompt quick HIV resistance. ETV is a strong antiviral treatment for the treatment of HBV disease. At first, it was trusted that this operator did not have action against HIV disease. In any case, resulting clinical and in vitro information demonstrated that utilization of ETV in a HIV contaminated patient with recognizable HIV RNA can prompt a M184V change which presents drug imperviousness to 3TC and FTC [43-50]. ETV ought to just be utilized to treat HBV in the HIV contaminated patient who has achieved complete viral concealment. ETV has not been assessed in patients with HIV and HBV co-disease who are not accepting successful treatment for HIV in the meantime [51-58]. TDF is a nucleotide reverse transcriptase inhibitor and it has been appeared to have intense in vitro movement against both wild sort and 3TC safe HBV [46-50]. The Department

e-ISSN:2319-9865
p-ISSN:2322-0104

of Health and Human Services (DHHS) rules propose consideration of TDF in addition to either 3TC or FTC in the antiretroviral regimens of patients co-contaminated with HIV and HBV who require treatment for their HIV disease [59-63]. FTC (despite the fact that not FDA affirmed for HBV) is an intense inhibitor of both HIV and HBV replication and has a more drawn out serum half-life than 3TC.

PEG IFN is not prescribed for patients with hepatic decompensation, immunosuppression, therapeutic or psychiatric contraindications. PEG IFN might be utilized as a part of remunerated cirrhotic patients who have typical manufactured capacity and no confirmation of entry hypertension [64-73]. Nucleos(t)ide analogs might be utilized as a part of patients with decompensated liver illness, contraindications to PEG IFN, capacity to resolve to long terms of treatment and in patients with low CD4 cell tallies (<200 cells/µl). ETV and TDF have the best security, adequacy and medication resistance profiles. TDF is favored in patients thinking about pregnancy and in patient already presented to 3TC or LdT. ETV is favored in patients with renal deficiency [74-88].

Administration of HBV Antiviral Medication Resistance

Development of antiviral medication resistance is showed by virologic leap forward, characterized as an expansion in serum HBV DNA levels of >1 log10 copies/ml from nadiror the discovery of HBV DNA after it had been imperceptible. Virologic achievement might be trailed by biochemical leap forward, characterized as expanded ALT levels in a patient who already had standardized ALT levels. It might likewise be seen in HBV flares and hepatic decompensation if rescue treatment is not started speedily. Virologic leap forward amid nucleos(t)ide simple treatment might be a consequence of antiviral medication resistance or solution nonadherence or discontinuance. Patients with virologic achievement ought to be guided in regards to medicine adherence and leap forward affirmed by retesting for serum HBV DNA levels following 1–3 months. Rescue treatment ought to be started promptly in patients who have decompensated liver infection or serious hepatitis flares, however in different patients, it can be conceded until after virologic achievement is affirmed to maintain a strategic distance from pointless changes in prescriptions [37,89-105]. It is vital to start treatment with a medication or medications that have a high hereditary obstruction to resistance (a low potential for medication resistance), in light of the fact that successive monotherapy may bring about the determination of different medication resistance transformations. All over again blend treatment may keep the development of numerous medication safe mutants [58].

CONCLUSION

HIV co-tainted HBV and HCV patients present incredible difficulties in administration for the clinician. Information of the intricate connections of these infections and in addition the impact of different treatment modalities on every infection is vital to comprehension and treating these patients viably. New research center tests and restorative alternatives make this an energizing time to watch over these patients and offers incredible any desire for a cure.

REFERENCES

1. Cooper CL, et al. CPG 7909 adjuvant improves hepatitis B virus vaccine seroprotection in antiretroviral-treated HIV-infected adults. AIDS. 2015;19:1473-1479.
2. Chinnadurai R, et al. Hepatic Transplant and HCV:A New Playground for an Old Virus. Am J Transplant. 2011;12:298-305.
3. McGovern BH. Hepatitis C in the HIV-infected patient. J Acquir Immune Defic Syndr. 2014;45:S47-56.
4. Hernandez MD and Sherman KE. HIV/hepatitis C coinfection natural history and disease progression. Curr Opin HIV AIDS. 2007;6:478-482.
5. Bica I, et al. Increasing mortality due to end-stage liver disease in patients with human immunodeficiency virus infection. Clin Infect Dis. 2011;32:492-497.
6. Weber R, et al. Liver-related deaths in persons infected with the human immunodeficiency virus:the D:A:D study. Arch Intern Med. 2006;166:1632-1641.
7. Thomas DL, et al.Effect of human immunodeficiency virus on hepatitis C virus infection among injecting drug users. J Infect Dis.1996;174:690-695.
8. Daar ES, et al. Relation between HIV-1 and hepatitis C viral load in patients with hemophilia. J Acquir Immune Defic Syndr. 2011;26:466-472.
9. Ghany MG, et la. Effect of human immunodeficiency virus infection on hepatitis C virus infection in hemophiliacs. Dig Dis Sci. 1996;41:1265-1272.

e-ISSN:2319-9865
p-ISSN:2322-0104

10. Cooper CL and Cameron DW. Effect of alcohol use and highly active antiretroviral therapy on plasma levels of hepatitis C virus (HCV) in patients coinfected with HIV and HCV. Clin Infect Dis. 2005;41:S105-S109.
11. Lin W, et al. HIV increases HCV replication in a TGF-beta1-dependent manner. Gastroenterology. 2008;134:803-811.
12. Rizza SA, et al. TRAIL dependent fratricidal killing of gp120 primed hepatocytes by hcv core expressing hepatocytes. PLoS One. 2011;6:e27171.
13. Danta M, et al. Impact of HIV on host-virus interactions during early hepatitis C virus infection. J Infect Dis. 2008;197:1558-1566.
14. Kim AY, et al. Impaired hepatitis C virus-specific T cell responses and recurrent hepatitis C virus in HIV coinfection. PLoS Med. 2006;3:e492.
15. Benhamou Y, et al. Liver fibrosis progression in human immunodeficiency virus and hepatitis C virus coinfected patients. The Multivirc Group. Hepatology. 1999;30:1054-1058.
16. Monga HK, et al. Hepatitis C virus infection-related morbidity and mortality among patients with human immunodeficiency virus infection. Clin Infect Dis. 2011;33:240-247.
17. Martinez-Sierra C, et al. Progression of chronic hepatitis C to liver fibrosis and cirrhosis in patients co-infected with hepatitis C virus and human immunodeficiency virus. Clin Infect Dis. 2003;36:491-498.
18. Goutagny N, et al. Evidence of viral replication in circulating dendritic cells during hepatitis C virus infection. J Infect Dis. 2003;187:1951-1958.
19. Blackard JT, et al. Extrahepatic replication of HCV: Insights into clinical manifestations and biological consequences. Hepatology. 2006;44:15-22.
20. Lanford RE, et al. Lack of detection of negative-strand hepatitis C virus RNA in peripheral blood mononuclear cells and other extrahepatic tissues by the highly strand-specific rTth reverse transcriptase PCR. J Virol. 1995;69:8079-8083.
21. Blackard JT, et al. Detection of hepatitis C virus (HCV) in serum and peripheral-blood mononuclear cells from HCV-monoinfected and HIV/HCV-coinfected persons. J Infect Dis. 2005;192:258-265.
22. Laskus T, et al. Hepatitis C virus in lymphoid cells of patients coinfected with human immunodeficiency virus type 1: evidence of active replication in monocytes/macrophages and lymphocytes. J Infect Dis. 2000;181:442-448.
23. Minosse C, et al. Possible compartmentalization of hepatitis C viral replication in the genital tract of HIV-1-coinfected women. J Infect Dis. 2066;194:1529-1536.
24. Nowicki MJ, et al. Presence of hepatitis C virus (HCV) RNA in the genital tracts of HCV/HIV-1-coinfected women. J Infect Dis. 2005;192:1557-1565.
25. Sulkowski MS, et al. Hepatitis C and progression of HIV disease. JAMA. 2012;288:199-206.
26. Sulkowski MS, et al. Rapid fibrosis progression among HIV/hepatitis C virus-co-infected adults. AIDS. 2007;21:2209-2216.
27. Shafran SD. Early initiation of antiretroviral therapy:the current best way to reduce liver-related deaths in HIV/hepatitis C virus-coinfected patients. J Acquir Immune Defic Syndr. 2007;44:551-556.
28. Puoti M, et al. Hepatocellular carcinoma in HIV-infected patients:epidemiological features, clinical presentation and outcome. AIDS. 2004;18:2285-2293.
29. Strader DB, et al. Diagnosis, management, and treatment of hepatitis C. Hepatology. 2004;39:1147-1171.
30. Ojo BA, et al. Assessment of blood cd4 count and antibiogram profile of bacteria isolated from hiv patients. HIV Curr Res. 2016;1:107.
31. Shingdang J. Serum albumin/globulin ratio in tuberculosis and hiv patients any relationship? Mycobact Dis. 2016;6:199.
32. Plumelle Y, et al. Eosinophils in hiv patients co-infected by htlv-1 and/or strongyloïdes stercoralis:protective or harmful depending on hiv infection stage. J AIDS Clin Res. 2015;6:507.
33. Isaac A. Antiretroviral therapy, CD4, viral load, and disease stage in saudi hiv patients:a cross sectional study of cases diagnosed between 2001-2013. J AIDS Clin Res. 2015;6:447.
34. Motilewa OO, et al. A comparative study of health related-quality of life among hiv patients on pre-HAART and HAART in Uyo South-South Nigeria. J Antivir Antiretrovir. 2015;7:060-068.
35. Patrikar S, et al. Profile of hiv patients on second line antiretroviral therapy: the indian experience. J AIDS Clin Res. 2015;6:459.

e-ISSN:2319-9865
p-ISSN:2322-0104

36. Mohammed M, et al. Prevalence of depression and associated factors among hiv patients seeking treatments in ART Clinics at Harar Town, Eastern Ethiopia. J AIDS Clin Res. 2015;6:474.
37. Holland JJ, et al. RNA virus populations as quasispecies. Curr Top Microbiol Immunol. 1992;176:1-20.
38. Davis GL. Hepatitis C virus genotypes and quasispecies. Am J Med. 1999;107:21S-26S.
39. Sherman KE, et al. Hepatitis C in human immunodeficiency virus-coinfected patients:increased variability in the hypervariable envelope coding domain. Hepatology. 1996;23:688-694.
40. Toyoda H, et al. Quasispecies nature of hepatitis C virus and response to alpha interferon:significance as a predictor of direct response to interferon. J Hepatol. 1997;26:6-13.
41. Sherman KE, et al. Hepatitis C virus (HCV) quasispecies complexity and selection in HCV/HIV-coinfected subjects treated with interferon-based regimens. J Infect Dis. 2010;201:712-719.
42. Blackard JT, et al. Compartmentalization of hepatitis C virus (HCV) during HCV/HIV coinfection. J Infect Dis. 2007;195:1765-1773.
43. McGovern BH, et al. Avoiding therapeutic pitfalls:the rational use of specifically targeted agents against hepatitis C infection. Hepatology. 2008;48:1700-1712.
44. Kuntzen T, et al. Naturally occurring dominant resistance mutations to hepatitis C virus protease and polymerase inhibitors in treatment-naive patients. Hepatology. 2008;48:1769-1778.
45. Gaudieri S, et al. Hepatitis C virus drug resistance and immune-driven adaptations:relevance to new antiviral therapy. Hepatology. 2009;49:1069-1082.
46. Ghany MG, et al. Diagnosis, management, and treatment of hepatitis C:an update. Hepatology. 2009;49:1335-1374.
47. Staples CT Jr, et al. Hepatitis C in the HIV (human immunodeficiency virus) Atlanta V.A. (Veterans Affairs Medical Center) Cohort Study (HAVACS): the effect of coinfection on survival. Clin Infect Dis. 1999;29:150-154.
48. Sherman KE, et al. Hepatitis C Virus prevalence among patients infected with Human Immunodeficiency Virus:a cross-sectional analysis of the US adult AIDS Clinical Trials Group. Clin Infect Dis. 2002;34:831-837.
49. Garfein RS, et al. Viral infections in short-term injection drug users:the prevalence of the hepatitis C, hepatitis B, human immunodeficiency, and human T-lymphotropic viruses. Am J Public Health. 1996;86:655-661.
50. Alter MJ, et al. Risk factors for acute non-A, non-B hepatitis in the United States and association with hepatitis C virus infection. JAMA. 1990;264:2231-2235.
51. Thomas DL, et al. Sexual transmission of hepatitis C virus among patients attending sexually transmitted diseases clinics in Baltimore--an analysis of 309 sex partnerships. J Infect Dis. 1995;171:768-775.
52. Quinn TC, et al. Viral load and heterosexual transmission of human immunodeficiency virus type 1. Rakai Project Study Group. N Engl J Med. 2000;342:921-929.
53. Osmond DH, et al. Risk factors for hepatitis C virus seropositivity in heterosexual couples. JAMA. 1993;269:361-365.
54. Urbanus AT, et al. Hepatitis C virus infections among HIV-infected men who have sex with men:an expanding epidemic. AIDS. 2009;23:F1-F7.
55. Danta M, et al. Recent epidemic of acute hepatitis C virus in HIV-positive men who have sex with men linked to high-risk sexual behaviours. AIDS. 2007;21:983-991.
56. Gotz HM, et al. A cluster of acute hepatitis C virus infection among men who have sex with men--results from contact tracing and public health implications. AIDS. 2005;19:969-974.
57. van de Laar TJ, et al. Increase in HCV incidence among men who have sex with men in Amsterdam most likely caused by sexual transmission. J Infect Dis. 2007;196:230-238.
58. Polis CB, et al. Impact of maternal HIV coinfection on the vertical transmission of hepatitis C virus:a meta-analysis. Clin Infect Dis. 2007;44:1123-1131.
59. Rivera-Rivera Y, et al. Impact of Depression and Inflammation on the Progression of HIV Disease. J Clin Cell Immunol. 2016;7:423.
60. Grifoni A and Amicosante M. Importance of HLA Class I Amino Acid Position 194 and LILRB1 Receptor in HIV Disease Progression. Immunogenet open access. 2016;1:e103.
61. Bodor J, et al. Hematopoietic Stem Cell-Based Therapy for HIV Disease:A Role for Regulatory T Cells. J Clin Cell Immunol. 2015;6:300.
62. Reeves RK. Mechanisms, consequences, and treatment of chronic inflammation in HIV Disease. J Antivir Antiretrovir. 2014;6:e117.

e-ISSN:2319-9865
p-ISSN:2322-0104

63. Ramana KV. HIV Disease Management in the Highly Active Antiretroviral Therapy (HAART) Era. J Medical Microbiol Diagnosis. 2012;1:e101.
64. Nole T, et al. Ethnomedical and ethnopharmacological study of plants used for potential treatments of diabetes and arterial hypertension by indigenous people in three phytogeographic regions of Cameroon. Diabetes Case Rep. 2016;1:110.
65. Kabore T and Lazar J. Prevalence and risk factors for pre-hypertension among adults in burkina faso, West Africa. J Comm Pub Health Nurs. 2016;2:130.
66. Chalupova L, et al. CTRP1: A molecular link between obesity and hypertension . J Mol Biomark Diagn. 2016;7:289.
67. Li M, et al. To Live Long, Eat Less Salt: Salt intake reduction promotion and hypertension control in China. Health Care:Current Reviews. 2016;4:169.
68. Trailokya A. Will Azilsartan - An eight arb bring paradigm shift in hypertension management practices in India? Cardiovasc Pharm Open Access. 2016;5:189.
69. Stoicescu M. The risk of sudden decrease of severe arterial hypertension. J Clin Exp Cardiolog. 2016;7:460.
70. Lv Y, et al. Non-hypersplenism causes of peripheral cytopenias in patients with cirrhotic portal hypertension:a review. J Hypertens. 2016;5:223.
71. Manolis A and Doumas M. Erectile function in cardiovascular disease and hypertension:the role of nebivolol. J Hypertens. 2016;5:226.
72. Berezin AE. Is elevated circulating galectin-3 level a predictor of pulmonary artery hypertension development and progression? Clin Med Biochemistry Open Access. 2016;2:114.
73. Skride A, et al. Pulmonary arterial hypertension associated with adult congenital heart disease, when inoperable becomes operable:a case report. J Pulm Respir Med. 2016;6:350.
74. Pourafshar N, et al. Need for raising awareness - monitoring urinary protein excretion in cardiac transplant patients receiving mTOR Inhibitors. J Nephrol Ther. 2016;6:254.
75. Mukherjee T. The Heart and the Kidneys:a Close Link. J Nephrol Ther. 2016;6:255.
76. Abu-Odah H, et al. End-stage renal disease in the gaza strip and its relationship to risk factors. J Nephrol Ther. 2016;6:248.
77. Lipe RA, et al. Cost/effectiveness treatment of acute renal failure secondary to multiple myeloma with filters high cut off. J Nephrol Ther. 2016;6:249.
78. Fouda H, et al. The epidemiology of acute kidney injury in a tertiary hospital in Cameroon: A 13 months review. J Nephrol Ther. 2016;6:250.
79. Laidoudi A, et al. Tubulointerstitial Nephritis and Uveitis (TINU) Syndrome, Two new cases and review of the literature. J Nephrol Ther. 2016;6:252.
80. Farahani ZB, et al. Iranian living related donors:motivation for kidney donation. J Nephrol Ther. 2016;6:253.
81. Galle J. Continuous Erythropoietin Receptor Activator (C.E.R.A.) Treatment of Renal Anemia in Patients with Chronic Kidney Disease:A Two-Year Observational Study. J Nephrol Ther. 2015;6:228.
82. Narain U and Gupta A. Pauci antibody anti-gbm nephritis:a case report. J Nephrol Ther. 2015;6:230.
83. Kamal I, et al. Kidneys:The Victim Of Hypertension:Review. J Nephrol Ther. 2015;6:231.
84. Kawakami E, et al. Kidneys:Application of meditation techniques in caregivers of children and adolescents with chronic kidney disease on hemodialysis therapy. J Nephrol Ther. 2016;6:232.
85. Kulkarni M. Cardio Renal Syndrome. J Nephrol Ther. 2016;6:233.
86. Al-Saqladi AWM. Acute kidney injury:New definitions and beyond. J Nephrol Ther. 2016;6:234.
87. Mrabet S, et al. IgA Nephropathy, β Thalassemia Minor, Psychosis and Deafness:A New Syndrome?. J Nephrol Ther. 2016;6:237.
88. Altaf J, et al. Serum electrolyte disturbances in benign prostate hyperplasia after transurethral resection of the prostate. J Nephrol Ther. 2016;6:238.
89. Mohammad AN and Morsy KH. scoring systems and risk stratification in cirrhotic patients with acute variceal Bleeding"Scoring in Variceal Bleeding". J Liver. 2016;5:195.
90. Fragoso RL, et al. Genistein produces hepatoprotection through modulating egfr expression and phosphorylation in experimental fibrosis. J Liver. 2016;5:196.
91. Taira J, et al. Relationship between the status of blood supply in the non-hypervascular hepatocellular nodules among chronic liver diseases and the hypervascular change. J Liver. 2016;5:197.
92. Majethia NK, et al. A histo-pathological study of liver in 118 cases of cirrhosis. J Liver. 2016;5:193.

e-ISSN:2319-9865
p-ISSN:2322-0104

93. Escorsell A, et al. Short-term survival in acutely decompensated cirrhotic patients. J Liver. 2016;5:194.
94. Wang X, et al. Early detection of metastases by bone scintigraphy in patients with hepatocellular carcinoma. J Liver. 2016;5:191.
95. Iraha R, et al. Hepatic Epithelioid hemangioendothelioma:vascular penetration in the tumor as a characteristic imaging finding. J Liver. 2015;4:184.
96. Silva ES, et al. Alpha-1- antitrypsin deficiency presenting as neonatal cholestasis:predictors of outcome and effect of ursodeoxycholic acid. J Liver. 2015;4:185.
97. Shizuma T. Pernicious anemia in patients with primary biliary cirrhosis, autoimmune hepatitis, and chronic viral hepatitis. J Liver;2015;4:186.
98. Bhattacharya PK and Roy A. Management of hepatitis c in the indian context: an update. J Liver. 2015;4:187.
99. Li A, et al. Difficulty in diagnosis of leiomyosarcoma of infrahepatic inferior vena cava. J Liver. 2015;4:180.
100. Hassan EA, et al. Non-invasive parameters of oesophageal varices diagnosis:which sensitive and applicable;a pilot study. J Liver. 2015;4:182.
101. Dabiri R, et al. Detection of undiagnosed wilson's disease after hepatitis a virus infection. J Liver. 2013;2:130.
102. Piechota M, et al. Liver cell carcinoma in poland:data reported to the national health fund in the years 2008-2012. J Liver. 2015;4:183.
103. León HD, et al. Modulation of the hepatic lipidome and transcriptome of apoe-/- mice in response to smoking cessation. J Liver. 2013;2:132.
104. Oldani A and Garavoglia M. Hepatocellular cancer arising from ectopic liver tissue on diaphragm in association with desmoid mesenteric tumor. J Liver. 2013;2:133.
105. Vitale A, et al. Could sorafenib disclose new prospects as bridging therapy to liver transplantation in patients with hepatocellular carcinoma? J Liver. 2013;2:134.