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Causant Of Hepatitis D Disease, Called Superinfection, And The State Of Disease In The Human

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Annotation. Chronic infection with hepatitis delta virus (HDV) affects between 12-20 million people worldwide and represents the most severe form of viral hepatitis, leading to accelerated liver disease progression, cirrhosis and its complications, such as end-stage-liver disease and hepatocellular carcinoma. Diagnosis of HDV is still challenging due to a lack of standardised assays, while accurate viral load quantification is needed to assess response and endpoints of antiviral treatment. Until recently, interferon has represented the only treatment option in patients with chronic hepatitis delta; however, it is associated with low efficacy and a high burden of side effects.

Keywords hepatitis D, HDV, virus, liver, inflammation, cirrhosis.

Introduction

Hepatitis D is an inflammation of the liver caused by the hepatitis D virus (HDV), which requires HBV for its replication. Hepatitis D infection cannot occur in the absence of hepatitis B virus. HDV-HBV co-infection is considered the most severe form of chronic viral hepatitis due to more rapid progression towards hepatocellular carcinoma and liver-related death. The human hepatitis D virus (HDV) is unique among animal viruses. Enveloped in the hepatitis B virus (HBV) surface proteins, HDV constitutes the smallest human virus with a diameter of 35–36nm. HDV requires HBV as a helper for entry into hepatocyte, intrahepatic spread and dissemination between its hosts. Although recent in vitro findings indicate that HDV may propagate independent from HBV, using envelope glycoproteins from several virus genera such as vesiculovirus, flavivirus and hepacivirus including hepatitis C virus (HCV), clinical investigations confirm its strong association with HBV infection (hepatitis B surface antigen, HBsAg positivity). Some estimates suggest that up to 60million individuals may be infected with HDV, however, another meta-analysis indicates that 12million people are affected. HBV/HDV coinfection is associated with a more severe course of the diseases and an increased mortality compared with HBV monoinfection. Simultaneous infection with HBV and HDV of adults results in clearance of both viruses in the majority of individuals. In contrast, superinfection of an HBV-infected patient with HDV typically results in the development of persistent HBV/HDV coinfection which may lead to liver cirrhosis, liver failure and eventually hepatocellular carcinoma (HCC) within short time. Indeed, 50%–70% of patients with chronic HBV/HDV coinfection develop cirrhosis within 5–10 years after diagnosis, corresponding to a threefold increase compared with HBV-monoinfected patients. The risk for HCC development is increased compared with HBV monoinfection with an odds ratio (OR) of 1.28–2.77, depending on the selection of studies included in the meta-analysis.11 Due to this increased complication rate, HDV coinfected patients account for approx. 25% of HBsAg-positive liver transplant recipients in the European Liver Transplant Registry. Until recently, no approved antiviral treatment was available against HDV, thus, a more precise understanding of HDV virology and anti-HDV immune responses is essential to develop and establish novel therapeutic regimens.

HDV genome structure The HDV genome consist of 1672–1697 ribonucleotides (genotype-dependent) and forms a single stranded covalently closed circular RNA molecule of negative polarity

(defined in relation to the (+) stranded mRNA encoding the hepatitis D antigen (HDAg)). Both, genomic and antigenomic RNA is characterised by a high degree of self-complementarity (>70%) leading to recurrent back-folded stretches of base paired rods, that are interrupted by short loops. This peculiar structure resembles the structure of plant viroid RNA and mimics a DNA double helix. Different from plant viroids, HDV RNA associates with the viral HDAg but also the protein bromodomain adjacent to zinc finger domain 2B (BAZ2B), involved in chromatin remodeling. Such 'molecular mimicry' complexes of dsDNA enables the host DNA-dependent RNA-polymerase (Pol II) to accomplish RNA-dependent RNA synthesis. HDAg is encoded as two isoforms within a segment of the HDV genome, namely the small HDAg (S-HDAg, 195 aa, 24kDa) and the large HDAg (L-HDAg, 214 aa, 27kDa). While S-HDAg is necessary to initiate and maintain replication, L-HDAg negatively regulates replication and triggers envelopment of the virus into the HBV surface proteins. Both antigens are post-translationally modified in order to fulfil their distinct functions.

The routes of HDV transmission, like HBV, occur through broken skin (via injection, tattooing etc.) or through contact with infected blood or blood products. Transmission from mother to child is possible but rare. Vaccination against HBV prevents HDV co-infection and hence expansion of childhood HBV immunization programmes has resulted in a decline in hepatitis D incidence worldwide. Chronic HBV carriers are at risk of infection with HDV. People who are not immune to HBV (either by natural disease or immunization with the hepatitis B vaccine) are at risk of infection with HBV, which puts them at risk of HDV infection. Those who are more likely to have HBV and HDV co-infection include indigenous people, people who inject drugs and people with hepatitis C virus or HIV infection. The risk of co-infection also appears to be potentially higher in recipients of haemodialysis, men who have sex with men and commercial sex workers.

In acute hepatitis, simultaneous infection with HBV and HDV can lead to a mild-to-severe hepatitis with signs and symptoms of indistinguishable from those of other types of acute viral hepatitis infections. These features typically appear 3–7 weeks after initial infection and include fever, fatigue, loss of appetite, nausea, vomiting, dark urine, pale-coloured stools, jaundice (yellow eyes) and even fulminant hepatitis. However, recovery is usually complete, development of fulminant hepatitis is infrequent, and chronic hepatitis D is rare (less than 5% of acute hepatitis). In a superinfection, HDV can infect a person already chronically infected with HBV. The superinfection of HDV on chronic hepatitis B accelerates progression to a more severe disease in all ages and in 70–90% of persons. HDV superinfection accelerates progression to cirrhosis almost a decade earlier than HBV mono-infected persons. Patients with HDV induced cirrhosis are at an increased risk of hepatocellular carcinoma (HCC); however, the mechanism in which HDV causes more severe hepatitis and a faster progression of fibrosis than HBV alone remains unclear.

Diagnosis

Serological diagnosis is made by:

detection of total antibody to HDV (anti-HDV) by enzyme-linked immunosorbent assay (ELISA); a positive HDV IgM result indicates ongoing replication

detection of HDV antigen in serum, which is present earlier than anti-HDV

detection of HDV-specific RNA by polymerase chain reaction (PCR) testing; PCR is the most sensitive assay for assessing HDV viraemia.

Endpoints of therapy. The ideal endpoint for any anti-HDV therapy would be HBsAg loss with anti-HBs seroconversion. Elimination of replicating HDV RNA from the liver in HBsAg positive patients would be an alternative, however, it would require biopsies from patients and is not applicable in clinical practice. A more practical primary endpoint outcome is serum or plasma HDV RNA (as a surrogate marker of liver HDV-RNA levels) below the limit of detection by a sensitive and specific PCR assay during therapy, at the end of treatment (EOT) and off-therapy, at least 24 weeks after treatment discontinuation. However, given the high risk of late post-treatment virological relapses described after IFN–based therapies, a

sustained off-therapy response should be confirmed over time, well beyond 24 weeks after treatment discontinuation. The proportion of patients with a≥2log IU/ml decline of HDV RNA coupled with normal ALT have also recently been suggested as reasonable secondary endpoints for clinical trials. To comply with these stringent virological endpoints, it is of paramount importance to rely on commercially available, validated, WHO standardised, sensitive and specific HDV RNA assays that may allow to compare viral kinetics within as well as across studies.

Conclusion

Conclusion, hepatitis D disease cannot develop independently, it manifests itself only when hepatitis B is present in the body, which is why it is called a superinfection. This disease is transmitted to humans through blood. Its treatment is carried out by a hepatologist. It is necessary to detect and treat this disease as early as possible.

References:

1. Masrurjon o'g'li, M. M. (2024). COMMON THYROID DISEAGES, CAUSES AND ITS TREATMENT METHODS. Miasto Przyszłości, 48, 223-232.
2. Masrurjon o'g'li, M. M. (2024, May). HUMAN GROWTH HORMONE. In Proceedings of Scientific Conference on Multidisciplinary Studies (Vol. 3, No. 5, pp. 117-125).
3. Muxammadrasul, M. (2024, May). Etiology and Pathophysiology of Diabetes Mellitus. In International Congress on Biological, Physical And Chemical Studies (ITALY) (pp. 92-96).
4. Kamalovich, S. I. (2024). Congenital Esophageal Malformations in Children, Symptoms, Diagnosis and Treatment. Miasto Przyszłości, 53, 1241-1243.
5. Boltabaev, M. U. (2023). KORONAVIRUS (COVID-19) XAMROH KASALLIK BILAN KECHGANDA KASALLIKDAN KEYINGI REABLITASIYA DAVRIDA ANIQLANADIGAN O'ZGARISHLAR VA ULARNI BARTARAF ETISH CHORALARI. Scientific Impulse, 2(13), 178-182.
6. Zakhriddinovich, I. B. (2024, June). Migraine in Children and its Causes, Symptoms and Treatment. In Interdisciplinary Conference of Young Scholars in Social Sciences (USA) (Vol. 7, pp. 29-32).
7. Erkinovich, M. B. (2023). IMPROVING THE EFFECTIVENESS OF FIRST AID TO PATIENTS WITH POLYTRAUMA. Western European Journal of Medicine and Medical Science, 1(4), 67-71.
8. Erkinovich, M. B. (2023). Prevention and Modern Treatment of Fatty Embolism in Traumatological Patients. Eurasian Medical Research Periodical, 21, 158-164.
9. Erkinovich, M. B. (2022). Increase the Effectiveness of Prevention and Treatment of Osteoporosis. Central Asian Journal of Medical and Natural Science, 3(3), 811-818
10. Zakhriddinovich, I. B. (2024, May). Febrile Seizure Disease and its Symptoms, Treatment. In International Congress on Biological, Physical And Chemical Studies (ITALY) (pp. 121-124).
11. Alimova, I. A., Rayimova, Z. M., Babadjanova, X. M., & AKTUALNOST, V. (2022). RANNEGO VMESHATELSTVA V SEMEYNIE POLIKLINIKI DETYAM RANNEGO VOZRASTA.

JOURNAL OF CLINICAL AND PREVENTIVE MEDICINE, 2, 5-11.

12. Alimova, I. (2021, January). BOLA TARBIYASIDA OTA-ONALARNING PSIXOLOGIK BILIMLARNI SHAKLLANTIRISHNING AHAMIYATI. In INTERNATIONAL CONFERENCES ON LEARNING AND TEACHING (Vol. 1, No. 1, pp. 131-132).
13. Anvarovna A.I., Melibaevna B.X., Maksamadjonovna R.Z., Zaxriddinoich I.B., Islomkulovich U.M. (2023). Aktualnost vnedreniya slujbi kompleksnogo rannego vmeshatelstva v semeynix klinikax. BioGecko jurnal novozelandskoy gerpetologii , 12 (03), 1139-1145.
14. Anvarovna, A. I., & Melibaevna, B. K. (2022). JUVENILE IDIOPATHIC ARTHRITIS. SCIENTIFIC JOURNAL OF RESEARCH IN MEDICINE (SJRM), 1(4), 6-8.
15. Melibayevna, B. X. (2023). Measures to Improve the Quality of Life of Patients with Comorbid Heart Pathology and Increase the Effectiveness of Their Treatment. Scholastic: Journal of Natural and Medical Education, 2(3), 34-36.
16. Kamalovich, S. I. (2024, May). CONGENITAL HEART DEFECTS IN CHILDREN. In Proceedings of International Conference on Modern Science and Scientific Studies (Vol. 3, No. 5, pp. 65-71).
17. Rayimov, G. N., Tillaboldiyev, A. R., Saloxiddinov, N., & Sh, D. S. (2022). Actical Errors in Surgical Treatment of Strengthened Abdominal Hernias. The Peerian Journal, 5, 130-135.
18. Mahmudov, U. I. (2024). MANAGEMENT OF THYROID NODULES. JOURNAL OF INNOVATIONS IN SCIENTIFIC AND EDUCATIONAL RESEARCH, 7(4), 1-7.
19. Isakjonovich, S. M. (2024). Effectivness of Aromatherapy in Post-Covid Syndrome. Miasto Przyszłości, 49, 1239-1242.
20. Mahmudov, U. I. (2023). COMPARATIVE CHARACTERISTICS OF CLINICAL AND LABORATORY PARAMETERS OF PATIENTS OF THE DIABETIC FOOT DEPARTMENT, DEPENDING ON THE PRESENCE OR ABSENCE OF DIABETES MELLITUS. SO 'NGI ILMIY TADQIQOTLAR NAZARIYASI, 6(12), 355-360.
21. Nazirtashova, R. M. (2023). XALQ TABOBATIDA MAKKAJO "RINING O "RNI. Journal of Chemistry of Goods and Traditional Medicine, 2(1), 210-216.
22. Mamadaliyevna, N. R. (2023). INSONIYAT O'ZINI O'ZI ZAHARLAMOQDA. "GERMANY" MODERN SCIENTIFIC RESEARCH: ACHIEVEMENTS, INNOVATIONS AND DEVELOPMENT PROSPECTS, 9(1).Izhar M, Alausa T, Folker A, Hung E, Bakris GL. Effects of COX inhibition on blood pressure and kidney function in ACE inhibitor-treated blacks and Hispanics. Hypertension 2004;43:573-7.
23. 23. Nazirtashova, R. M., & Kirgizov, S. M. (2021). Research Of Pentosal Hydrolysis Products Of

Plant Waste. The American Journal of Applied sciences, 3(04), 126-130.

24. 24. Matyakubov, R., & Nazirtashova, R. M. (2021). Valuable Raw Materials For Producing Furfural. The American Journal of Interdisciplinary Innovations and Research, 3(06), 159-165.
25. 25. Nazirtashova, R. M. (2022). DINAMICHESKOE ISSLEDOVANIE KARDIORESPIRATORNOY SISTEMI UCHENIKOV SPORTIVNIX SHKOL K OBUCHENIYU V USLOVIYAX POVISHENNOY SLOJNOSTI. BARQARORLIK VA YETAKCHI TADQIQOTLAR ONLAYN ILMIY JURNALI, 90-94.
26. 26. Anvarova, Z. (2024). SPID/VICH IFISIROVANIE I DETI. THEORY AND ANALYTICAL ASPECTS OF RECENT RESEARCH, 2(22), 41-45.
27. 27. Anvarova, Z. (2024). ZADERJKA VNUTRIUTROBNOGO RAZVITIYA PLODA KAK FAKTOR NARUSHENIYA GARMONICHNOGO RAZVITIYA DETEY. THEORY AND ANALYTICAL ASPECTS OF RECENT RESEARCH, 2(21), 234-237.
28. 28. Qosimovna, A. Z. (2023). Factors that lead to asphyxia in babies. American Journal of Pediatric Medicine and Health Sciences (2993-2149), 1(10), 740-743.
29. Abdullaev, S. (2024). AKTUALNOST PROBLEM RAZVITIYA OSTRIX PNEVMONIY U DETEY. THEORY AND ANALYTICAL ASPECTS OF RECENT RESEARCH, 2(22), 29-33.
30. Mukhtarzhanovna, I. G. (2024, May). Development of Principles of Study and Treatment of Vaginal Dysbiosis During Pregnancy. In International Congress on Biological, Physical And Chemical Studies (ITALY) (pp. 112-115).
31. Mukhtorjonovna, I. G. (2024). Modern Surgical Methods of Placental Aggregation. Web of Semantics: Journal of Interdisciplinary Science, 2(5), 412-416.
32. Solijon o'g'li, A. S. (2024). BACTERIAL, VIRAL AND MUCOPLASMA PNEUMONIA IN CHILDREN. American Journal of Pediatric Medicine and Health Sciences (2993-2149), 2(1), 273-280.
33. Abdullaev, S. (2024). PSIXOLOGICHESKIE OSOBENNOSTI UCHEBNIX IGR V PODGOTOVKE STUDENTOV MEDISINSKIX INSTITUTOV. FORMATION OF PSYCHOLOGY AND PEDAGOGY AS INTERDISCIPLINARY SCIENCES, 2(25), 222-224.
34. 34. Aleksandrovna, A.E. (2023). OSNOVNIE ASPEKTI RESPIRATORNOY REABILITASII POSLEDSTVIY NOVOY KORONAVIRUSNOY INFEKTSII U DETEY S BRONXOLEGOCHNIMI ZABOLEVANIYAMI. Vsemirniy byulleten sosialnix nauk , 18 , 81-83.
35. Abdullaev, S. S. (2023). TO THE QUESTION OF COMMUNITY-ACCOMPANIED PNEUMONIA IN YOUNG CHILDREN. Journal of Social Sciences and Humanities Research Fundamentals, 3(05), 51-53.

36. Xudaynazarova, S. R., Kuryazova, Sh. M., & Oxunova, M. J. (2023). OSOBENNOSTI BRONXOOBSTRUKTIVNOGO SINDROMA PRI VNEBOLNICHNOY PNEVMONII U DETEY RANNEGO VOZRASTA. Interpretation and researches, 1(6).

37. Anvarova, Z. (2024). SPID/VICH IFISIROVANIE I DETI. THEORY AND ANALYTICAL ASPECTS OF RECENT RESEARCH, 2(22), 41-45.

38. Anvarova, Z. (2024). ZADERJKA VNUTRIUTROBNOGO RAZVITIYA PLODA KAK FAKTOR NARUSHENIYA GARMONICHNOGO RAZVITIYA DETEY. THEORY AND ANALYTICAL ASPECTS OF RECENT RESEARCH, 2(21), 234-237.

39. Alexandrovna, A. E. (2023). Clinical and functional features of the bronchopulmonary system in chronic kidney disease. Texas Journal of Medical Science, 16, 57-59.