MEDICAL PRACTICE

Clinical Problems

Hepatitis in Dialysis Units in the United Kingdom

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British Medical Journal, 1972, 3, 94-99

Summary

A prospective study of hepatitis in 20 dialysis units in the United Kingdom showed that the number of units with outbreaks increased from one in 1968 to three in 1969 and the overall attack rate rose from 1.7 to 5.3% in patients and from 0.5 to 1.3% in staff.

In July 1969 a pilot study of patients' sera showed that all of the outbreaks were associated with Australia (Au) antigen. Au antigen was not detected in the unaffected units or in one unit in which there were several patients with abnormal liver function.

In January 1970 systematic prospective testing for Au antigen was begun. Au antigen was detected in single patients in five more of the 20 units. The transfer of the affected patients to isolation for dialysis seemed to prevent the spread of infection in the units. The incidence of hepatitis in 1970 was 5.6% in patients and 0.4% in staff.

Introduction

Hepatitis is a particular hazard in dialysis units. Awareness of the problem resulted from reports of outbreaks in individual units in many countries,¹⁻³ but this is the first prospective study of the frequency of outbreaks in dialysis units in the United Kingdom and of the incidence of hepatitis among patients and staff.

In January 1968 a survey of hepatitis in dialysis units was

begun at the Epidemiological Research Laboratory, Central Public Health Laboratory, with the collaboration of consultants in charge of 20 haemodialysis units in the United Kingdom. In July 1969 facilities for testing for Australia (Au) antigen became available at the Virus Reference Laboratory, Central Public Health Laboratory. The results of a pilot survey in July 1969 suggested that Au antigen should be a useful marker of infection which, used systematically, could provide early identification of infection and indicate how hepatitis enters and spreads within dialysis units.

Regular screening tests were introduced in January 1970 and it was hoped to use the information to prevent outbreaks either by blocking the routes of entry of infection or, when this was not achieved, by identifying infected patients within the unit and isolating them as soon as possible.

General Plan and Methods

SURVEY OF INCIDENCE OF HEPATITIS

Consultants in charge of 20 dialysis units in the United Kingdom participated in the investigation from January 1968. Another unit was included in 1969 and a further seven in 1970, making 28 in all. The units were: *Belfast City Hospital*; *United Cambridge Hospitals (Addenbrooke's Hospital)*; *Cardiff Royal Infirmary*; *Carshalton, St. Helier Hospital*; *Charing Cross Hospitals (Fulham Hospital)*; *Derby City Hospital*; *Exeter and Mid-Devon Hospitals (Whipton Hospital)*; *Glasgow Royal Infirmary*; *Glasgow Stobhill General Hospital*; *Glasgow Western Infirmary*; *Guy's Hospital*; *Hull Royal Infirmary*; *King's College Hospital (Dulwich Hospital)*; *Leeds General Infirmary*; *Liverpool Sefton General Hospital*; *the London Hospital*; *Manchester Withington Hospital*; *Middlesbrough North Ormesby Hospital*; *Newcastle-upon-Tyne, Royal Victoria Infirmary*; *Newcastle General and Rye Hill Hospitals*; *United Oxford Hospitals (the Churchill Hospital)*; *Plymouth General Hospital*; *Portsmouth St. Mary's General Hospital*; *Royal Air Force (Princess Mary's Hospital)*; *Royal Free Hospital*; *Royal Postgraduate Medical School (Hammersmith Hospital)*; *St. Bartholomew's Hospital (St.*

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Leonard's Hospital); Sheffield Royal Hospital and Lodge Moor Hospital; Sunderland Royal Infirmary. The 20 units in italic entered the survey in 1968.

Personal records were completed for patients and staff in the 28 collaborating units and collected centrally. Special records were completed for each case of hepatitis. Staff who left the units were sent an inquiry six months later to find if they had had an illness with jaundice.

AU ANTIGEN PILOT SURVEY JULY 1969

Sera from patients and staff in 15 of the 21 survey units were examined at the Virus Reference Laboratory. Similar tests were made for a further three of the survey units by Dr. G. C. Turner, Liverpool Public Health Laboratory Service, and Dr. J. R. Morgan, department of medical microbiology, Welsh National School of Medicine.

Thereafter, in units where Au antigen had been detected sera were tested at about two-weekly intervals for patients and usually at monthly intervals for staff until an outbreak was thought to be over.

PROSPECTIVE STUDY IN 1970 IN UNITS WITHOUT OUTBREAKS

Sera from all patients and staff were examined at the outset. Subsequently sera from patients were tested at quarterly intervals and all newcomers to the unit were tested on arrival.

Blood for unit patients was tested for Au antigen before transfusion whenever possible. Otherwise retrospective tests were made if specimens were available and/or sera from the patients were tested four to six weeks after transfusion.

When Au antigen was first detected in any unit the infected patient was dialysed in isolation as soon as possible in another part of the hospital, in another hospital, or preferably at home. Sera from all patients were tested at about two-weekly intervals for six months after the last date on which any infected patient was dialysed in the unit. Sera from staff were tested at monthly intervals. Liver function tests were usually made in parallel with Au antigen tests.

Cross-infection precautionary measures were reviewed and possible improvements were instituted. A detailed account of these measures is available.⁴

LABORATORY TESTS FOR AU ANTIGEN

All specimens were tested by gel diffusion by the method described by Prince.⁵ Various hyperimmune antisera were used.

TABLE 1—Number of Dialysis Units in Survey and Number Reporting Hepatitis

Year	No. of Units in Survey	No. of Units with Hepatitis Outbreaks*	No. with Sporadic Hepatitis*		Total with Evidence of Hepatitis
			Clinical	Other	
1968	20	1 (5%)	2	2	5 (25%)
1969		3 (15%)	3†	2†	7 (35%)
1969		3† (15%)	1	4	8 (40%)
1969	21 (one unit entered survey in 1969)	4 (19%)	2	2	8 (38%)
1970	28 (seven more units entered survey in 1970)	4 (14%)	1	5	10 (36%)

*For definition of terms see text.

†One unit had one clinical and seven "other" cases in 1969. This unit came into the outbreak category in 1970 but the outbreak was not associated with Au antigen.

TABLE II—Haemodialysis Units with and without Outbreaks or Sporadic Cases of Hepatitis according to Results of Gel Diffusion Tests for Au Antigen made in July 1969

Units from which Specimens were Examined	No.	Units in which Au antigen was Detected in One or More Specimens	
		No.	%
With outbreaks beginning or continuing in 1969	4	4	100
With sporadic* hepatitis at some time in 1969	4†	0	0
Without hepatitis throughout 1969	10	0	0
Total	18	4	22

*For definition of terms see text.

†One unit (unit 5) had eight patients with abnormal results of liver function tests but only one had clinical hepatitis.

Specimens giving doubtful or unexpectedly negative results in the gel test were re-examined by either complement fixation test or immune electron microscopy or both. The methods used have been described in detail.⁶

Definitions

The following terms used throughout the report:

Patients.—A patient with irreversible renal failure having maintenance dialysis, usually haemodialysis.

Clinical Hepatitis.—An illness with either jaundice or serum bilirubin above the normal maximum and/or enlarged liver not attributed to any cause other than viral hepatitis.

Other Possible Hepatitis.—Laboratory evidence of infection—that is, Au antigen detected in serum—or possible infection—that is, abnormal results of serum transaminase tests—with or without symptoms such as anorexia, malaise, abdominal pain, etc.

Liver Function Tests.—Serum transaminase or serum bilirubin tests or both. All results reported to be abnormal are included.

Outbreak of Hepatitis.—Two or more cases of clinical hepatitis detected within six months of each other in patients and/or staff of one unit.

Sporadic Hepatitis.—Other instances of clinical hepatitis, Au antigen carriage, or abnormal liver function.

Unit Technical Staff.—Staff employed in maintaining and cleaning equipment.

Unit "Other Staff."—Clerks, orderlies, secretaries, etc.

Results

There were 35, 39, and 43 dialysis units in the United Kingdom in the years 1968, 1969, and 1970 respectively. Of these, 20 participated in the survey in 1968, 21 in 1969, and 28 in 1970.

SURVEY OF HEPATITIS IN 1968 AND 1969

In 1968 there were no new outbreaks but attacks continued in one unit in which an outbreak began in 1966 (Table I). In 1969 outbreaks began in two more of the 20 units and another (unit 5) reported abnormal liver function in eight patients. There had been an outbreak in unit 5 in 1966-7⁷ but further cases were not reported in 1968.

During the two years there were five unrelated sporadic cases of clinical hepatitis. Except in one unit (5) they were not associated with other hepatitis.

Another unit entered the survey in 1969 after an outbreak had begun.

AU ANTIGEN PILOT SURVEY

Tests of sera from patients in 18 of the survey units in July 1969 showed that Au antigen was present in four units in which outbreaks were in progress or began shortly afterwards (Table II) but it was not detected in sera from 13 units without outbreaks, nor could it be found in any sera from unit 5, in which there were patients with abnormal liver function.

INCIDENCE OF HEPATITIS IN UNIT PATIENTS AND STAFF

In the 20 units the number of patients with clinical hepatitis rose from four in 1968 to 12 in 1969 (Table III). The increase in incidence (0.9% to 2.0%) was almost entirely due to outbreaks in two units. Most of the increase in "other" hepatitis (0.9% to 3.3%) resulted from the intensive laboratory testing in 1969 in these two units, but a substantial contribution was made by unit 5.

Staff also experienced hepatitis more often in 1969 (1.3%) than in 1968 (0.5%).

Comparisons of the incidence of hepatitis per 100 weeks of treatment or duty showed differences similar to those between attack rates based on the number of persons.

Almost all the cases of hepatitis in 1969 were in the four units with outbreaks (Table IV). The hepatitis risk to staff in the units seemed very small in the absence of outbreaks.

The Outbreaks

The incidence of hepatitis during the four antigen-associated outbreaks is shown in Table V. The outbreak in unit 2 was still in progress at the end of 1970, and attacks in the first six months of 1971, in persons present in 1970, are included.

The overall incidence of hepatitis in patients was high, ranging

from 48% to 22%. Of the staff groups the nurses suffered the highest attack rate (21%) but there were wide differences between the outbreaks in this respect. Infection was clinical more often in staff (87%) than in patients (38%). This difference is statistically significant at the 0.1% level, but staff had fewer laboratory tests than patients. Nevertheless, it seems unlikely that the observed difference between patients and staff was wholly artificial. During 1968-70 88% of staff and 64% of patients with clinical hepatitis had frank jaundice.

Details of illness were received for 57 cases of hepatitis in staff and previously healthy home contacts of patients in units with outbreaks. Of these, 7 (12%) were considered severely ill. There were no deaths.

The first cases in all four outbreaks appeared before the Au antigen pilot study in July 1969, and the source of infection could be ascertained only in unit 4. In this unit Au antigen was detected in the serum of one patient in July 1969. Tests of stored sera showed the antigen in his first sample (March 1969) after admission to the unit in February 1969. The first of the secondary cases appeared in September 1969.

In unit 1 an outbreak continued from 1966 despite increased precautions against cross-infection, regular liver function tests, and the use of human normal immunoglobulin. These measures were also taken in the three other units in which outbreaks began in 1969, but in addition in all four units intensive Au antigen screening tests were introduced in 1969 and efforts were made to dialyse infected patients in isolation either at home or in hospital. Three of the four outbreaks ended before or early in 1970.

In unit 2 facilities for dialysis in isolation were limited. Although infected patients were transferred to home dialysis whenever possible and some received renal transplants, no fewer than five Au antigen carriers were, of necessity, still being dialysed in the unit at the end of 1970. New patients were not

TABLE III—Incidence of Hepatitis in Patients and Staff in Survey Units during 1968-9

Year	No. of Survey Units	Category	No. of Persons in Unit During Year*	No. of Cases of Hepatitis			Attack Rate (per 100 Persons)	Attack Rate (per 100 Person- weeks)
				Clinical	Other†	Total		
1968	20	Patients All unit staff	468	4	4	8	1.7	0.06
1969	20 (continuing from 1968)		820	3	1	4	0.5	0.02
			608	12	20	32	5.3	0.20
			All unit staff	903	10	2	12	1.3
1969	1 (joined survey in 1969)	Patients All unit staff	67	5	6	11	16.4	0.77
1969	21 (total in 1969)		55	3	0	3	5.4	0.19
			675	17	26	43	6.4	0.24
			All unit staff	958	13	2	15	1.6

*Those with at least one week of staff duty or consecutive dialyses.

†Other possible hepatitis (for definition of terms see text).

TABLE IV—Incidence of Hepatitis in Staff and Patients in 1969 in Four Haemodialysis Units with Outbreaks and in 17 Units without Outbreaks

Units in Terms of Outbreaks	Category	No. of Persons* in Unit During 1969	No. of Cases of Hepatitis		
			Clinical†	Other†	Total
4 Units beginning or continuing outbreaks	Patients All unit staff Patients All unit staff	169	14	19	33 (19.5%)
17 Units not experiencing outbreaks		260	12	2	14 (5.4%)
		506	3‡	7§	10 (2.0%)
		698	1	0	1 (0.1%)

*Persons with at least one week of staff duty or consecutive dialyses.

†For definition of terms see text.

‡One in unit 5.

§All in unit 5.

TABLE V—Incidence of Hepatitis in Four Haemodialysis Units during Course of Outbreaks which began before or during 1969

Unit No.	No. of Patients				No. of Staff											
	Attacked				Medical				Nursing				Technical and "Other"			
	Exposed	Clinical	Other*	Total	Exposed	Attacked	Attacked	Total	Exposed	Attacked	Attacked	Total	Exposed	Attacked	Attacked	Total
1	28	2	7	9 (32%)	14	0	0	0	49	4	0	4 (8%)	9	0	0	0
2	75	14	22	36 (48%)	8	2	0	2 (25%)	41	18	2	20 (49%)	28	0	1	1 (4%)
3	37	2	6	8 (22%)	10	3	0	3 (30%)	34	5	2	7 (21%)	41	0	0	0
4	44	6	4	10 (23%)	22	0	0	0	26	1	0	1 (4%)	18	0	0	0

*Other possible hepatitis (for definition of terms see text).

†Persons infected before 1968 are not included.

accepted after March 1970, but new cases appeared throughout 1970 and in 1971. Infection spread beyond the unit to involve five members of renal transplant teams, five laboratory workers, two ward staff, and even the wife of one of the infected surgeons. Eleven attacks of hepatitis among the families of infected patients were reported.

There is no evidence that the use of human normal immunoglobulin affected the course of any of the outbreaks of Au antigen-associated hepatitis.

Tests for Au antigen in the outbreaks

The pattern of Au antigen detection in unit 4 differed from that in the other three outbreaks as regards both the amount present in the patients' sera and the duration of carriage. The results of testing sera from affected patients in units 2 and 4 are compared in Table VI. Both units sent the samples to a single laboratory where they were tested by uniform methods.

The different detection rates in the two units could not be explained by differences in the frequency of sampling. More patients were transplanted or transferred to home dialysis in unit 2 so that samples were fewer and the intervals between them more irregular than in unit 4.

The outbreaks in units 1 and 3 have been described.^{8, 9} Both were similar to the outbreak in unit 2 in the ease of detection of Au antigen and its persistence. The selection and treatment of patients in the four units appeared to be similar. It is possible that different strains of the infecting agent could cause differences

between outbreaks. Au antigen in all four outbreaks, however, is of the a+y+ subtype of Le Bouvier.¹⁰

PROSPECTIVE STUDY COMBINED WITH REGULAR TESTS FOR AU ANTIGEN IN 1970

In 1970 a further seven units entered the survey, bringing the total to 28 units. Some units were able to arrange to have all blood for transfusion screened for Au antigen at source. Others followed one of the alternative procedures (see above). Altogether 2,457 units of blood were given to patients in the 28 dialysis units. In only one instance was there circumstantial evidence that infection was introduced into a previously unaffected unit by blood transfused to a patient.

In January 1970 24 of the 28 units had not had outbreaks of antigen-associated hepatitis. During 1970 Au antigen was not detected in sera from 18 of the 24 units, and these 18 units did not have outbreaks of hepatitis, but Au antigen was detected in specimens from the remaining six units. Details of events in the six units are given in Table VII.

Infection was introduced into four of the six units (units 6, 8, 9, and 5) by carrier patients without clinical hepatitis then or subsequently. The route of entry appeared to be direct from transfused blood in unit 7. The source of infection in unit 10 could not be determined.

Au antigen carriage was established before admission of the patients to units 6 and 9, and one of these had certainly never received a blood transfusion. In units 8 and 5 the carrier patients

TABLE VI—Detection of Au Antigen and its Persistence in Haemodialysis Patients between July 1969 and December 1970 in Two Units with Outbreaks

Patients										Unit 2	Unit 4
No. with any evidence of infection	..	..	..	..	..	..	..	..	..	32	10
No. with clinical hepatitis	..	..	..	..	..	..	..	..	..	13	6
Any evidence* of abnormal liver function	..	..	..	..	..	..	..	..	..	22	10
Any evidence† of Au antigen in month of onset	..	..	..	..	..	..	..	..	..	17	10
Evidence of Au antigen by gel diffusion test in month of onset	..	..	..	..	..	..	..	..	..	30	10
Evidence of Au antigen by gel diffusion test in month after month of onset	..	..	..	..	..	..	..	..	..	30	8
Evidence of Au antigen by gel diffusion test in month after month of onset	..	..	..	..	..	..	..	..	..	26	10
Evidence of Au antigen by gel diffusion test six months after month of onset	..	..	..	..	..	..	..	..	..	23	7
										15	0
										13	7
										10	0

*Serum aspartate transaminase tests.

†Any test result above normal maximum limit.

‡Gel diffusion tests, complement-fixation tests, and immune electronmicroscopy.

TABLE VII—Events in Six Dialysis Units Without Previous Outbreaks of Antigen-associated Hepatitis into which Au Antigen was Introduced During 1970

Unit No.	Specimens in Which Au antigen First Detected			Action Taken when First Antigen-positive Specimen Reported	Outcome
	Date (1970)	Category	Comment		
6	29 May	Regular specimen from patient in unit. No symptoms of hepatitis	Patient never had blood transfusion. Previous sample January 1970 reported negative but readily positive on retesting	Patient (partially trained for home dialysis) and continued dialysis training in isolation. Later transferred to home dialysis*	No infection in unit staff or patients. One laboratory worker with antigen-negative hepatitis. Antigen detected in another without symptoms (June 1970)
	19 May	Specimen tested before admission	Patient from abroad	Patient not admitted to unit*	No infection
7	19 May	Specimen from patient in unit who developed clinical hepatitis	No known source of infection in unit. Blood for transfusion in unit not screened. Patient given 2 pints (1,140 ml) Jan. 1970 + five pints (2,850 ml) March 1970	Patient transferred to home dialysis*	No infection in unit staff or patients. One laboratory worker with antigen-positive hepatitis (July 1970). No known contact with specimens from infected patient
8	22 June	Quarterly specimen from patient in unit. No symptoms of hepatitis	Patient readmitted after transplant rejection. Admission sample negative. Transplant had been performed in another unit during an outbreak	Patient transferred to unit in which infection almost certainly acquired*	No infection
9	10 Aug.	Specimen from patient in unit who developed clinical hepatitis	Previous quarterly specimens negative	Immediate complete screening revealed carrier patient who began dialysis. Peritoneal March 1970. Haemodialysis July 1970. Admission sample omitted. Patients (case and carrier) transferred to infectious diseases hospital for dialysis in isolation. Later home dialysis*	Outbreak. Another patient developed antigen-positive hepatitis Feb. 1971. Also transferred to infectious diseases hospital. No further cases or carriers detected in subsequent nine months, this outbreak was limited to three patients
5	22 Oct.	Quarterly specimen from patient in unit. No symptoms of hepatitis	Patient worked in a drug addiction clinic	Patient continued dialysis in isolation*	No subsequent Au antigen-associated infection in unit patients or staff. Outbreak not associated with Au antigen
10	14 Oct.	Specimen from patient who developed clinical hepatitis. Patient transferred to home dialysis 3 August	Source of infection speculative. Regular sampling in 1970 sometimes incomplete. Most blood for transfusion screened in 1970	Screening of patients in late October 1970 did not reveal carrier*	Outbreak. In December 1970 antigen detected in four unit patients, one with clinical hepatitis. Outbreak continued in 1971

*Sera from all patients tested at about two-weekly intervals for six months after last date positive case or carrier dialysed in unit. Intervals between staff tests variable, usually monthly intervals.

appeared to have acquired the infection from sources outside the unit during the period of study.

In unit 9 two other patients were infected after the inadvertent admission of a carrier patient. All three were isolated and there were no other cases.

Unit 10 had a larger outbreak which began in December 1970. In this unit some specimens had been omitted. Retrospective tests of available sera did not determine the original source. A patient who was readmitted from home dialysis without a screening test may have introduced the infection in October 1970. Earlier in the year this patient had received an unscreened blood transfusion from a donor who was later identified as a carrier of Au antigen. Nevertheless, the possibility of an unknown source in the unit cannot be discounted.

The outbreak in unit 5 was unlike all other outbreaks. Au antigen was not detected at any time except in specimens from one patient who worked in a drug addiction clinic and who presumably acquired his infection there late in 1970. Abnormal liver function was detected in patients throughout 1969 and only one of the staff developed hepatitis although 26 patients were affected in a two-year period. Events in this unit illustrate the complexity of the situation in dialysis units where two types of hepatitis may be present simultaneously or where drugs in use may give rise to hepatic dysfunction.

Survey Results in 1970.—Of the original 20 units two had outbreaks of antigen-associated hepatitis in 1970 compared with three in 1969 and one in 1968 (Table I). By the end of the third year of the survey (1970) five of the original 20 units had been affected at some time by outbreaks of antigen-associated hepatitis, and Au antigen was found in the sera of patients in a further three of the units. In the 20 units in 1970 an overall attack rate of 6% in previously uninfected patients is shown in Table VIII. This is similar to the corresponding attack rate in 1969 (5%), but half of the cases of hepatitis in 1970 were contributed by unit 5 compared with only a quarter in 1969. In 1970 the overall attack rate in previously uninfected staff (0.4%) was much lower than the corresponding attack rate (1.3%) in 1969.

None of the seven units first included in the survey in 1970 had outbreaks.

Discussion

In 1968 and 1969 hepatitis was neither epidemic nor endemic in most of the survey units. Nevertheless, the increase in outbreaks in 1969 suggested that in the course of time more of the units would be affected unless some means of reversing the trend could be found. Surveillance reports from the U.S.A.^{11 12} show that hepatitis was reported from 80% of their participating units during 1967-70.

From the present results it seemed reasonable to assume that most outbreaks of hepatitis in dialysis units are antigen-associated, and the tendency of patients with chronic renal failure to become long-term carriers with little biochemical evidence of liver disease¹³ has been confirmed by this study. Au antigen screening tests may be the only way of finding such carriers.

The experience of unit 4 suggested that the presence of an asymptomatic carrier of Au antigen in a unit need not necessarily lead to the immediate spread of infection but that the continued treatment of such a patient in the open unit would eventually lead to infection of other patients.

Infection spread among patients in all the outbreaks even when equipment was not shared, and there seemed to be little opportunity for parenteral spread. Hepatitis has been transmitted by the ingestion of serum containing Au antigen.¹⁴ Much contamination of the environment with blood may follow shunt haemorrhage¹⁵ and patients treated with anticoagulants often bleed from other sites. In these circumstances it is not surprising that cross-infection is common.

The immediate practical question which arose from the findings in 1969 was how best Au antigen testing could be used to control the outbreaks which were in progress and to prevent outbreaks in units as yet unaffected.

The most important approaches to the control of the outbreaks which were in progress seemed to be: (1) to identify by means of intensive screening tests carriers of Au antigen and persons in the early stages of infection; (2) to dialyse carriers and cases in isolation from the unit; and (3) to review and strengthen, if necessary, cross-infection precautionary measures.

Three of the four outbreaks appeared to be controlled in 1970 by the application of these principles despite the fact that the method of testing in general use was relatively insensitive. In the one outbreak which defeated control the chief obstacles were that isolation facilities were limited and that home dialysis could not be arranged for all infected patients.

Staff in the most direct contact with patients suffered the highest attack rates in the outbreaks but, besides the quality of the precautions taken to avoid infection, the incidence of hepatitis in staff is likely to be related to several other factors such as the number of infected patients treated, the persistence of Au antigen carriage, and the duration of their treatment in the unit.

Apart from the immediate danger to staff and patients hepatitis in a dialysis unit delays the treatment of new patients and restricts the transplantation programme. Some infected patients usually remain persistent carriers, and they are a continuing hazard to those assisting their dialyses whether at home or in hospital isolation; they also reintroduce infection when emergency hospital treatment is necessary. It is obvious that the number of carrier patients should be kept to a minimum and that this is best done by preventing outbreaks rather than by controlling them.

The study in 1970 showed that the most common route of entry of Au antigen to dialysis units was via a carrier patient infected from a source outside the unit, either before admission or during the period of treatment at the unit. The screening of blood for transfusion, although necessary, is not the complete solution to the problem of hepatitis in dialysis units.

In the outbreaks in the survey none of the 38 infected staff died, although some of the illnesses were severe. In contrast, case fatality rates among staff in some other outbreaks were large. In one outbreak in the U.K. in 1965 there were three deaths among 14 infected staff.¹⁶ In another outbreak in the U.K. in 1969 there were four deaths among 12 infected staff.¹⁷

TABLE VIII—Incidence of Hepatitis in Patients and Staff in Survey Units during 1970

No. of Survey Units	Category	No. of Persons in unit During Year	No. of Cases of Hepatitis		
			Clinical*	Other*	Total
20†	Patients	645	8	28	36 (5.6%)
	All unit staff	1,136	3	1	4 (0.4%)
1‡	Patients	37	6	15	21 (56.8%)
	All unit staff	60	14	3	17 (28.3%)
7§	Patients	88	1	0	1 (1.1%)
	All unit staff	225	0	0	0 (0.0%)
Total 28	Patients	770	15	43	58 (7.5%)
	All unit staff	1,421	17	4	21 (1.5%)

*For definition of terms see text.

†Entered survey in 1968.

‡Entered survey in 1969.

§Entered survey in 1970.

Although all the relevant factors cannot be assessed it is possible that the differences in case fatality rates among staff in individual outbreaks reflect strain differences of the infecting agent.

Au antigen testing is the most valuable laboratory method for the detection of hepatitis in dialysis units, but liver function tests may provide useful support by showing infection in a small portion of patients in whose sera Au antigen cannot be detected readily and by indicating the presence of infection not associated with Au antigen.

The procedures adopted in 1970 were limited by the resources available for testing, especially the supply of antibody for use as a reagent. Blood for transfusion was not then screened at source and few dialysis units had isolation accommodation suitable for haemodialysis. Although the procedures were by no means comprehensive the results of the study in 1970 are encouraging. Au antigen was detected in specimens from each of six previously unaffected units. Four of the six units escaped outbreaks. It seems significant that in the two units in which outbreaks did follow the appearance of Au antigen it was found that the procedures of the prospective study had not been adequately observed. One of these units, in fact, inadvertently acted as a "control" by admitting an unscreened carrier patient. Secondary infection of other patients followed. Fortunately prompt initiation of isolation and intensive screening procedures limited this outbreak to three patients.

The rise from 1968 to 1969 in the incidence of hepatitis among patients and staff was halted in 1970. It seems likely that this was related to the measures of control and prevention initiated in July 1969 and January 1970 respectively.

Regular laboratory testing with sensitive methods combined with isolation of infected patients seems to be the most effective means available at present of controlling and preventing hepatitis in dialysis units. Some interval, however, must always exist between tests, and tests are not infallible. Units must therefore be considered at risk at all times and effective cross-infection precautions should be observed routinely.

Until effective immunization against hepatitis becomes available continued surveillance of dialysis units is essential and it should be extended to other associated groups—for example, surgical transplant teams. Present systems of screening should be improved in sensitivity and frequency. These measures should be supported by the screening of all blood for transfusion and the provision of adequate isolation facilities for the

dialysis of any patient found to be a carrier of Au antigen or showing any other evidence of hepatitis.

We are grateful to the dialysis unit staff who completed the records and dispatched the specimens. We thank Miss N. Morris and Mrs. J. Miller for helping with the co-ordination of the studies, Miss S. P. March and Mr. F. D. Hargreaves, who tested the specimens from most of the units, and Dr. Anne A. Field, who performed the electron microscopy.

We are grateful to Dr. R. S. Fox (Department of Microbiology, Royal Free Hospital), Dr. F. O. MacCallum (Radcliffe Infirmary, Oxford), Dr. J. R. Morgan (Department of Medical Microbiology, Welsh National School of Medicine), Dr. J. Nagington (Cambridge Public Health Laboratory), and Drs. G. C. Turner and G. B. B. White (Liverpool Public Health Laboratory), who kindly supplied their results of Au antigen testing, and to Dr. G. L. Le Bouvier (Department of Epidemiology and Public Health, Yale University) for allowing us to quote the subtyping results.

We acknowledge with gratitude the assistance of many members of the Public Health Laboratory Service who collaborated with the dialysis units in their areas.

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Contemporary Themes

Effects of Legal Abortion on Gynaecology

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British Medical Journal, 1972, 3, 99–102

Summary

The effect of the Abortion Act on gynaecological work in one provincial teaching hospital is that despite an increase in patient turnover of 45% the waiting list has

increased by 200%. It is suggested that the Act has had little effect on the birth rate or illegitimacy and that there may have been an increase in criminal abortion.

Introduction

When the Abortion Act 1967 was implemented in April 1968 it was expected that it would result in an increase in gynaecological work in the United Kingdom. It was hoped that this increase could be assimilated into existing services without embarrassment to routine gynaecology. Keith,¹ however, showed that in

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