

# NOTES

## Possible Transmission of Serum (Australia-Antigen-Positive) Hepatitis Via the Conjunctiva

M. C. KEW

*Department of Medicine, Johannesburg General Hospital and University of the Witwatersrand, Johannesburg, South Africa*

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A nursing sister developed Australia-antigen-positive hepatitis 101 days after the blood of an Australia-antigen-positive patient was accidentally introduced into her eye. There may be important implications for those caring for patients with Australia-antigenemia or undertaking laboratory investigations on their blood.

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Serum (long incubation; MS-2; Australia-antigen-positive) hepatitis has long been known to be transmitted parenterally by infected blood or blood products or the use of contaminated syringes or needles. More recently it has been discovered that the disease may be spread in other ways. Infected blood can be transmitted orally via unsterilized dental instruments, or when a laboratory technician aspirates blood into his mouth, or through the skin breached by cuts or scratches (11, 12). There is some evidence that the disease may also be transmitted by inhalation (2) and via the genital tract (9, 11). The Australia-antigen has been found in urine (4), bile (1, 3), and feces (5, 7), and it now seems likely that there are sources of infection other than blood.

Certain viruses, e.g., measles (10) and Sindbis (6), and rickettsia, such as that responsible for tick bite fever (J. H. S. Gear, 1972, personal communication), can be transmitted via the conjunctiva. Although Marmion and Tonkin (8) have suggested the possibility that serum hepatitis may be spread by blood introduced into the eye, well-documented cases of transmission in this way appear to be lacking. This report concerns a patient in whom conjunctival transmission of Australia-antigen-positive hepatitis seems very likely, although other routes of infection cannot entirely be excluded.

**Case report.** M.T., a 29-year-old nursing sister, worked for one month in the renal unit of

the Johannesburg General Hospital as part of an intensive care nursing course run by the hospital. On 13 March 1972, while measuring the whole-blood clotting time of an Australia-antigen-positive patient undergoing hemodialysis, she accidentally squirted some of the patient's blood into her right eye. She automatically rubbed the eye once with the back of her hand, but then thought better of this and bathed the eye, first with "Savlon" (cetrimide 3%, chlorhexidine 0.3%) and then with normal saline. She was wearing a mask, surgical gown, and gloves at the time. She was not given an injection of immune globulin. Her blood was checked the following day for the presence of the Australia-antigen and this was negative. (It had also been negative on one previous occasion during the intensive care course.) "Baseline" liver function tests were performed at the same time and these were normal.

She felt well until 22 June (101 days later) when she experienced upper abdominal discomfort, nausea, anorexia, malaise, and headache. She did not notice any change in the color of her urine and thought that she had influenza. She continued to work but stayed in bed when not on duty. On 29 June her senior noticed that she was jaundiced and she was admitted to the hospital. At this time her symptoms were unchanged. There was no history of jaundice or previous liver disease, and she was not taking any drugs apart from an occasional aspirin. She

had not been in contact with any jaundiced patients except for a day in February 1972 when she nursed a patient with Australia-antigen-positive acute hepatitis. During this day, and throughout her time in the renal unit, she wore gloves, a gown, and a mask, and did not prick or cut herself or come into direct contact with any of the patients' blood.

On examination, her temperature was 37.8 C (100 F), her pulse was 84 beats per minute, and her blood pressure was 100/64 mm of Hg. She was moderately jaundiced. The liver was enlarged to 2 cm below the costal margin and was tender; the spleen was 1 to 2 cm larger. The remainder of the examination was unremarkable. The urine was dark brown and contained bilirubin (3+) and urobilin (3+). The total serum bilirubin was 6.1 mg%, of which 3.8 mg% was conjugated, serum glutamic oxaloacetic transaminase was 920 Reitman-Frankel units, serum glutamic pyruvic transaminase was 1,240 Reitman-Frankel units, alkaline phosphatase was 19.7 King-Armstrong units, total protein was 6.5, albumin was 3.4, and gamma globulin was 1.7 g%. The Australia-antigen was detected in the serum by gel diffusion, counter-immunoelectrophoresis, and complement fixation (titer  $\frac{1}{2}$ ). (The titer of the Australia-antigen in the dialysis patient's serum at the time of the accident was  $\frac{1}{8}$  by the complement fixation test.) Neither the Australia-antigen in the nursing sister's serum nor that in the dialysis patient was subtyped. Three days later the biochemical changes were at their worst (total serum bilirubin, 15.2 mg%; conjugated bilirubin, 8.5 mg%; serum glutamic oxaloacetic transaminase, 1,740 units; serum glutamic pyruvic transaminase, 2,120 units; alkaline phosphatase, 16.5 units; total protein, 7.2; albumin, 3.6; gamma globulin, 1.45 g%), and thereafter they improved, returning to normal early in August. The Australia-antigen continued to be detected in the serum, in decreasing titers, until the 20th of July. Liver biopsy was not performed. The patient was treated with bed rest alone. Her symptoms subsided after a few days in the ward and she recovered completely from the acute attack.

The possibility that the eye may serve as a portal of entry for serum hepatitis emphasizes the need of adequate protection, for nurses and doctors when caring for patients with Australia-antigenemia and for laboratory technicians when handling their blood, serum, and possibly urine and faeces. For nursing and medical personnel, the problem is most acute in renal dialysis and transplant units. The modes of spreading of the virus from patients to staff in

these units have recently been reviewed by Marmion and Tonkin (8). Particularly important in the context of possible conjunctival transmission is a spray of blood into the face from a detached or ruptured blood line when an infected patient is undergoing hemodialysis, or the vomiting of blood-stained material into the face. Similar situations might arise in patients in hepatic coma from fulminant serum hepatitis, who require intensive nursing care and who may be treated by exchange transfusion or heterologous liver perfusion. Marmion and Tonkin have advised that staff looking after patients with Australia-antigen in their blood should wear safety glasses in addition to masks, gloves, and surgical gowns, and the present case provides support for this suggestion. Laboratory technicians handling infected blood or excreta should do likewise.

The entry of the antigen via the conjunctiva may be facilitated by rubbing the eye or bathing it with an antiseptic solution. It would seem preferable, therefore, just to bathe the eye with water or normal saline. The possibility that immunoglobulin (or hyperimmune globulin when it is available) introduced into the eye shortly after the exposure may prevent infection is intriguing, and since it should do no harm, should probably be tried.

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