CDR REVIEW

Volume 1
Review Number 8
19 July 1991
ISSN 0144-3186

Communicable Disease Report

Outbreaks of hepatitis B virus infection associated with infected surgical staff

J Heptonstall

Summary

Twelve outbreaks of hepatitis B virus (HBV) infection associated with HBV infected surgical health care workers (11 surgeons; one perfusion technician) were reported between 1975 and 1990 in England, Wales and Northern Ireland. A total of 95 infections was identified. Transmission rates ranged from one to nine per cent but were higher for patients who had undergone major surgical procedures. The number of infections reported under-estimates the total number of patients who will have acquired HBV infection from HBV infected surgeons during this period because subclinical infections will have been missed and other outbreaks may not have been recognised or reported.

Introduction

Hepatitis B virus (HBV) infection was first recognised as an occupational hazard for health care workers in 1949, when blood bank technicians, whose jobs included sharpening needles from reusable transfusion-giving sets, developed acute hepatitis[1]. Outbreaks which affected staff in renal dialysis units and diagnostic laboratories followed[2,3]. It became apparent that surgeons and dentists were also at risk, and that HBV infected health care workers could, in turn, transmit the virus to their patients while carrying out invasive surgical or dental procedures[4-6]. The risks to health care workers have been reduced considerably by the recognition that percutaneous exposure to infective blood carries the highest risk of transmission[7]; by advances in hospital and laboratory infection control[8,9]; by the development of occupational health services; by the provision of hepatitis B immunoglobulin for post exposure prophylaxis[10,11], and by the availability of an effective vaccine[12,13]. Nevertheless, HBV infected health care workers continue to infect patients.

The detection of two outbreaks associated with HBV infected surgeons in 1990 prompted this review of similar outbreaks reported between 1975 and 1990.

Data sources

Since 1972, consultant microbiologists in England, Wales and Northern Ireland have reported the results of laboratory tests for serological markers of HBV infection to the PHLS at Colindale. A case of acute HBV infection is defined as an individual, with or without jaundice, with a reported diagnosis of acute hepatitis confirmed by positive results of tests for HBsAg and/or anti-HBc IgM. Results of tests for HBeAg and anti-HBe may also be reported. Demographic details and information about occupation, risk behaviour and exposure to iatrogenic risk – including a history of blood transfusion, surgery, dentistry, or injection in the six months preceding onset of illness – are recorded on the report form.

Data on the outbreaks presented here were derived from the confidential laboratory reports received between 1980 and 1990 in which surgical exposure was recorded. This information was supplemented by a review of published reports of outbreaks of HBV infection associated with surgical health care workers in the United Kingdom between 1975 and 1990, and by personal communication with reporting consultant microbiologists. An outbreak was defined as two or more cases of acute HBV infection in patients known to have had an invasive surgical procedure performed by a surgical team which included an HBV infected health care worker.

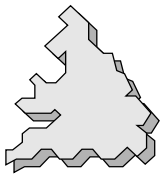
Outbreaks of hepatitis B virus infection associated with infected surgical staff
J Heptonstall
R81

Rubella vaccination in pregnancy
P A Tookey
G Jones
B H R Miller
C S Peckham
R86

The COVER scheme: a survey of immunisation coordinators
S J Gillam
N T Begg
R88

'COVER' (Cover of vaccination evaluated rapidly): 18
S G Handford
J M White
N T Begg
R90

Toxic shock syndrome in the UK: 1985-1990
R R Marples
A A Wieneke
R91



Correspondence to: Editor CDR,
PHLS Communicable Disease Surveillance Centre,
61 Colindale Avenue, London NW9 5EQ
Tel: 0181 200 6868 Fax: 0181 200 7868 Telex: 8953942

Table 1 Outbreaks of HBV infection associated with infected surgical staff

Outbreak	Publication (reference)	Transmission period	Specialty	Serological survey	Number of infections detected		Partner transmission
					Icteric	Anicteric	
A	No	1976-7	General surgery	No	3	2	Yes
B	14	1978	Gynaecology	No	8	ND	ND
C	15	1976-9	Gynaecology	No	9	ND	ND
D	16	1983 (?)	Cardiothoracic surgery	No	6	ND	ND
E	16	1983 (?)	Cardiothoracic surgery	No	5	ND	ND
F	No	1983-4	Cardiothoracic surgery	No	3	1	Yes
G	19*	1986-7	Not reported	No	4	ND	ND
H	19*	1986	Gynaecology, cardiothoracic and general surgery	No	3	ND	ND
J	17,19	1986	Gynaecology	Yes	5	17	None
K	18,19	1987-8	Cardiothoracic surgery	Yes	5	12	Yes
L	*	1983-9	General surgery	No	3	ND	ND
M	*	1990	Cardiothoracic surgery	Yes	2	3	None

ND Not determined * Full publication awaited

Laboratory reports of acute HBV infections were also a source of data on acute HBV infections in surgeons. Data on outbreaks or cases of acute HBV infection associated with infected dental staff have not been included.

The outbreaks

Twelve outbreaks of HBV infection associated with HBV infected health care workers were reported in the period 1975 to 1990 (Table 1). Three occurred between 1975 and 1979, three between 1980 and 1984 and four between 1985 and 1989.

Two were investigated in 1990. Detailed reports of outbreaks B[14], C[15], D[16], E[16], J[17] and K[18] have been published and an account of outbreak M is in preparation.

The methods of case ascertainment varied. In most of the outbreaks, an active search for further icteric cases was made following the recognition of acute icteric HBV infection in two or more patients with a history of surgical exposure and in whom there was an association in terms of time, place and surgical team. In four investigations (B, C, D and H), questionnaires were distributed to the general practitioners of those patients who had been exposed to the source health care worker. In outbreaks C and L, local laboratory records of positive results of tests for HBsAg were matched to operating theatre records. In three of the twelve outbreaks (J, K and M) serological surveys of patients exposed to the source health care worker were conducted. Although no formal case finding exercise was undertaken in outbreaks A and F, other information available suggests that any patient with acute HBV infection on whom laboratory tests were performed would have been recognised as being associated with the outbreaks. The methods of case ascertainment for outbreaks E and G were not reported.

A total of 91 surgical patients who acquired acute HBV infection from the HBV infected source health care workers was detected in the 12 outbreaks. Fifty-six of these had icteric and 35 had asymptomatic infections. The number of cases of acute icteric HBV infection per outbreak ranged from two to nine (mean – five) (Table 1). The three serological surveys of patients exposed to an HBV infected health care worker revealed 44 of the acutely infected patients, of whom only 12 became icteric. In three of the outbreaks (A, F and K), HBV was transmitted from infected surgical patients to their sexual partners, resulting in a further four infections. Thus, 95 infections were identified in total.

Some of the HBV infected health care workers transmitted the virus over several years and the recognition that surgical patients were being infected was sometimes delayed because transmission events had been widely spaced in time. For example, in outbreak B, the investigation was triggered by the diagnosis of three cases of acute icteric HBV infection following gynaecological surgery in 1979: of the six further cases identified, two underwent surgery in 1976, one in 1977, and three in 1978[14]. In outbreak L, the simultaneous discovery in 1990 of a chronically HBV infected surgeon and an acutely infected patient (upon whom that surgeon had performed an abdominal operation in 1989) led to an investigation which identified two further patients who developed acute icteric HBV infections in 1983 and 1985 after operations performed by that surgeon.

The transmission rate over a selected period was determined for five outbreaks (B, C, J, K and M) (Table 2). In outbreaks B and C, only the rate for acute icteric HBV infections was estimated. The transmission rates for both acute icteric and recent anicteric HBV infections were determined in the three outbreaks (J, K and M) in which serological surveys of exposed patients were conducted. More than 90% of exposed patients were followed up in four of these five investigations. The percentage of patients found to have acquired acute icteric HBV infections ranged from one to two per cent. For the three outbreaks in which anicteric infections were also determined, overall transmission rates ranged from four to nine per cent.

The transmission rate cannot be calculated for seven of the outbreaks because the number of patients exposed to the source health care worker, and selected for follow-up, was not reported. In episode E, five of the patients operated on by a cardiothoracic surgeon in the eighteen day period prior to the development of his symptoms, developed icteric acute HBV infections[16], giving a transmission rate of at least five per cent.

In the three outbreaks associated with gynaecological surgery (B, C and J), the surgical procedures were categorised either as 'major' or 'minor', or as 'high', 'medium', or 'low' risk

Table 2 Transmission rates for outbreaks of HBV infection associated with infected surgical staff

Outbreak	Specialty	Exposed patients selected for follow-up	Number (%) of patients followed up	Number (%) of acute icteric infections	Number (%) of acute anicteric infections	Overall transmission rate (%)
B	Gynaecology	646	589 (91)	8 (1.4)	ND	–
C	Gynaecology	–	1020	9 (0.9)	ND	–
J	Gynaecology	268	247 (92)	5 (2.0)	17 (6.8)	9
K	Cardiothoracic surgery	361	279 (77)	5 (1.8)	12 (4.3)	6
M	Cardiothoracic surgery	128	123 (95)	2 (1.6)	3 (2.4)	4

ND not determined

and transmission rates were reported for each category[14,15,17]. The way in which the procedures were categorised differed between studies, so the rates are not strictly comparable, but it is clear that the risk of acquiring HBV infection during 'major' or 'high risk' surgery was greater than that during 'minor', 'medium' or 'low risk' surgery. Between four and five per cent of patients categorised as having 'major' or 'high risk' obstetric or gynaecological surgery subsequently developed acute icteric HBV infection. In outbreak J, where asymptomatic infections were also determined, a further 16 (15%) of the 108 patients investigated had serological evidence of a recent acute HBV infection, giving an overall transmission rate for the 'high risk' category of 20%[17]. In the same outbreak, only one of 139 patients in 'medium' and 'low risk' categories developed serological markers for HBV and this followed a delivery in which Keilland's and Simpson's forceps were used and an episiotomy performed. In outbreak C, one of 517 patients who underwent a 'minor' procedure (a forceps delivery with manual removal of the placenta) developed acute icteric HBV infection[15].

Complete information about the outcome for patients who acquired HBV as a result of surgery is available only for outbreaks J, K and M. None of the patients infected in outbreak M or the five patients who developed icteric infection in outbreak J remained HBsAg positive. However, one of the 17 patients with anicteric infection in outbreak J was still HBeAg positive one year after diagnosis[17]. Four of the 17 infected patients in outbreak K became HBeAg positive carriers and were offered alpha interferon therapy[18].

In all but one of the outbreaks, the source health care worker was HBeAg positive. The HBeAg status of the surgeon involved in outbreak G was not reported. Ten of the other eleven source health care workers were surgeons, of whom nine were HBeAg positive carriers and one had acute hepatitis B. Three of the nine HBeAg positive carriers had a history of acute icteric HBV infection. Three others had received all or part of a course of HBV vaccine and the absence of detectable anti-HBs after immunisation had been documented in two of them. Both the outbreaks associated with general surgery involved consultant surgeons. Trainee surgeons were implicated in eight of the other nine outbreaks. The remaining health care worker was a cardiothoracic perfusion technician who had become an HBeAg positive carrier following anicteric acute HBV infection. The HBsAg subtype of the infected patients matched that of the infected health care worker in all nine outbreaks in which subtyping was reported.

The career outcome was reported for nine of the HBV infected surgeons. The surgeon with acute HBV infection became HBsAg negative within weeks. One of the HBeAg positive surgeons was successfully treated with alpha interferon and another eventually became anti-HBe positive without treatment; both resumed their careers. Another six, two of whom had transmitted HBV after recognition of their HBeAg positive status and appropriate counselling, ceased to perform surgical procedures on patients. The perfusion technician, who had also transmitted HBV to patients after his HBeAg positive status became known, accepted an offer of retraining[16].

Discussion

It is clear that the 91 HBV infected surgical patients reported do not represent the total number of patients infected in these outbreaks. Serological surveys are time consuming and the follow-up of large numbers of patients may present considerable difficulties. The date on which a health care worker acquired HBV infection will rarely be known and, because an HBV infected health care worker may have transmitted the virus during invasive procedures for much longer than the minimum incubation period of three months, it will rarely be possible to identify and investigate all the patients exposed to an HBV infected health care worker. The serological follow-up of patients exposed to HBV infected surgical staff has other benefits beyond determining the extent of the outbreak. It allows appropriate immunoprophylaxis to be offered to the sexual partners of infected patients and early treatment given to patients who develop chronic HBV infection[18,20].

Serological surveys were conducted in only three of the twelve outbreaks. All three demonstrated that patients had become infected asymptomatically and that the number of asymptomatic infections exceeded the number of icteric infections. The number of infections recognised was three times greater than would have been found by the ascertainment of icteric infections alone. If there had been a similar proportion of asymptomatic infections in the other nine outbreaks and if, as is likely, some clinical infections were not diagnosed, there could have been over 200 infections in the twelve reported outbreaks.

The recognition and reporting of the outbreaks described here was facilitated by the existence of a laboratory reporting system and good communications within a network of medical microbiologists in close contact with clinicians. It is, however, possible that other outbreaks occurred in the United Kingdom between 1975 and 1990.

In most of the outbreaks reported here, specimens were not requested from surgical team members until two or more patients had developed acute icteric HBV infections within six months of surgery and the association between them had been recognised. Many patients with a history of surgical exposure have also received blood transfusions, and it has been usual to exclude transfusion acquired HBV infection before investigating the possibility of HBV transmission from an infected health care worker. Outbreaks of the kind described here would be recognised earlier, and transmission of

HBV to patients would be reduced, if the surgical team members involved were asked to provide specimens for HBV testing whenever a patient developed acute HBV infection within six months of a surgical procedure.

The problem could be eliminated entirely if there were no HBV infected surgical staff. An effective HBV vaccine is available to which more than 90% of healthy adults produce an antibody response[12,13] and occupationally acquired HBV infection is, therefore, largely preventable. Nevertheless, surgeons continue to acquire HBV infection. Reports of acute HBV infections received by the PHLS between 1985 and 1990 included five surgeons, none of whom had received HBV vaccine. Studies of vaccine uptake by surgeons in the United Kingdom suggest that between 64% and 90% of surgeons have received complete courses of HBV vaccine[21-23]. The reasons given by surgeons for vaccine refusal included fear of needles, lack of concern, and the belief that 'being infected with HBV was God's punishment for sloppy surgery'[23]. These studies also reported that 54% of general surgeons and 45% of vascular surgeons who received vaccine had not had their levels of anti-HBs checked afterwards[21,22]. Individuals who are HBsAg positive do not develop detectable anti-HBs following HBV vaccine.

Three of the surgeons implicated in the outbreaks reported here had received all or part of a course of HBV vaccine. Unresponsiveness to vaccine had been documented, though not acted upon, in two. None of the three had a history of acute hepatitis. Although it is not recommended that tests for HBV infection are performed routinely before vaccination, it is certain that one surgeon, and probable that the other two, were HBeAg positive when the first dose of vaccine was given. Transmission of HBV from surgeons to patients during invasive procedures would not, therefore, be eliminated by universal acceptance of HBV vaccine by surgeons followed by post-immunisation testing for anti-HBs alone.

It has been suggested that surgeons should be known to have been immunised and to have detectable anti-HBs before they operate[24], and that all medical students should receive HBV vaccine[17]. Most of the medical and dental schools in the United Kingdom recommend that their students should receive HBV vaccine, but not all insist, and many do not perform serological tests for anti-HBs on students after immunisation[25]. Testing for anti-HBs after immunisation would not, by itself, identify the small proportion of students who may have acquired HBV infection before entry to medical school.

Surveys of the prevalence of HBV in surgical health care workers have been conducted in the United States[26] but not in the United Kingdom. However, it is unlikely that the proportions of surgeons with HBV infection would greatly differ, or that injuries to health care workers during invasive surgical procedures which could result in transmission of HBV would occur in the United Kingdom at rates different from those reported in the United States[27-29]. Surgical techniques, patients' access to surgery, the prevalence of HBV infection in patients, and vaccine availability are closely comparable in the United Kingdom, North America and Europe. The paucity of published reports of outbreaks of HBV infection associated with infected surgical health care workers in North America and Europe[30-35] suggests that, as in the United Kingdom, such outbreaks may have passed unrecognised or unreported.

Conclusion

Patients surgically exposed to an HBV infected health care worker are at a demonstrable and avoidable risk of acquiring HBV infection. On average, one outbreak of HBV infection was associated with an HBV infected surgical health care worker each year from 1975 to 1990. The extent of HBV transmission to patients undergoing surgical procedures has, however, probably been greater.

It is expected that universal immunisation of medical students and of health care workers involved in invasive surgical procedures, followed by determination of their HBV immune status, will progressively reduce the transmission of HBV from health care workers to patients. Health care workers who are already HBsAg positive, however, do not respond to vaccine. It may now be justified for surgical health care workers known to be HBeAg positive, including those who will have been identified as a result of their failure to respond to vaccine, to be asked to desist from performing invasive surgical procedures, even before transmission to their patients has been documented.

Acknowledgements

The invaluable contributions made by reporting microbiologists and the Virus Reference Laboratory, Colindale, are gratefully acknowledged.

J Heptonstall MRCP MRCPath
HIV/STD/Hepatitis Division,
Communicable Disease Surveillance Centre

References

1. Trumbull ML, Greiner DJ. Homologous serum jaundice. An occupational hazard to medical personnel. JAMA 1951; 145: 965-7.
2. Pattison CP, Maynard JE, Berquist KR, Webster HM. Serological and epidemiological studies of hepatitis B in haemodialysis units. Lancet 1973; ii: 172-4.
3. Pattison CP, Boyer KM, Maynard JE, Kelly PC. Epidemic hepatitis in a clinical laboratory. Possible association with computer card handling. JAMA 1974; 230: 854-7.
4. Rosenberg JL, Jones DP, Lipitz LR, Kirsner JB. Viral hepatitis: An occupational hazard to surgeons. JAMA 1973; 223: 395-400.
5. Foley FE, Gutheim RN. Serum hepatitis following dental procedures: a presentation of 15 cases, including three fatalities. Ann Intern Med 1956; 45: 369-80.
6. Levin ML, Maddrey WC, Wands JR, Mendeloff AI. Hepatitis B transmission by dentists. JAMA 1974; 228: 1139-40.
7. Werner BG, Grady GF. Accidental hepatitis-B-surface-antigen-positive inoculations. Ann Intern Med 1982; 97: 367-9.
8. Department of Health and Social Security, Scottish Home and Health Department, Welsh Office. Hepatitis and the treatment of chronic renal failure: report of the Advisory Group 1970-1972. HMSO, 1972.
9. UK Health Departments. Guidance for clinical health care workers: protection against infection with HIV and hepatitis viruses: recommendations of the Expert Advisory Group on AIDS. HMSO, 1990.
10. Seeff LB, Wright EC, Zimmerman HJ, Alter HJ, Dietz AA, Felsher BF, et al. Type B hepatitis after needle-stick

exposure: prevention with hepatitis B immune globulin. Final report of the Veterans Administration Cooperative Study. Ann Intern Med 1978; 88: 285-93.
11. Grady GF, Lee VA, Prince A M, Gitnick GL, Fawaz KA, Vyas GN, et al. Hepatitis B immune globulin for accidental exposures among medical personnel: final report of a multicenter controlled trial. J Infect Dis 1978; 138: 625-37.
12. Szmuness W, Stevens CE, Hanley EJ, Zang EA, Oleszho WR, William DC, et al. Hepatitis B vaccine: demonstration of efficacy in a controlled clinical trial in a high-risk population in the United States. N Engl J Med 1980; 303: 833-41.
13. Zajac BA, West D J, McAleer W J, Scolnick E M. Overview of clinical studies with hepatitis B vaccine made by recombinant DNA. J Infect 1986; 13 Suppl A: 39-45.
14. PHLS Collaborative Study Report. Acute hepatitis B associated with gynaecological surgery. Lancet 1980; i: 1-6.
15. A District Control of Infection Officer. Acute hepatitis B following gynaecological surgery. J Hosp Infect 1987; 9: 34-8.
16. Polakoff S. Acute hepatitis B in patients in Britain related to previous operations and dental treatment. BMJ 1986; 293: 33-6.
17. Welch J, Webster M, Tilzey AJ, Noah ND, Banatvala JE. Hepatitis B infections after gynaecological surgery. Lancet 1989; i: 205-7.
18. Flower AJE, Prentice M, Morgan G, Mitchell CJ, Nicholson KG, Rana B, et al. Hepatitis B infection following cardiothoracic surgery. International symposium on viral hepatitis and liver disease, 1990; Abstract.
19. Polakoff S. Acute viral hepatitis B: laboratory reports 1980-4. BMJ 1986; 293: 37-8.
20. Perrillo RP, Schiff ER, Davis GL, Bodenheimer HC, Lindsay K, Payne J, et al. A randomised controlled trial of interferon alfa-2b alone and after prednisone withdrawal for the treatment of chronic hepatitis B. N Engl J Med 1990; 323: 295-301.
21. Williams JR, Flowerdew ADS. Uptake of immunisation against hepatitis B among surgeons in Wessex Regional Health Authority. BMJ 1990; 301: 154.
22. Berridge DC, Galea MH, Evans DF, Pugh S, Hopkinson BR, Makin GS. Hepatitis B immunization in vascular surgeons. Br J Surg 1990; 77: 585-6.
23. Porteous MJLeF. Operating practices of and precautions taken by orthopaedic surgeons to avoid infection with HIV and hepatitis B virus during surgery. BMJ 1990; 301: 167-9.
24. O'Dowd TC, O'Dowd SMM. Precautions taken by orthopaedic surgeons to avoid infection with HIV and hepatitis B virus. BMJ 1990; 301: 440-1.
25. Scully C, Matthews R. Uptake of hepatitis B immunisation amongst United Kingdom dental students. Health Trends 1990; 22: 92.
26. West DJ. The risk of hepatitis B infection among health professionals in the United States: a review. Am J Med Sci 1984; 287: 26-33.
27. Gerberding JL, Littell C, Tarkington A, Brown A, Schecter WP. Risk of exposure of surgical personnel to patients' blood during surgery at San Francisco General Hospital. N Engl Med 1990; 322: 1788-93.
28. Tokars JI, Bell D, Marcus R, Culver D, Chamberland M, McKibben P, et al. Percutaneous injuries during surgical procedures. Seventh International Conference on AIDS, Florence 1991; Abstract TH.D.108
29. Panlilio AL, Foy DR, Edwards JR, Bell DM, Welch BA, Parrish CM, et al. Blood contacts during surgical procedures. JAMA 1991; 265: 1533-7.
30. Grob PJ, Moeschlin P. Risk to contacts of a medical practitioner carrying HBsAg. N Engl J Med 1975; 293: 197.
31. Grob PJ, Bischof B, Naeff F. Cluster of hepatitis B transmitted by a physician. Lancet 1981; ii: 1218-20.
32. Haerem JW, Siebke JC, Ulstrup J, Geiran O, Helle I. HBsAg transmission from a cardiac surgeon incubating hepatitis B resulting in chronic antigenaemia in four patients. Acta Med Scand 1981; 210: 389-92.
33. Coutinho RA, Albrecht-van Lent P, Stoutjesdijk L, Meerburg-Snarenburg P, Couroucé-Pauty AM, van Dijk BA, et al. Hepatitis B from doctors. Lancet 1982; i: 345-6.
34. Carl M, Blakey DL, Francis DP, Maynard JE. Interruption of hepatitis B transmission by modification of a gynaecologist's surgical technique. Lancet 1982; i: 731-3.
35. Lettau L A, Smith JD, Williams D, Lundquist WD, Cruz F, Sikes RK, et al. Transmission of hepatitis B with resultant restriction of surgical practice. JAMA 1986; 255: 934-7.

Citation of articles published in the *Communicable Disease Report*

Articles published **before** the *Communicable Disease Report* became a formal publication at the beginning of 1991 should be cited with the weekly issue number in parentheses eg:

Newman CPS, Peel RH, Jones DM. Meningococcal meningitis in a military establishment. Communicable Disease Report 1990;(33):3-4. Internal publication of the Public Health Laboratory Service, London.

Citations for articles published **after** the beginning of this year should use the appropriate volume and page numbers as follows:

Maguire HC, Begg NT, Handford SG. Meningoencephalitis associated with MMR vaccine. Communicable Disease Report 1991;1:R60-1.

Page numbers for the four-weekly review require the prefix 'R' in contrast to the simple pagination of the weekly bulletin.

Rubella vaccination in pregnancy

PA Tookey, G Jones, B H R Miller, CS Peckham

Summary

Ninety-two pregnancies complicated by rubella vaccination were reported to the Rubella Vaccination in Pregnancy Study between 1981 and 1990. These pregnancies resulted in 87 live births, 3 stillbirths and two spontaneous abortions. One infant had serological evidence of congenital rubella infection but was healthy and normal with no indication of rubella damage at the age of two and half years. These findings suggest that the risk of rubella-associated damage following inadvertent rubella vaccination in pregnancy or shortly before is low, and support the current advice that termination of pregnancy should not be routinely recommended in these circumstances.

Introduction

Concern about the number of terminations of pregnancy associated with rubella vaccination (Figure) led to the setting up of the Rubella Vaccination in Pregnancy Study (RVPS) in 1981, under the auspices of the National Congenital Rubella Surveillance Programme[1,2]. The aim was to establish whether rubella vaccines were teratogenic, so that recommendations could be made concerning the timing of rubella vaccination in relation to conception, and about the management of pregnancies complicated by rubella vaccination. Pregnancy is one of the few contraindications to rubella vaccine; the Department of Health (DOH) now recommends avoiding conception for one month following vaccination[3] but, until 1989, a delay of three months was advised.

Patients and methods

Obstetricians throughout the UK were asked to report women who had received rubella vaccine during pregnancy or up to one month before conception (up to three months until the change in the DOH recommendations in 1989) and who had decided to continue the pregnancy. The notifying obstetrician provided details of the type of vaccine, timing of vaccination and the expected date of delivery. The paediatrician completed a short form recording neonatal details as soon as possible after delivery. Blood samples were taken from the woman and her baby; the presence of rubella specific IgM in the baby at birth was taken as evidence of intra-uterine infection.

The paediatrician was asked to examine the infant at nine months of age to identify any defects not apparent at birth. A second blood sample was taken at this time, since persistence of IgG antibody at this age strongly suggests the presence of congenital infection[4]. The general practitioner was asked to arrange a further examination close to the child's third birthday to ensure that any late sequelae were reported.

Figure Terminations of pregnancy associated with rubella vaccination (OPCS data for England and Wales)

Results

By the end of 1990, information was available on the outcome of 92 pregnancies; 63 were reported prospectively and 29 retrospectively. These pregnacies resulted in 87 live births, 3 stillbirths and 2 spontaneous abortions. Sixty-one women were known to have been vaccinated with RA27/3, the vaccine now in routine use. Of the remaining 31 women, seven received Cendehill vaccine and vaccine type was not recorded for 24. These 31 women all delivered live infants. One infant had pulmonary atresia and another a systolic murmur consistent with a closing ventricular septal defect; maternal vaccination was carried out 62 and 21 days after the last menstrual period (LMP), respectively, and vaccine type was not known for either. Neither of these two infants had serological evidence of rubella infection and both were healthy at three years with no further defects detected.

Among the 61 women known to have received RA27/3

Table 1 Immune status and outcome of pregnancy in women receiving RA27/3 vaccine

Immune status before vaccination	Number	Outcome of pregnancy			Specific IgM at birth (number tested)
		Live births	Stillbirths	Spontaneous abortions	
Susceptible	30	26	2	2	1 (20)
Immune	1	1	–	–	– (1)
Not known	30	29	1	–	– (23)
Total	61	56	3	2	1 (44)

vaccine, 42 were notified to the study before delivery and 19 after. Their pregnancies resulted in 56 live births, two spontaneous abortions and three stillbirths, as shown in table 1. One miscarriage occurred at nine weeks' gestation, following vaccination close to the estimated date of conception. No laboratory investigations were carried out and notification was retrospective. The other miscarriage occurred at 26 weeks' gestation, following vaccination about three weeks after the LMP. Fetoscopy was performed six weeks before the miscarriage but rubella-specific IgM was not detected, either then or in cord blood, and rubella virus was not isolated from placental tissue. This notification was prospective. Both women were reported to be susceptible to rubella prior to vaccination.

The three stillborn infants were delivered between 35 and 38 weeks' gestation; in no case was a clear cause of death established. Post-mortem examination was carried out on one infant and did not provide evidence of congenital infection; neither of the other two stillborn infants had external features of congenital rubella or serological evidence of rubella infection. Two of the mothers had been vaccinated more than one month before their LMP after being identified as susceptible on pre-conception screening. The third, whose immune status was unknown, was vaccinated 25 days after her LMP. Two of the three stillbirths were notified retrospectively.

Rubella serology was performed on 46 of the 56 live-born infants. One, who was normal at birth, had serological evidence of congenital infection i.e. rubella-specific IgM in the newborn period and persistent IgG. This child had no defects when examined at nine months and the general practitioner confirmed that the child was healthy and normal at the age of two and half years. His mother was reported to be susceptible and vaccinated five weeks after her LMP.

Forty of the 56 children were examined at around nine months of age, and 20 of them plus two others were examined at three years or older. One child had an 'innocent systolic murmur' noted at nine months of age but was normal at three years, and another was reported to have a Marcus Gunn phenomenon affecting one eye at the age of three years.

Discussion

The absence of defects characteristic of congenital rubella in this study of rubella vaccination in pregnancy is consistent with experience in the United States[5], West Germany[6,7] and Sweden[7] and suggests that any risk to the fetus is small. These studies occasionally identified infants with laboratory evidence of congenital rubella infection, although none of them was found to have rubella-associated defects. Combination of the available data from the UK, US, Sweden and West Germany (personal communication) for infants born to susceptible women given rubella vaccine of any type shortly before conception or during pregnancy, reveals an observed risk of congenital rubella syndrome of 0/492 (Table 2). The 37 cases from the UK include the women from the present study who received any form of rubella vaccine after they were found to be susceptible on testing. Using the 95% confidence limits of the binomial distribution, the maximum risk of congenital rubella syndrome in an infant whose mother's pregnancy is complicated by rubella vaccination is not likely to be greater than 0.75%.

Only eight live-born infants in the present study and 74 in the US study were born to susceptible women known to have received RA27/3 vaccine during the likely period of greatest vulnerability i.e. from one week before to four weeks after conception (conception estimated as 14 days after LMP). The observed number of infants born with congenital rubella syndrome in this group (0/82) gives a maximum theoretical risk of 4.4% based on the binomial distribution.

Three of the women notified to the UK study gave birth to stillborn infants, and another had a late spontaneous abortion, but there was no evidence to suggest that rubella vaccination was the cause. Furthermore, two of the stillbirths were reported retrospectively by obstetricians because they were adverse outcomes following inadvertent rubella vaccination in pregnancy. Studies elsewhere have not identified an increased risk of stillbirth or miscarriage associated with rubella vaccination in pregnancy.

The findings from this study reinforce the current advice[3] that there is no evidence that rubella vaccine is teratogenic, and therefore no reason for routinely recommending termination of pregnancy following inadvertent vaccination near to the time of conception. However, the UK study is continuing in view of the small numbers followed up after vaccination in the period when the risk of damage is likely to be greatest (i.e. one week before conception to four weeks after).

Clinicians caring for women known to have been immunised against rubella at any time during pregnancy or up to one month before conception, and who are continuing with the pregnancy, are asked to contact Gill Jones at the RVPS, Epidemiology and Biostatistics Unit, Division of Public Health, Institute of Child Health, 30 Guilford Street, London WC1N 1EH; telephone 071 242 9789 extension 2606.

Table 2 Prevalence of congenital rubella syndrome (CRS) in live infants born to susceptible women who received rubella vaccine during pregnancy

Country	Vaccinated within 3 months of conception or during pregnancy (all vaccine types)	Subset vaccinated from 1 week before to 4 weeks after conception (RA27/3 only)	Abnormalities compatible with CRS
USA[5]	307	74	0/307
West Germany[6,7]	143	–	0/143
Sweden[7]	5	–	0/5
UK	37	8	0/37
Total	492	82	0/492

References

1. Sheppard S, Smithells RW, Dickson A, Holzel H. Rubella vaccination and pregnancy: preliminary report of a national survey. BMJ 1986; 292: 727.
2. Miller E, Waight PA. Vurdien JE, White JM, Jones G, Miller BHR, et al. Rubella surveillance to December 1990: a joint report from the PHLS and National Congenital Rubella Surveillance Programme. Communicable Disease Report 1991; 1: R33-7.
3. Department of Health. Immunisation against infectious disease. London: HMSO, 1990.
4. Public Health Laboratory Service. TORCH screening reassessed. London: PHLS, 1990.
5. MMWR. Rubella vaccination during pregnancy - United States 1971-1988. 1989; 38: 289-93.
6. Enders G. Rubella antibody titers in vaccinated and nonvaccinated women and results of vaccination during pregnancy. Rev Infect Dis 1985; 7 Suppl 1: S103-107.
7. Banatvala J, Best J. Rubella vaccines. In: Zuckerman AJ, editor. Recent developments in prophylactic immunisation. Dordrecht: Kluwer Academic Publishers; 1989.

P A Tookey MSc
B H R Miller BSc
G Jones MSc
C S Peckham FRCP FFPHM
Epidemiology and Biostatistics Unit,
Institute of Child Health, London.

The COVER scheme: a survey of immunisation coordinators

SJ Gillam, N T Begg

Introduction

The COVER (Cover of vaccination evaluated rapidly) scheme was started in 14 districts in 1987. The aim was to improve vaccination cover by providing immunisation coordinators in each health district with relevant, timely information[1]. Quarterly data have been obtained from districts, analysed, collated, and promptly returned to them. There has been a steady improvement in immunisation coverage over the last four years and more than 195 (95%) districts are now involved in England, Wales and Northern Ireland. Among districts participating in the COVER scheme, the mean increase in uptake for third diphtheria, third pertussis and measles / MMR vaccines was 8%, 16% and 20%, respectively, between May 1987 and May 1991. Several initiatives are likely to have contributed to these increases, for example, the introduction of MMR in October 1988, incentives under the new general practitioner contract of April 1990 and, most recently, the accelerated schedule for primary immunisation.

As part of an ongoing review of the COVER scheme, it was decided to seek information from all participants. 203 immunisation coordinators in England, Wales and Northern Ireland were sent a questionnaire to ascertain their views on the scheme, suggestions for its improvement, uses to which the data are put, and factors contributing to changes in uptake.

Results

One hundred and seventy-three replies were received, a response rate of 85%. All but 3 were participants in the scheme. 97% of respondents have found the scheme helpful. Only 4% felt that supplying data added greatly to their workload.

Evaluation of the COVER scheme

COVER data is frequently used in annual public health reports, in feedback to service providers, in teaching presentations and reports to managers (Table 1). Many respondents cited its value for planning purposes; its use in in press releases and newsletters.

Table 1 Frequency of use of COVER data

	Regularly	Occasionally	Never
Annual public health reports	67%	14%	20%
Teaching presentations	41%	53%	7%
Feedback to service providers	69%	27%	4%
Reports to managers	59%	36%	5%

The majority (89%) of respondents preferred to continue receiving quarterly reports although eight requested annual reports in addition and 88% requested graphical as well as tabulated data. While over half of the respondents wanted further statistical analysis only five made specific suggestions. These included comparison with other districts' coverage and more detailed analysis of long-term trends. Fifty-eight percent would like reports produced by the Family Health Service Authority (FHSA) as well as the District Health Authority (DHA). Several respondents who did not want FHSA reports were working in coterminous districts or were already supplying their FHSA with COVER data.

Factors contributing to improved vaccine coverage

Local initiatives: the feedback of data to service providers was perceived as the most important local initiative (Table 2). 65% of respondents considered that this contributed a great deal to raising uptake rates. Upgrading of the child health computer system and the dissemination of local policy guidelines were also rated. Only a minority of districts run an immunisation advisory clinic or a domiciliary immunisation service. Other initiatives mentioned included the establishment of district immunisation groups and the value of regular training sessions for general practitioners, health visitors and clinical medical officers.

Table 2 Perceived contribution of local initiatives to improvements in the immunisation rates of respondents' districts over the past 3 years

	Major	Minor	Not at all	Not known
Local publicity campaigns	19%	53%	8%	19%
Changes to child health computer system	40%	31%	15%	15%
Dissemination of local guidelines or policy	45%	35%	2%	18%
Immunisation advisory clinic	11%	24%	7%	58%
Domiciliary immunisation service	7%	27%	10%	56%
Feedback of immunisation data to service providers	65%	23%	1%	11%

National initiatives: the introduction of MMR vaccine was perceived as the most important national initiative (Table 3). The new general practitioner contract, the accelerated immunisation schedule, publication of the 'green book'[2], the appointment of immunisation coordinators and the COVER scheme were felt to have contributed to a similar degree in raising uptake rates. The Health Education Authority's campaigns and material were felt to have contributed to a lesser extent.

Table 3 Perceived contribution of national initiatives to improvements in the immunisation rates of respondents' districts over the past 3 years

	Major	Minor	Not at all	Not known
1990 GP contract	46%	39%	7%	8%
Introduction of accelerated immunisation schedule	39%	45%	4%	13%
Introduction of MMR	72%	24%	1%	3%
HEA campaigns or material	5%	58%	17%	20%
New edition of immunisation guidelines[2]	43%	47%	2%	8%
Appointment of immunisation coordinator	46%	38%	2%	13%
COVER scheme	40%	45%	7%	8%

Discussion

It is possible that the views of non-responders differed from those who replied to this survey. The likeliest reason for failure to reply is a recent change of personnel. Nevertheless, the COVER scheme has clearly been of help to immunisation coordinators in their work. The districts that have achieved the largest increases in immunisation coverage over the last three years were using COVER data more regularly and comprehensively than the other districts. Once established, the provision of quarterly data does not add substantially to the workload of immunisation coordinators. However, several districts pointed out that they were unsure how onerous the task was for their information department. COVER data was used in various ways and all but 20% of the respondents supply data for their district's annual public health report. The absence of communicable disease information from many annual reports has been noted in the past (R Mayon-White, personal communication).

The survey suggested that the timeliness of COVER data is particularly valued by immunisation coordinators. Information must be up-to-date for feedback to service providers to be effective. In 1986, less than half the districts were providing feedback of immunisation data to general practitioners and community medical officers[3]. The proportion of respondents now doing so has reached 96%.

Many immunisation coordinators would like to receive data in graphical form and would value data for local FHSAs as well as DHAs. Several sets of data may need to be provided. Conflicts may arise where several DHAs with differing coverage rates lie within a single FHSA. The separation of purchasers and providers adds a further dimension to such disparities. Coverage targets are easily incorporated into contracts. According to some respondents, COVER data are already being used to monitor the fulfilment of contractual obligations. The amalgamation of DHAs and FHSAs may obviate the need for separate figures in future.

The COVER scheme is one among several initiatives that have contributed to the dramatic rise in immunisation coverage over the last four years. Immunisation coordinators believe that the introduction of MMR has had the greatest impact on coverage rates. Introduction of the new general practitioners' contract and the accelerated schedule for immunisation have had a more recent effect on coverage figures. A higher proportion of districts providing a domiciliary immunisation service had obtained large recent increases in uptake. In future, such outreach work is likely to play an increasing part in maintaining uptake rates as they approach full coverage.

Conclusion

Ninety-seven percent of immunisation coordinators report that the COVER scheme is helpful to their work. It is widely used in annual public health reports, teaching, reports to managers and feedback to service providers. The introduction of MMR vaccine is perceived by immunisation coordinators as the most important initiative contributing to the recent improvement in immunisation coverage. The continued commitment of immunisation coordinators will be essential to sustain these improvements.

References

1. Begg NT, Gill ON, White JM. COVER (Cover of vaccination evaluated rapidly). Description of the England and Wales scheme. Public Health 1989; 103: 81-9.
2. Department of Health, Welsh Office and Scottish Home and Health Department. Immunisation against infectious disease. London: HMSO, 1990.
3. Begg NT, White JM. A survey of pre-school immunisation programmes in England and Wales. Community Medicine 1988; 10: 344-50.

S J Gillam MRCP
N T Begg MFPHM
Immunisation Division
Communicable Disease Surveillance Centre

‘COVER’ (Cover of vaccination evaluated rapidly): 18

The COVER programme, a scheme for the rapid evaluation of vaccine coverage, started in January 1987 with 14 districts contributing data (*Communicable Disease Report* 1987; (12): 3-6). By February 1991, the number of participating districts had increased to 192. This eighteenth quarterly report includes follow-up information from 191 of these, as well as a further 3 districts, ie, 194 in all.

Methods

The data were collected at the beginning of May 1991. Vaccination data were requested for quarterly cohorts whose youngest member had reached the target ages for completion of immunisation: 18 months for the third dose of diphtheria (D3) and pertussis (P3) vaccines and 24 months for measles. For D3 and P3, data were also requested for quarterly cohorts whose youngest member had reached a lower target age of 12 months. The cohorts studied were those born in January to March 1990 (for D3 and P3 by 12 months), July to September 1989 (for D3 and P3 by 18 months) and January to March 1989 (for measles by 24 months).

Results

Altogether, 194 districts participated from 14 English regions, Wales and Northern Ireland. Data were available for every district in nine English regions, Wales and Northern Ireland. In all other regions (except Trent) at least 80% of districts participated. The average cover (Table) by 12 months was 90% for D3 (district range 57–98%) and 85% for P3 (district range 55–94%). Cover by 18 months was 91% for D3 (district range 59–98%) and 86% for P3 (district range 58–95%). For measles, average cover by 24 months was 90% (district range 58–98%).

Comment

We report data from 194 of 203 (96%) districts in England, Wales and Northern Ireland. Vaccine coverage has improved by 2% for P3 (18 months) and 1%for both D3 (18 months) and measles (24 months) since the previous report. Ninety per cent coverage has now been achieved by 145 districts (for D3 at 18 months), 52 districts (for P3 at 18 months) and 125 districts (for measles at 24 months).

It is likely that some of the children evaluated at 12 months were immunised according to the new accelerated schedule of immunisation; others, however, may have remained on the previous schedule. The 12 months' cohort to be evaluated in the next COVER report should have been immunised exclusively under the new schedule and will provide an assessment of its impact.

The Government's health strategy[1] has proposed a target vaccine coverage of 95% by the year 1995. Based on current trends, it seems likely that this can be achieved nationally for all antigens. Nevertheless, some districts (particularly in the Thames regions) are still falling well short of the 90% target, and further efforts are required in these areas.

Reference

1. Department of Health. The health of the nation. London: HMSO, 1991.

S G Handford BSc
JM White BSc
N T Begg MFPHM
Immunisation Division
Communicable Disease Surveillance Centre

Table Diphtheria and pertussis vaccination at 12 and 18 months, and measles vaccination at 24 months: May 1991

Region	Number of participating districts (total)	Coverage at 12 months: % had D3 by evaluation date	Coverage at 12 months: % had P3 by evaluation date	Coverage at 18 months: % had D3 by evaluation date	Coverage at 18 months: % had P3 by evaluation date	Coverage at 24 months: % had measles by evaluation date
England						
Northern	15 (16)	92 *(82–98)	86 *(77–94)	93 *(87–98)	87 *(80–92)	92 *(82–98)
Yorkshire	17 (17)	92 (87–97)	87 (82–92)	93 (87–98)	88 (82–93)	91 (85–95)
Trent	9 (12)	91 (85–96)	87 (82–92)	90 (86–95)	86 (82–92)	89 (81–93)
E Anglia	8 (8)	94 (86–98)	89 (83–93)	95 (89–97)	89 (84–92)	94 (86–98)
NW Thames	12 (13)	89 (78–96)	85 (76–92)	88 (76–97)	84 (73–93)	86 (72–93)
NE Thames	16 (16)	86 (75–96)	82 (70–93)	90 (84–96)	85 (77–93)	87 (75–96)
SE Thames	15 (15)	86 (57–96)	82 (55–93)	86 (59–96)	82 (58–92)	85 (58–96)
SW Thames	13 (13)	88 (71–97)	85 (68–94)	89 (73–97)	85 (71–95)	86 (71–96)
Wessex	10 (10)	93 (88–97)	88 (84–93)	95 (88–98)	90 (83–95)	93 (89–98)
Oxford	8 (8)	89 (79–95)	86 (76–92)	92 (83–96)	88 (79–91)	92 (86–96)
S Western	11 (11)	93 (88–96)	89 (86–92)	95 (90–98)	90 (85–93)	93 (89–97)
W Midlands	22 (22)	89 (76–97)	84 (72–91)	91 (82–96)	85 (78–91)	89 (75–95)
Mersey	8 (10)	89 (83–94)	83 (76–90)	91 (85–96)	84 (77–90)	91 (83–95)
N Western	17 (19)	91 (80-95)	86 (73–91)	92 (83–97)	86 (77–92)	91 (75–96)
Wales	9 (9)	92 (88–95)	82 (77–88)	92 (91–97)	82 (77–92)	88 (86–96)
N Ireland	4 (4)	92 (90–94)	87 (85–90)	93 (91–95)	86 (84–89)	91 (88–94)
Total	194 (203)	90 (57–98)	85 (55–94)	91 (59–98)	86 (58–95)	90 (58–98)

* The district range is given in brackets

Toxic shock syndrome in the UK: 1985-1990

R R Marples, A A Wieneke

Introduction

This summary reviews cases of toxic shock syndrome (TSS) ascertained during the years 1985-90 in the United Kingdom. It includes cases from 1985-86 previously reported in the *Communicable Disease Report* [1]. Earlier results were reported by de Saxe et al[2]. Cases were classified as confirmed or probable TSS from data supplied initially or from questionnaires sent out by the reference laboratory along with phage typing or toxin detection results on strains of *Staphylococcus aureus* submitted for study.

The toxic shock syndrome was first defined by Todd in 1978[3]. Most cases occur in women during menstruation. The clinical presentation includes myalgia, headache, fever, vomiting and diarrhoea, leading to hypovolaemic shock and the development of diffuse erythema and conjunctival inflammation, within a few hours. A similar condition had been recognised earlier as staphylococcal scarlet fever[4]. Non-menstrual cases occur in association with localised *S. aureus* infections, e.g. abscesses, osteomyelitis, wound infections and pneumonia.

The diagnostic criteria were refined by the Centers for Disease Control (CDC)[5] in Atlanta to investigate an apparent epidemic in 1980[6]. They required the presence of high fever, erythematous rash, desquamation, marked hypotension and the failure of three independent organ systems. Chesney et al[7], however, suggested that a minimum of three of the four basic criteria, with exclusion of confounding alternative diagnoses, was acceptable, particularly in early deaths. We have used the CDC criteria for **confirmed** cases and have diagnosed patients in whom one of the criteria does not occur as cases of **probable** toxic shock syndrome.

Two toxins have been implicated in toxic shock syndrome: toxic shock syndrome toxin-1 (TSST-1), formerly known as enterotoxin F[8] or pyrogenic toxin C[9], and produced by strains of *S. aureus* of phage group I is the most frequent, but staphylococcal enterotoxin B (SEB)[10], produced mainly by strains of phage group V, has also been reported. The exfoliative toxins implicated in staphylococcal scalded skin syndrome may also play a part in severe cases of toxic shock syndrome associated with a rash.

Results

During the six years under review, 822 cases of various toxic staphylococcal diseases (not food poisoning) yielded 868 strains of *S. aureus* which have been phage typed and tested for enterotoxin and TSST-1 production. The response to the questionnaire has been incomplete but 106 patients were accepted as having confirmed or probable toxic shock syndrome. One hundred and twelve strains of *S. aureus* were isolated from these 106 patients.

Table 1 shows the numbers of patients for which both clinical information and laboratory studies were available and where a diagnosis of confirmed or probable toxic shock syndrome could be supported on the evidence available to us. The table indicates that it is rare for more than 20 cases to be validated in the United Kingdom per year. In 315 cases, adequate data were not available, although a diagnosis of TSS was often made. If these additional cases are included the total number is estimated to approach 40 per year. There is, however, no indication of any change in the number of cases diagnosed annually during this 6 year period. The association of toxic shock syndrome with menstruation and tampon usage is well accepted[6], although this presentation is not exclusive. There has been little change during the review period in the proportion of non-menstrual toxic shock syndrome cases.

Table 2 Relationship of toxin production to presentation

Toxin	Presentation		Total
	Menstrual	Non-menstrual	
TSST-1	55	26	81
SEB	–	11	11
Other	–	8	8
None	4	8	12
Total	59	53	112

Table 3 Relationship of phage group to presentation

Phage group	Presentation		Total
	Menstrual	Non-menstrual	
I	40	23	63
V	1	12	13
Other	9	14	23
NT*	9	4	13
Total	59	53	112

* NT non-typable

Table 2 relates the clinical presentation to the production of TSST-1, enterotoxin B (SEB) and other enterotoxins. TSST-1 was significantly associated with a menstrual presentation and SEB production with a non-menstrual presentation (Chi square = 30.5, $p < 0.01$). Table 3 compares the phage group of the strains isolated for the two forms of presentation. The association of phage group V strains with a non-menstrual presentation is the only significant finding (Chi square = 16.6, $p < 0.01$). Taking these two tables together, it is clear that

Table 1 Toxic shock syndrome (confirmed and probable cases)

	1985	1986	1987	1988	1989	1990	Total
All cases	19	23	15	18	11	20	106
All deaths	2	4	2	2	3	3	16
Menstrual cases	7	11	7	9	9	15	58
Menstrual deaths	–	1	–	1	2	1	5

TSST-1 is produced mainly by strains of phage group I whereas SEB is produced by strains of phage group V. However, TSST-1 producing group I strains can be associated with a non-menstrual presentation. Strains of phage type 95 associated with production of enterotoxin C accounted for the third most frequently identified group.

Tampon usage was reported infrequently but there was an excess of brands without an introducer and the ages of the menstrual cases were low. Thirty-six (67%) of the 54 menstrual patients whose age was known were less than 20 years old compared with 21 (43%) of the non-menstrual cases. Thus, inexperience in tampon use and personal hygiene may be important.

Comment

About 20 cases of confirmed and probable toxic shock syndrome are identified each year. Taking the incomplete data received for many cases into account, it seems likely that some 40 cases may present each year in the United Kingdom. It is unlikely that more than 2-4 deaths occur annually as such an event, particularly in teenage women, is dramatic and often involves the coroner.

Most cases of toxic shock syndrome associated with menstruation (and its management) are mediated by TSST-1 produced by phage group I strains of *S. aureus*. The syndrome in non-menstrual cases is often associated with phage type V enterotoxin B (SEB) producing strains. Staphylococcal exotoxins may also be involved in septicaemia[11] and infections complicating burns[12]. Better diagnostic criteria will help to distinguish their importance in these syndromes. Prospective studies are required to assess the effect of education on personal hygiene in preventing tampon associated disease.

References

1. Marples RR. Toxic shock and allied syndromes in 1985 and 1986. Communicable Disease Report 1987; (30): 3-4. Internal publication of the Public Health Laboratory Service, London.
2. de Saxe MJ, Hawtin P, Wieneke AA. Toxic shock syndrome in Britain - epidemiology and microbiology. Postgrad Med J 1985; 61 Suppl 1: 5-21.
3. Todd J, Fishaut M, Kapral F, Welch T. Toxic-shock syndrome associated with phage-group-I staphylococci. Lancet 1978; ii: 1116-8.
4. Waldvogel FA. Staphylococcus aureus (including toxic shock syndrome). In: Mandel GL, Douglas RG, Bennett JE, editors. Principles and practice of infectious diseases. New York: Churchill Livingstone, 1990: 1489-1510.
5. MMWR 1990 19 October; 39 (RR-13): 38-9.
6. Shands KN, Schmid GP, Dan BB, Blum D, Guidotti RJ, Hargrett NT, et al. Toxic-shock syndrome in menstruating women. N Engl J Med 1980; 303: 1436-42.
7. Chesney PJ, Bergdoll MS, Davis JP, Vergeront JM. The disease spectrum, epidemiology, and etiology of toxic-shock syndrome. Annu Rev Microbiol 1984; 38: 315-38.
8. Bergdoll MS, Crass BA, Reiser RF, Robbins RN, Davis JP. A new staphylococcal enterotoxin, enterotoxin F, associated with toxic-shock syndrome *Staphylococcus aureus* isolates. Lancet 1981; i: 1017-21.
9. Schlievert PM, Shands KN, Dan BB, Schmid GP, Nishimura RD. Identification and characterization of an exotoxin from *Staphylococcus aureus* associated with toxic-shock syndrome. J Infect Dis 1981; 143: 509-16.
10. Schlievert PM. Staphylococcal enterotoxin B and toxic-shock syndrome toxin-1 are significantly associated with non-menstrual TSS. Lancet 1986; i: 1149-50.
11. Arbuthnott JP, Coleman DC, de Azavedo JS. Staphylococcal toxins in human disease. Soc Appl Bacteriol Ser 1990; 19: 101S-7S.
12. Frame JD, Eve MD, Hackett ME, Dowsett EG, Brain AN, Gault DT, Wilmshurst AD. The toxic shock syndrome in burned children. Burns 1985; 11: 234-41.

R R Marples FRCPath
Division of Hospital Infection
A A Wieneke Drs
Food Hygiene Laboratory
Central Public Health Laboratory

Submission of manuscripts to the *Communicable Disease Report*

Authors are invited to submit papers on all aspects of surveillance, prevention and control of communicable diseases to be considered for publication in the *Communicable Disease Report*.

Manuscripts and their references should conform to the "Uniform requirements for manuscripts submitted to biomedical journals" (ie, the Vancouver style)[1].

1. International Committee of Medical Journal Editors. Uniform requirements for manuscripts submitted to biomedical journals. BMJ 1991; 302: 338-41.