Hepatitis in Nursing Homes
Incidence and Management Strategies

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Contents

Summary

Due to improved sanitation and prevention of parenterally transmitted diseases, a trend towards a decreasing incidence of acute viral hepatitis has been observed in several countries in the last years. These changes in epidemiology affect elderly people in different ways, especially if they are residents of homes for the aged. The decrease of hepatitis A virus (HAV) infection in childhood has resulted in less antibody protection during adulthood and old age. This in turn has led to an increased risk of acute and severe hepatitis due to HAV infection, and/or community outbreaks even in elderly people.

The spread of parenterally transmitted viruses, such as hepatitis B virus (HBV), hepatitis C virus (HCV) and hepatitis delta virus (HDV), occurs in homes for the aged, mainly through the common use of toiletries and nondisposable syringes. Improvement in hygiene standards has lead to a marked decrease of these infections.

In conclusion, provided that the current hygiene standards are maintained, the risk of parenterally transmitted hepatitis viruses (HBV, HCV and HDV) in homes for the aged will be low. Paradoxically, the risk of enterically transmitted HAV may, in the near future, increase in wealthy institutions and/or in Western countries.

Long term residents of institutions have an increased risk of hepatitis virus infection. Several reports deal with hepatitis B virus (HBV) infection in institutions housing children,[1,2] mentally retarded people[3-11] and chronically ill people.[12] However, only sporadic papers have described the epidemiology of hepatitis virus infection in homes for the aged. Nevertheless, from the epidemiological point of view, we should distinguish studies carried out before and after the 1980s.

Table I. Interferon treatment of chronic hepatitis: prevalence of major adverse effects (autoimmunity, immunological disorders, low platelet count) according to age (Chiaramonte et al.[47])

Age (years)	Adverse effects (% of patients)
<30	13.6
31-40	16.1
41-50	32.2
51-65	32.2

1. Hepatitis A Virus Infection

Hepatitis A virus (HAV) is an enterally transmitted virus. Closed communities can become potential sites of outbreaks.[13,14] So far, nursing homes for the aged are not at an increased risk for this infection, since many of the adults and aged have anti-HAV antibodies acquired earlier in life.[15-17] However, because of improved sanitation,[18-20] HAV infection is becoming progressively less frequent in childhood or adolescence, when it is asymptomatic. We are, therefore, moving towards a situation where a large proportion of adults, and in the near future, of aged people, will be susceptible to HAV infection. This phenomenon is more likely and occurring faster in countries with high levels of sanitation[20-22] and/or in more wealthy classes.

Small food-related HAV outbreaks have already been described, even in the aged.[23,24] This may become an important problem in geriatric medicine, as HAV hepatitis is severe, frequently cholestatic and possibly lethal in the aged.[25]

Vaccines against HAV infection are now available.[26,27] They are based on inactivated virus, attenuated live virus or recombinant subunit proteins, administered by parenteral routes. Antibody response occurs in nearly 100% of adults, provided the full cycle of 3 injections is given.[28] So far, no studies involving elderly patients have been completed. Good sanitation minimises the risk of transmission in the community, although outbreaks can occur even through unexpected, occasionally contaminated foods (such as commercially available frozen fruits).[29]

No outbreaks have been reported in nursing homes so far. However, this possibility should be considered especially in developed countries.

2. Hepatitis B Virus Infection

The spread of this virus is parenteral or vertical. Household transmission has been found to be the main factor responsible for the high number of hepatitis B surface antigen (HBsAg) carriers in medium and high prevalence countries.[30,31] Clusters of HBV carriers have been described in institutions housing children,[1,2] mentally retarded people[3-11] and chronically ill people.[12] However, only sporadic papers have described the epidemiology of HBV infection in homes for the aged.

The first report in the literature deals with an outbreak attributed to the occasional sharing of bath brushes.[32] Sexual contact has also been reported to be a risk factor for HBV transmission in a nursing home in Denver, US.[33] This route was found responsible for the transmission of HBV from an HBsAg carrier to 2 people.

In 1978 we carried out a seroepidemiological study in 2 different types of institution for the elderly.[34] In this study, we found serological evidence of HBV infection and an HBsAg carrier rate significantly higher in these nursing homes than in the noninstitutionalised population. We also observed the occurrence of repeated cases of acute HBV infection between 1976 and 1978, mainly in the upper class residences. The relative risk for acute events was highest in non-immune (anti–HBs-negative) people who received injections from nondisposable materials. People from lower socioeconomic classes were more frequently antiHBs-positive and, thus, were protected from new infections.

However, in a recent survey carried out in the same institutions 12 years later (1990), we found a significant decrease in the HBsAg carrier rate.[35] Furthermore, the prevalence of any HBV markers in this group of aged people was similar to that observed in a pre-geriatric population from the same area 10 years earlier.[36] However, this

suggested a cohort effect and demonstrated the absence of new infections during this period.

HBV infection is declining in many countries.[37-40] This is only partially due to vaccination campaigns, generally directed to children or high risk groups.[41] In our experience, the decrease in HBV infection in nursing homes, as well as in other closed communities, is due to the improvements in hygiene standards and medical care.[35]

In fact, HBV transmission in homes for the aged is classically parenteral, through nondisposable syringes, toiletries and the like. Transmission from staff to residents has rarely been described,[42] although transmission from residents to staff has been reported.[43] However, vaccination of healthcare staff is now compulsory in many parts of the world.[41] Vaccination of aged people is not recommended since the response to the vaccine (evaluated by the production of anti-HBs) is poor, and less than 50% of people older than 50 years develop protective titres.[44]

Furthermore, at least in high prevalence countries and/or in low socioeconomic classes, a large proportion of elderly people have protective anti-HBV antibodies.[36]

HBV infection can lead to different clinical situations in different hosts. This is a particular problem in aged people. Infected elderly people often develop a subclinical or an oligosymptomatic hepatitis with a low rate of HBV clearance, possibly due to their impaired immunonological status. This phenomenon can produce a large percentage of highly infective chronic HBsAg carriers who are substantially healthy.[45]

Chronic hepatitis and/or cirrhosis (due to infection acquired earlier in life) are generally inactive and with a slow progression (possibly because patients with more severe disease do not reach old age). Elderly patients are not candidates for interferon treatment because of the expected low rate of response[46] and the high rate of adverse effects (table I).[47] Other candidate treatments have been the antiviral nucleoside analogues (e.g. ribavirin) and immunomodulators (e.g. levamisole and thymosin α_1). However, both were abandoned

because of an excess of severe adverse effects and no effect on HBV replication, respectively.[48]

Corticosteroids should be avoided because they increase HBV replication and may even cause deterioration of the disease.[49] Furthermore, elderly people have a high probability of adverse effects, such as iatrogenic diabetes, osteoporosis and hypertension, even after a short course of corticosteroids.

The problem of HBV in homes for the aged persists only in situations of poor hygiene, and only if the infection is introduced into the community from without.

In conclusion, prevention is simple, through the introduction of inexpensive hygiene measures.[50]

3. Hepatitis Delta Virus Infection

Hepatitis Delta Virus (HDV) is a defective virus, which requires HBV particles for its transmission.[51] No special infection has been described in homes for the elderly. HDV infection is also declining in high prevalence countries.[52]

4. Hepatitis C Virus Infection

Hepatitis C virus (HCV) is a parenterally transmitted virus, responsible for many cases of non-A, non-B hepatitis and for cryptogenic cirrhosis in Mediterranean and Far East countries.[53] From 1989, its diagnosis has been possible through serological tests.[54] Household clusters have been observed,[55] but vertical transmission is rare[56,57] and sexual transmission is still being debated.[58-60]

New cases of HCV infection rarely occur in the elderly. Most of them are associated with blood transfusions or surgery.[61,62] In general, acute post-transfusional hepatitis is an oligosymptomatic disease, jaundice being observed in only 25% of patients.[62] Despite the paucity of symptoms, 30% of the patients may have marked biochemical alterations in cholestatic indices.[62]

Epidemiological studies on the prevalence of anti-HCV antibodies in the general population are lacking. In fact, data are generally obtained from blood donors, which means that elderly patients are

excluded. Nonetheless, an epidemiological survey carried out in 11 117 blood donors in Italy seems to indicate an increase in anti-HCV prevalence with advancing age.[63] These data are also in agreement with those of Esteban and colleagues in Spain.[64] However, this trend is not confirmed by other surveys of blood donors in the Tokyo area[65] or in Sydney.[66] In fact, these discrepancies may have been due to the epidemiological differences between high prevalence countries, where HCV infection was mainly community-acquired, and low prevalence countries, where HCV spread occurred mainly through drug addiction.

HCV infection in institutions for the aged has been rarely studied.[67,68] Using a first-generation test [actually detecting an antibody against a nonstructural part of the HCV genome (anti C-100)] we found a prevalence of anti-HCV antibodies of 2.2% in a nursing home with 315 residents.[65] Clinical examination of the anti-HCV antibody-positive people failed to indicate any evidence of chronic hepatitis.

In the same year, in a study carried out in a randomly selected urban population in Italy (age range 30 to 69 years), anti-HCV antibodies were detected in 0.87% of 1484 people.[69] The highest prevalence (1.35%) was observed in the group aged 50 to 59 years. In the group aged 60 to 69 years the prevalence was 0.67%. According to these results, the prevalence of anti-HCV antibodies in the aged is less than in pre-geriatric adults, but at the same time is higher in institutionalised than in the non-institutionalised populations. Unless a clear exposure to HCV is identified in each person, the probable source of infection could be the use of nondisposable syringes earlier in life.

Two explanations could be given for the lower prevalence of anti-HCV antibodies in older ages. First, there might be a higher precocious mortality in anti-HCV antibody-positive people than in anti-HCV antibody-negative people as a result of end-stage liver disease. Secondly, HCV-related antibodies, such as anti-C 100-3, might disappear in long-lasting infections. In fact, all the previous studies were carried out using first-generation tests, detecting anti-C 100-3 only. Further studies, carried out using second-generation tests, detecting antibodies against other HCV proteins, such as c22, 5.1.1 and c33, may verify this hypothesis.

In any case, no anti-HCV antibody-positive elderly people are candidates for interferon treatment, since as with those with chronic HBV-related hepatitis, they are at high risk of adverse effects[47] and the expected response is poor.[70] Alternative treatments include: antiviral agents, such as zidovudine and ribavirin, N-acetylcysteine, iron reduction therapy and corticosteroids. Controlled trials of these treatments are in progress.[71] Corticosteroids seem to increase HCV replication; therefore, their use is discouraged at present. Since the spread of HCV is mainly parenteral,[72] no restrictions to social or household life are suggested. A vaccine against HCV is not available. Risk of HCV infection in healthcare workers should be prevented through the use of universal precautions for bloodborne pathogens.

5. Conclusions

A trend towards a decreasing incidence of HAV, HBV and HCV infections has been observed in several countries. This changing epidemiology also affects the elderly population.

So far, new cases of parenterally transmitted viral infections (HBV, HDV and HCV) in homes for the aged have been quite rare, and occur mainly through syringes or nondisposable medical devices. The widespread introduction of simple and inexpensive hygienic measures has proved to be useful in reducing the impact of parenterally transmitted infections.

By contrast, the risk of enterically transmitted HAC virus, may, in the near future, increase in Western countries and/or in wealthy institutions housing people who were never immunised in earlier life. When treating acute infections, it should be kept in mind that cholestasis may develop, and there is a frequent need for prolonged courses.

Chronic HBV or HCV infections in patients reaching older ages are generally long-lasting and rather inactive. The management of such patients should be conservative and symptomatic, since aggressive treatment, such as interferon or antiviral agents, is not justified because of the high risk of adverse effects and the limited expected benefit.

References

1. Krugman S, Giles JP. Viral hepatitis: new light in an old disease. JAMA 1970; 212: 1019-29
2. Richer G, Desrochers M, Guevin R, et al. Hepatitis B antigen in Montreal blood donors: childhood institutionalization as an epidemiological factor. Can Med Assoc J 1975; 112: 49-52
3. Szmuness W, Pick R, Prince AM. The serum hepatitis virus specific antigen (SH): a preliminary report of epidemiologic studies in an institution for the mentally retarded. Am J Epidemiol 1970; 95: 51-61
4. Szmuness W, Prince AM. The epidemiology of serum hepatitis (SH) infections: a controlled study in two closed institutions. Am J Epidemiol 1971; 94: 585-95
5. Szmuness W, Prince AM, Etling GF, et al. Development and distribution of haemoagglutinating antibody against the hepatitis B antigen in institutionalized populations. J Infect Dis 1972; 126: 498-506
6. Tevaluoto-Aarnio M. Epidemiology of hepatitis B antigenemia in an institution for the mentally retarded. Scand J Infect Dis 1974; 6: 309-13
7. Tevaluoto-Aarnio M, Werner B, London WT. Antibody to hepatitis B antigen (Australia antigen) among residents of a Finnish institution for the mentally retarded. Med Biol 1975; 53: 51-9
8. Perry E, Chaudhary RK, Cleary TE, et al. Hepatitis B surface antigen and its subtypes in an institution for the mentally retarded. Can Med Assoc J 1975; 112: 46-8
9. Nordenfelt E, Andren-Sandberg M, Lundquist A, et al. Persistence of Australia (Au) antigen among patients in a hospital for the mentally retarded and its relation to chronic liver disease. Scand J Infect Dis 1971; 3: 103-7
10. Boughton CR, Hawkes RA, Schroeter DR, et al. The epidemiology of hepatitis B in a residential institution for mentally retarded. Aust N Z J Med 1976; 6: 521-4
11. Kingham JGC, McGuire M, Paine DHD, et al. Hepatitis B in a hospital for the mentally subnormal in southern England. BMJ 1978; 2: 594-6
12. Fitzerald GR, Grimes H, Reynolds M, et al. Hepatitis-associated-antigen positive hepatitis in a tuberculosis unit. Gut 1975; 16: 1543-5
13. Szmuness W, Dienstag JL, Purcell RH, et al. The prevalence of antibody to hepatitis A antigen in various parts of the world: a pilot study. Am J Epidemiol 1977; 106: 392-8
14. Majeed FA, Stuart JM, Cartwright KAV, et al. An outbreak of hepatitis A in Gloucester, UK. Epidemiol Infect 1992; 109: 167-73
15. Vendramini R, Fiaschi E, Naccarato R, et al. Epatite virale tipi A: indagine sieroepidemiologica in Padova e provincia. Boll Ist Sieroter Milan 1980; 59: 338-47
16. Turner PC, Eglin RE, Woodward CG, et al. Screening before hepatitis A vaccination. Lancet 1992; 340: 1160
17. Finkelstein MA, Freedman ML, Shenkman L, et al. Evidence of prior hepatitis B and hepatitis A virus infections in an ambulatory geriatric population. J Gerontology 1981; 36: 302-5
18. Kremastinou J, Kalapothaki V, Trichopoulos D. The changing epidemiologic pattern of hepatitis A infection in urban Greece. Am J Epidemiol 1984; 120: 703-6
19. Hsu HJ, Chang MH, Chen DS, et al. Changing seroepidemiology of hepatitis A infection in Taiwan. J Med Virol 1985; 17: 297-301
20. Chiaramonte M, Moschen ME, Stroffolini T, et al. Changing epidemiology of hepatitis A virus (HAV) infection: a comparative seroepidemiological study (1979 vs 1989) in north-east Italy. Ital J Gastroenterol 1991; 23: 344-6
21. Papaevangelou V, Roumelioto-Karayannis A, Contoyannis P. Changing epidemiological characteristics of acute viral hepatitis in Greece. Infection 1982; 10: 1-4
22. Dubois F, Thevenas C, Caces E, et al. Seroepidemiologie de l'hepatite A dans six departements du Centre-Ouest de la France en 1991. Gastroenterol Clin Biol 1992; 16: 674-9
23. Stroffolini T, Biagini W, Lorenzoni L, et al. An outbreak of hepatitis A in young adults in central Italy. Eur J Epidemiol 1990; 6: 156-9
24. Papaevangelou G. Epidemiology of hepatitis A in Mediterranean countries. Vaccine 1992; 10 Suppl. 1: S63-S66
25. Fenstel LF. Viral hepatitis in the elderly. Gastroenterology 1965; 49: 262-71
26. Werzberger A, Mensch B, Kuter B, et al. A controlled trial of a formalin-inactivated hepatitis A vaccine in healthy children. N Engl J Med 1992; 327: 453-7
27. Siegl G, Lemon SM. Recent advances in hepatitis A vaccine development. Virus Res 1990; 17: 75-92
28. Scheifele DW, Bjornson GJ. Evaluation of inactivated hepatitis A vaccine in Canadians 40 years of age or more. Can Med Assoc J 1993; 148: 551-5
29. Niu MT, Polish LB, Robertson BH, et al. Multistate outbreak of Hepatitis A associated with frozen strawberries. J Infect Dis 1992; 166: 518-24
30. Szmuness W, Harley EJ, Prince AM. Intrafamilial spread of asymptomatic hepatitis B. Am J Med Sci 1975; 270: 293-6
31. Bruguera M, Bosh J, Rodes J, et al. Familial clustering of hepatitis B antigen: a study in relatives of patients with liver diseases and hepatitis B antigenaemia. BMJ 1974; 2: 495-6
32. Braconier JH, Nordenfelt E. Serum hepatitis at a home for the aged. Scand J Infect Dis 1972; 4: 79-82
33. Wright RA. Hepatitis B and the HBsAg carrier: an outbreak related to sexual contact. JAMA 1975; 232: 717-21
34. Chiaramonte M, Floreani A, Naccarato R. Hepatitis B virus infection in homes for the aged. J Med Virol 1982; 9: 247-55
35. Floreani A, Bertin T, Soffiati G, et al. Are homes for the elderly still a risk area for HBV infection? Eur J Epidemiol 1992; 8: 808-11
36. Trivello R, Chiaramonte M, Naccarato R, et al. Epatite virale B: indagine sieroepidemiologica nel Veneto. Boll Ist Sieroter Milan 1982; 61: 136-43
37. Papaevangelou GJ, Roumeliotou A. Reduction of HBV infections and mass immunization. Lancet 1988; 2: 53-4
38. Matsuo A, Kusumoto Y, Ohtsuka E, et al. Changes in HBsAg carrier rate in Goto Islands, Nagasaki Prefecture, Japan. Lancet 1990; 1: 955-7
39. Mele A, Stazi MA, Corona RM, et al. Decline of incidence of A, B and non-A, non-B hepatitis in Italy: results of four years surveillance (1985-88). Ital J Gastroenterol 1990; 22: 274-80
40. Chiaramonte M, Trivello R, Stroffolini T. Changing pattern of hepatitis B infection in children: a comparative sero-

epidemiological study (1979 vs 1989) in north-east Italy. Ital J Gastroenterol 1991; 23: 347-50

41. Kane MA. Progress on the control of hepatitis B infection through immunization. Gut 1993; 34 (Suppl.): S10-S12

42. Snydman DR, Hindman SH, Wineland MD, et al. Nosocomial viral hepatitis B: a cluster among staff with subsequent transmission to patients. Ann Int Med 1976; 86: 573-6

43. Pattison CP, Maynard JE, Berquist KR, et al. Epidemiology of hepatitis B in hospital personnel. Am J Epidemiol 1975; 101: 59-62

44. Denis F, Mounier M, Hessel L, et al. Hepatitis-B vaccination in the elderly. J Infect Dis 1984; 149: 1019-21

45. Chiaramonte M, Floreani A, Naccarato R. Hepatitis B virus (HBV) in the elderly: an underestimated problem? Biomed Pharmacother 1987; 41: 121-3

46. Wong DKH, Cheung AM, O'Rourke K, et al. Effect of alpha interferon treatment in patients with hepatitis B and antigen-positive chronic hepatitis B: a meta-analysis. Ann Intern Med 1993; 119: 312-23

47. Chiaramonte M, Floreani A, Delaito M, et al. Predictive factors of major side effects during Interferon (IFN) therapy [abstract]. Hepatology 1994. In press

48. Sherlock S. Strategies for treating chronic hepatitis B and chronic hepatitis C infections. L'inernista 1993; 3: 139-43

49. Lam KC, Lai CL, Ng PR, et al. Deleterious effect of prednisolone in HBsAg-positive chronic active hepatitis. N Engl J Med 1981; 304: 380-6

50. Centers for Disease Control: MMWR. Update: universal precautions for preventions of human immunodeficiency virus, hepatitis B virus, and other bloodborne pathogens in health-care settings. MMWR Morb Mort Week Rep 1988; 37: 377-88

51. Rizzetto M. The delta agent. Hepatology 1983; 3: 729-37

52. Sagnelli E, Stroffolini T, Ascione A, et al. Epidemiology of hepatitis delta infection in Italy. J Hepatol 1992; 15: 211-5

53. Chiaramonte M, Stroffolini T, Caporaso N, et al. Hepatitis C virus infection in Italy: a multicentric serepidemiological study. Ital J Gastroenterol 1991; 255-8

54. Kuo G, Choo Q-L, Alter HJ, et al. An assay for circulating antibodies to a major etiologic virus of human non-A, non-B hepatitis. Science 1989; 244: 362-4

55. Chang T-T, Liou T-C, Young K-C, et al. Intrafamilial transmission of hepatitis C virus: the important role of inapparent transmission. J Med Virol 1994; 42: 91-6

56. Reesink HW, Wong VCW, Ip HMH, et al. Mother-to-infant transmission and hepatitis C virus. Lancet 1990; 335: 1216-7

57. Ohto H, Terazawa S, Sasaki N, et al. Transmission of hepatitis C virus from mothers to infants. New Engl J Med 1994; 330: 744-50

58. Melbye M, Biggar R, Wantzin P, et al. Sexual transmission of hepatitis C virus: cohort study (1981-89) among European homosexual men. BMJ 1990; 301: 210-2

59. Tedder RS, Gilson RJ, Briggs M, et al. Hepatitis C virus: evidence for sexual transmission. BMJ 1991; 302: 1299-1302

60. Pachucki CT, Lentino JR, Schaaff D, et al. Low prevalence of sexual transmission of hepatitis C virus in sex partners of seropositive intravenous drug users. J Infect Dis 1991; 164: 820-1

61. Goodson JD, Taylor PA, Campion EW, et al. The clinical course of acute hepatitis in the elderly patients. Arch Intern Med 1982; 142: 1485-2

62. Sonnenblick M, Oren R, Tur-Kaspa R. Non A, non B hepatitis in the aged. Postgrad Med J 1990; 66: 462-4

63. Sirchia G, Almini D, Bellobuono A, et al. Prevalence of hepatitis C virus antibodies in Italian blood donors: the Italian Cooperative Group. Vox Sang 1990; 59: 26-9

64. Esteban JI, Esteban R, Viladomiu L, et al. Hepatitis C virus antibodies among risk groups in Spain. Lancet 1989; 2; 294-7

65. Watanabe J, Minegishi K, Mitsumori T, et al. Prevalence of anti-HCV antibody in blood donors in Tokyo area. Vox Sang 1990; 59; 86-8

66. Archer GT, Buring ML, Clark B, et al. Prevalence of hepatitis C virus antibodies in Sydney blood donors. Med J Aust 1992; 157: 225-7

67. Simor AE, Gordon M, Bishai FR. Prevalence of hepatitis B surface antigen, hepatitis C antibody, and HIV-1 antibody among residents of a long-term-care facility. J Am Geriatr Soc 1992; 40: 218-20

68. Floreani A, Bertin T, Soffiati G, et al. Anti-hepatitis C virus in the elderly: a seroepidemiological study in a home for the aged. Gerontology 1992: 38: 214-6

69. Rapicetta M, Attili AF, Mele A, et al. Prevalence of hepatitis C virus antibodies and hepatitis C virus-RNA in an urban population. J Med Virol 1992; 37: 87-92

70. Tiné F, Magrin S, Craxi A, et al. Interferon for non-A, non-B chronic hepatitis: a meta-analysis of randomised clinical trials. J Hepatol 1991; 13: 192-9

71. Lindsay KL. Chronic hepatitis C: issues and problems with current therapy. Postgraduate course of the American Association for the Study of Liver Diseases, November 4-5, 1993, Chicago, Illinois: 131-50

72. Alter HJ, Hadler SC, Judson FN, 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: 2231-5

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