Technical method

(a) Fix immunostained sections with 10% buffered formalin for 30–60 min.
(b) Rinse in water.
(c) Stain with Gill’s haematoxylin for 10–60 s.
(d) Differentiate in water for 10–30 min.
(e) Mount in glycerol-formalin (9:1).

The availability of a technique for nuclear counterstaining of IF preparations represents an important step forward in immunohistology. It appears to be particularly useful in the analysis of complex tissues where phase-contrast examination reveals insufficient morphological detail.

References


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Letters to the Editor

Disseminated herpes simplex virus infection with misleading clinical presentation.

A young woman, born in 1950, developed chronic renal failure in 1977 for which she received a completely matched cadaver transplant later in the same year. She made an uneventful recovery and was maintained on immunosuppressive drugs (prednisolone 12 mg/bd; propanolol 10 mg/bd; azothioprine 150 mg/day; benzofluazide 5 mg/day).

Two weeks before admission she developed a cold. This appeared to resolve but four days before admission she developed a sore throat which did not respond to treatment with ampicillin. On admission she had a swinging temperature of 39–5°C. There was no evidence of jaundice, hepatomegaly, or skin lesions.

By the next day, she was very ill and her renal function began to deteriorate rapidly with the development of proteinuria and oliguria. On the third day after admission, she developed small vesicles on the back of the left alveolar region and at the same time the liver was noted to be palpable and firm. The serum transaminase activities were grossly abnormal indicating acute hepatitis. Blood culture was negative. A throat swab failed to grow any viruses; paired sera, taken early and later during the illness indicated that there had been an infection with cytomegalovirus at some time in the past but no evidence of previous or current infection with herpes simplex...
The patient deteriorated progressively and died nine days after admission from fulminant hepatitis.

At necropsy, there were no vesicular lesions present on skin, mouth, nasopharynx, oesophagus, external or internal genitalia. The liver was massively enlarged (2550g) and there were innumerable small yellowish necrotic foci surrounded by areas of haemorrhage and congestion. Histological examination showed massive focal necrosis in zones 2 and 3. In zone 1, most of the viable hepatocytes contained Cowdry type A intranuclear inclusions, characteristic of herpes group virus infection (Fig. 1).

Electron microscopic examination of thin sections of liver, negatively stained, showed the presence of a virus of the herpes group (Fig. 2). Herpes simplex virus was recovered in tissue cultures inoculated with lung, kidney, brain, and liver tissue.

In this case, the only clinical sign which suggested a herpetic infection was the transient vesicular lesions on the lingula but the absence of antibodies to herpes simplex virus was taken, apparently reasonably, as strong evidence that this virus was not contributing to her clinical condition. For this reason, no antiviral treatment was given. We have since become aware of Flewett’s report of an adult with herpetic hepatitis in whom serology was unhelpful, the correct diagnosis being made on liver biopsy and electron microscopy.

The case of this unfortunate woman leads us to two conclusions. Firstly, a liver biopsy should be very helpful in reaching an early diagnosis in patients under immunosuppression who present with an otherwise undiagnosable deterioration in their clinical state and liver function tests. Secondly, in agreement with Walker and his colleagues, we consider that the administration of acyclovir, even in the absence of supporting conventional viral diagnostic evidence, may be a life-saving measure. The importance of attempting a histological diagnosis and the feasibility of administering acyclovir “blindly” are consequences of the efficacy and relative non-toxicity of this antiviral drug.

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References


Letters to the Editor

Serum antibodies to hepatitis B virus in alcoholic liver disease

Conflicting data have been reported concerning the higher prevalence of serum antibody to hepatitis B virus antigens in patients with alcoholic cirrhosis. We have compared 60 hospital patients with biopsy-proven alcoholic cirrhosis (mean age 46 yr ± 10 SD; 45 men) with an age- and sex-matched hospital control population, a mixed group of HBsAg-negative chronic alcoholic cirrhosis (HBsAg-veCAC) (47 ± 19 yr; 6 men) and 32 renal unit patients (43

References

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