

Case report

Endoscopic transmission of hepatitis B virus

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SUMMARY Although transmission of hepatitis B virus (HBV) infection has long been recognised as a potential hazard of gastrointestinal endoscopy, there has been little evidence of direct patient-to-patient cross-infection after such procedures. We wish to report a case of type B viral hepatitis almost certainly acquired at endoscopy from an instrument sterilised in the conventional manner, but which had been used on the previous day on a patient with bleeding oesophageal varices who was incubating type B viral hepatitis.

Case report

KP, a 50-year-old man with alcoholic cirrhosis, was admitted on 30 September 1980 after a major upper gastrointestinal haemorrhage. Upper gastrointestinal endoscopy was performed on the morning of admission by one of us (EQ) and revealed profuse continuing bleeding from oesophageal varices. A Sengstaken-Blakemore tube was immediately inserted and the haemorrhage successfully controlled. Eight units of packed red cells were transfused. The tube was removed 48 hours later. There was no recurrence of bleeding and his condition remained stable until nine days after admission when deepening jaundice and encephalopathy were noted. Investigation suggested rapidly progressive acute hepatitis: SGOT 1920 IU/l, SGPT 1060 IU/l, and bilirubin 203 $\mu\text{mol/l}$. Hepatitis B surface antigen (HBsAg) and hepatitis BeAg (HBeAg) were both positive by radio-immunoassay. Repeated serological checks had always previously shown him to be HBsAg negative, the last being performed three months before his admission. Despite intensive care he developed progressive liver failure with jaundice, ascites, encephalopathy, and coagulopathy and died 34 days after admission with a fulminant hepatitis. Histological examination of a necropsy liver biopsy

demonstrated a severe acute hepatitis superimposed on a micronodular cirrhosis.

On 1 October 1980, the day after KP's endoscopy, JC, a 78-year-old woman, had also presented with upper gastrointestinal haemorrhage, and had an upper gastrointestinal endoscopy performed in the same endoscopy suite, using the same instrument by a different endoscopist (GB). A bleeding gastric ulcer was identified and biopsies were not taken. Over the next 48 hours, she was transfused 7 units of packed red cells. There was no subsequent recurrence of upper gastrointestinal haemorrhage, and after eight days of conservative management in hospital she was discharged home.

On 4 January 1981, 96 days after the endoscopic examination, JC became jaundiced with biochemical evidence of hepatocellular injury: SGOT, 725 IU/l; SGPT, 590 IU/l; and bilirubin, 325 $\mu\text{mol/l}$. Histological examination of a liver biopsy revealed a severe acute hepatitis. Both HBsAg and HBeAg were positive. No evidence was obtained to indicate that any of the common routes of transmission of hepatitis B virus was a possibility. The 7 units of packed red cells given on her previous admission were all re-examined for both HBsAg and anti-HBc with negative results.

She made a complete recovery and on 1 March 1981, 150 days after endoscopy, HBeAg could no longer be detected, and on 20 April 1981 she also became HBsAg negative.

Endoscopic examination of both patients was performed within 24 hours in the same endoscopy

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suite using the same Olympus GIF-IT instrument. After the initial examination on KP (on 20 September 1980) the instrument had been thoroughly cleaned in the following conventional manner. First detergent and distilled water were aspirated through the suction channel and then a cleaning brush was passed through the channel. The biopsy valve was removed and the top end of the biopsy channel was flushed through using the channel adaptor and a fresh biopsy valve was inserted. The air/water channel was cleaned in the recommended fashion using 'plenty of air and water', the channel was not flushed through with activated glutaraldehyde because this is not recommended in the manufacturer's instructions. The outside of the instrument was washed in water, detergent, and freshly activated glutaraldehyde. The endoscope was left immersed in activated glutaraldehyde for a period of 21 hours until the endoscopy on JC, which is more than three times the recommended period. Before this examination the instrument was again washed with water and detergent. The two patients were managed on different wards throughout their stay in hospital and there was no direct contact between them. Hospital staff involved in the management of both patients have been tested for HBsAg and were found to be negative. Four other patients who were endoscoped on days 3, 4, and 5 after KP, using the same instrument, were checked between 110 and 125 days after endoscopy and were all found to be HBsAg and anti-HBc negative.

Subtyping of the HBsAg for the patients (JC and KP) revealed both to be type ay. This subtype accounts for only 30% of the HBV subtypes seen in blood donors in the West of Scotland.¹

Discussion

The sequence of events as illustrated in the Figure, our failure to identify any other source of hepatitis B virus, and the demonstration of an HBsAg subtype identical to that of the first patient (KP), all support our contention that the second patient (JC) acquired viral type B hepatitis as a result of patient-to-patient transmission of HBV at the time of endoscopy.

In view of the vast numbers of endoscopies performed daily on patients with gastrointestinal bleeding whose hepatitis B virus antigen status is unknown it is remarkable that endoscopic transmission of HBV does not occur more frequently. Only a small number of papers, however, have dealt with the possible consequences of examination of HBsAg carriers.²⁻⁷ Morris *et al*² in a short report, examined 32 patients who were endoscoped after the examination of a patient who

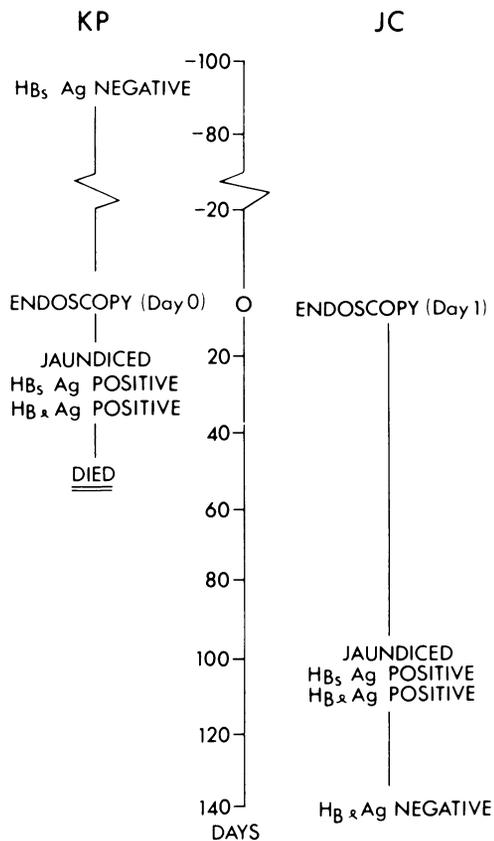


Figure Timing of events for patients KP and JC.

was HBsAg positive. Only one patient (the 23rd to be examined) became HBsAg positive between 65 and 290 days after the examination, without any clinical evidence of transmission of HBV.³⁻⁷ It is probable that instances of endoscopic transmission of HBV have not been detected because the subsequent attack of hepatitis B has been subclinical. In our case, two factors may have contributed to transmission of the virus with a subsequent symptomatic hepatitis: (1) initial patient (KP) had fulminant hepatitis at a maximally infective stage being both HBsAg and HBeAg positive and (2) the contamination to the endoscope was unusually heavy with infected blood because of the presence of actively bleeding oesophageal varices.

We can only speculate on the exact route of transmission of the infection to JC. The endoscope was cleaned and disinfected along conventional lines, being immersed in freshly activated glutaraldehyde for three times longer than the

recommended period.

There are two areas in conventional endoscopes which are particularly difficult to clean: (1) the air/water channel: the manufacturers recommend that it is cleaned using plenty of air and water. They do not specifically recommend the introduction of activated glutaraldehyde into the channel presumably because it is expected that this channel should remain uncontaminated. In practice, this is not the case and the Endoscopy Committee of the British Society of Gastroenterology (unpublished recommendations) now recommends that this channel should be soaked with disinfectant. (2) The controls and connecting tube. The design of all ranges of endoscopes, including the Olympus range, does not allow for the controls and the connecting tube to be immersed in fluid. These parts of the instrument are wiped with 70% ethyl alcohol, a measure which does not effectively inactivate HBV.⁸ It is possible that HBV survived on the controls of the instrument and was later transferred from there to the shaft, either by the endoscopist or the nurse, and then to the patient. A less likely possibility is that HBV may have survived on other surfaces in the endoscopy suite – for example, on the endoscopy trolley – and may have been transferred from there to the shaft in a similar manner. We feel this to be unlikely as disposable surfaces were used throughout.

Our report confirms that transmission of HBV can occur at endoscopy. It is important to minimise this risk. One solution to this problem would be to screen the HBsAg of all patients referred for endoscopy by radioimmunoassay before the investigation. Those patients found to be HBsAg positive could then be examined at the end of the list with an instrument specifically reserved for these patients. This solution is cumbersome and would inevitably result in some delay in performing the procedure. Financial stringencies would usually determine that such an endoscope would be often old and inefficient. We do not think that this is a practical solution to the problem.

A second solution would be disinfection with ethylene oxide. This is a time-consuming method which is expensive and not readily available in all hospitals. It seems unlikely that this method could be accepted as a routine method of disinfecting endoscopes. It should, however, be used in the specific circumstances where an endoscope is known to have been contaminated with HBV. To be effective in preventing the transmission of HBV at endoscopy this type of disinfection would again have to be coupled with a routine screen of HBsAg of all patients endoscoped.

A third solution is the use of activated

glutaraldehyde with endoscopes of a totally immersible design. A 2% alkaline aqueous solution of glutaraldehyde has been shown to be highly effective in destroying HBV. It has the added advantages of being cheap and freely available. The major disadvantage at present is that, because of their design, most endoscopes cannot be totally immersed in the fluid.

The risk of transmitting hepatitis B appears to be small. The importance of preventing the transmission, however, should not be underestimated. The infection is potentially fatal, the possibility of transmission exists, and both patients and endoscopy staff are at risk. It is important that the problem is recognised and improvements in equipment and techniques should be expedited to minimise their risks.

We would also like to draw attention to the possibility of HBV surviving on surfaces within the endoscopy suite. There is no simple method of repeatedly disinfecting the whole endoscopy suite and it is important that all surfaces with which an endoscope comes in contact should be covered with disposable towels. These towels should have an absorbent surface with an impervious material beneath. The towels should be changed after each examination.

The Endoscopy Committee of the British Society of Gastroenterology (unpublished recommendation) has recently produced a list of recommendations for the cleaning and sterilisation of endoscopes. In these recommendations they make it clear that it is impossible to formally sterilise an endoscope. With specific reference to HBV we would like to make the following recommendations.

(1) High risk patients should be screened for HBsAg before endoscopy. This screening should include patients with recent blood transfusions, infusions of pooled blood products, drug addicts, and patients with alcoholic liver disease. (2) HBsAg positive patients should be examined at the end of a list using an instrument specifically reserved for them. This instrument should then be sterilised using ethylene oxide. (3) The air/water channel should be soaked with activated glutaraldehyde during cleaning procedures. (4) Disposable covers should cover all surfaces with which the endoscope and accessories come in contact. (5) A more effective method of cleaning the controls and connecting tube should be sought. This will probably require the redesign of endoscopy equipment to make it possible to immerse these parts of the endoscopes in disinfecting fluid. (6) It is important that the cleaning and sterilisation of endoscopes should be carried out only by staff specifically trained to do these specialised tasks.

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