Lesson of the Week

Deaths associated with intrauterine contraceptive devices in the United Kingdom between 1973 and 1983

P A SMITH, C J ELLIS, R A SPARKS, J GUILLEBAUD

Almost half a million women currently use the intrauterine contraceptive device in the United Kingdom (estimate of Family Planning Association based on data from manufacturers, clinics, and family practitioner committees). The complications associated with these devices are well described, and should be outlined to any woman considering their use.¹ Rarely, however, serious and even fatal complications occur, and in many cases avoidable factors are present.

We describe four deaths associated with the use of an intrauterine contraceptive device and one tragic near fatality.

Case reports

Case 1—In 1978 a 26 year old woman presented to her general practitioner with mild lower abdominal pain, for which he found no cause. Two days later she complained of nausea and occasional vomiting but not of pain. Abdominal examination was again unremarkable, as it was the next day, when she complained of vague epigastric symptoms and was seen by a third doctor. Twenty four hours later, after reporting only persistent anorexia and lethargy, the patient died in her own home. Seven months previously she had had a copper 7 intrauterine contraceptive device (Gravigard) inserted. The tail of this device had disappeared by the time of an examination at two months, but its presence within the pelvis had been confirmed by x ray examination, and pelvic examination at that time showed no abnormality. The patient died while waiting for curettage to remove the device. At necropsy, a loop of terminal ileum had strangulated round the thread attached to the perforated intrauterine contraceptive device, which had adhered to the lateral pelvic wall. The loop of bowel was isolated deep in the pouch of Douglas, accounting for the minimal symptoms and the lack of signs in the abdomen. A pelvic examination had not been performed.

Case 2—In 1976 a 42 year old woman complained to her general practitioner of recent heavy and painful periods. A Saf-T-coil had been in situ for about 12 months. No evidence of pelvic disease was found on examination, and in an attempt to alleviate her symptoms the Saf-T-coil was removed and replaced with a copper 7 (Gravigard). Two days later she was admitted to hospital with gross pelvic sepsis, which progressed to peritonitis and septicaemia and resulted in her death.

King's College Hospital, London SE5 8RX

P A SMITH, MRCOG, registrar in obstetrics and gynaecology

Department of Communicable and Tropical Diseases, East Birmingham Hospital, Birmingham B9 5ST

C J ELLIS, MRCP, consultant physician

Queen Elizabeth Medical Centre, Birmingham B15 2TG

R A SPARKS, MRCOG, senior lecturer in genitourinary medicine

Margaret Pyke Centre for Study and Training in Family Planning, London W1V 5TW

J GUILLEBAUD, FRCSE, MRCOG, medical director

Correspondence to: Dr P A Smith.

Doctors should remember when managing sick young women that deaths may occasionally be associated with intrauterine contraceptive devices

She had been receiving steroid treatment for rheumatoid arthritis for the preceding four years, but details of treatment could not be obtained. It was suggested at the inquest into her death that a vaginal infection was probably present at the time of reinsertion of the intrauterine contraceptive device, and that her resistance to the pelvic infection that developed during the next 24 hours was very much impaired by the systemic steroid treatment.

Case 3—In 1982 a previously healthy 34 year old woman became unwell with vague pains and feverishness. The next day she developed lower abdominal pain and visited her general practitioner, mentioning that she thought her intrauterine contraceptive device might be causing her symptoms. He performed a vaginal examination and reassured her that he could find no evidence of infection. Her symptoms persisted, and two days later she experienced pain, stiffness, and coldness in her right leg. A few hours later all her limbs were cold and cyanosed, and she was admitted to hospital. She had had two pregnancies, both necessitating caesarean section. She had used an intrauterine contraceptive device for several years, the device being changed every two years according to the manufacturers' instructions. The present device, a copper 7 (Gravigard) had been inserted eight weeks before her symptoms began. She had a normal period two weeks after insertion, but two weeks later further vaginal bleeding occurred, which continued until her admission; this was heavy at first but had almost ceased by the time her symptoms started. On admission she was alert and had no fever. Her limbs were cyanosed and cold. Blood pressure was 100/80 mm Hg supine but was unrecordable when she attempted to stand. On vaginal examination the pelvic organs were normal, with no tenderness or masses and no discharge through the cervical os. The tail of the intrauterine contraceptive device was just visible and the device was easily removed. Septicaemic shock was diagnosed and treatment started immediately with parenteral antibiotics. Despite the rapid intravenous infusion of fluids, she died within five hours of admission. Gram positive cocci were seen on a smear from the intrauterine contraceptive device and a group A β haemolytic streptococcus was grown from the device and from peripheral venous blood. At necropsy the heart valves were not diseased. The uterus and tubes appeared normal to the naked eye but numerous Gram positive cocci were seen in the endometrium, suggesting that this was the portal of entry of the infection.

A further case of a woman who apparently died with septicaemic shock one month after insertion of an intrauterine contraceptive device has been reported, but no further details are available. This woman was the only fatality of the four device users from whose blood a group A β haemolytic streptococcus was isolated that was reported to the Communicable Diseases Surveillance Centre at Colindale between 1975 and 1981.²

Although non-fatal, we report one more previously unrecorded case, the details of which come from an uncorroborated letter from the patient's mother to one of us (JG). A 32 year old woman had had an intrauterine contraceptive device inserted after the birth of her third child. One year later she became pregnant with the device in situ. She

was admitted for termination of the pregnancy at 10 weeks' gestation. One hour before the operation she complained of severe one sided pelvic pain, but the attendant nurse is said to have ascribed this to "nerves." After the operation she collapsed with severe chest and abdominal pain and was admitted to the intensive therapy unit. When tests for suspected pulmonary embolism proved negative she was returned after two days to the general ward. She subsequently lapsed into coma and finally had a laparotomy (for "suspected ruptured appendix") six days after admission, when a massive haemoperitoneum due to a ruptured tubal ectopic pregnancy was found. The patient is now severely brain damaged, practically blind, and quite incapable of caring for her three young children.

Comment

In 1975 De Swiet *et al* reported a case of bacterial endocarditis after insertion of an intrauterine contraceptive device,³ and in 1977 a case of septicaemia after insertion of an intrauterine contraceptive device was reported.⁴ Extrauterine pregnancies associated with intrauterine contraceptive devices have also led to deaths. A ruptured primary intraperitoneal pregnancy was reported in 1974.⁵ During the decade under discussion there have been two *Reports on Confidential Enquiries into Maternal Deaths in England and Wales*, for 1973-5 and for 1976-8.^{6,7} Three deaths due to ectopic pregnancy associated with intrauterine contraceptive devices were reported in the first of these, but there were none in the more recent report.

It is important to emphasise the extreme rarity of these cases. In the decade in question (1973-83) about 400 000-600 000 women were using intrauterine contraceptive devices and no cases additional to these discussed have been reported to the medicines division of the Department of Health and Social Security.

It is debatable whether the deaths and the case of brain damage due to extrauterine pregnancy should even be included. Although intrauterine contraceptive devices are associated with pelvic infection and hence the risk of tubal damage,⁸ statistical analysis suggests that intrauterine contraceptive devices may actually reduce the rate of extrauterine pregnancy by reducing the number of fertilisations that occur.⁹ Nevertheless, as the number of intrauterine implantations is so much more reduced, up to 5% of all pregnancies in women using intrauterine contraceptive devices are extrauterine. Whatever the aetiology, therefore, in practice the safest axiom is "All pregnancies in the presence of an intrauterine contraceptive device are ectopic until proved otherwise." Had this rule been followed in the non-fatal case reported, the disastrous outcome might have been avoided.

The case of strangulated ileum after perforation of the uterus by the intrauterine contraceptive device (case 1) underlines several principles in the management of patients with lost intrauterine contraceptive device threads: the unhelpfulness of a plain abdominal x ray examination in the absence of a uterine marker; the necessity for patients with "lost" threads to be examined, and treated expeditiously, most particularly if they develop abdominal pain; and the need for alternative contraception, as many cases of perforation present with an avoidable pregnancy. This subject has been reviewed recently.^{10,11}

There is little doubt that the initial trauma causing perforation of the uterus occurs at the time of insertion.¹¹ This is also the time of maximum risk for introducing a severe infection, as shown in case 2 and several reports.^{3,4}

It would appear from case 2 that systemic corticosteroid treatment, or any other form of immunosuppression, should be considered as a strong relative contraindication to insertion of an intrauterine contraceptive device. Although there are no published series, our experience suggests that cautious use of an intrauterine contraceptive device is acceptable in some cases of valvular heart disease, with antibiotic cover at the time of insertion and removal as recommended in the *British National Formulary* for genitourinary procedures,¹² and advice to the woman to report urgently any symptoms that could signify infection, especially pelvic pain or dyspareunia. A cardiologist opinion seems advisable, however, if it is known in advance that

there is a heart valve lesion (not so in the case reported by De Swiet *et al*³).

Persistent colonisation of the lower uterine cavity is the rule in all women fitted with a device containing a tail or thread¹³—a point which is probably relevant to case 3. This case was unusual in that there was no evidence of pelvic sepsis and a long interval between insertion and the onset of any symptoms necessitating a medical consultation. If the β haemolytic streptococcus was introduced with the intrauterine contraceptive device at insertion then it must have acted as a non-pathogenic colonist for some weeks before invading the endometrium and thence the bloodstream. Alternatively, it could have colonised the vagina some weeks after insertion and then ascended the intrauterine contraceptive device tail to the uterus.

Three more cases have been reported from outside the United Kingdom of fatal non-gonococcal septicaemia in non-pregnant women in the presence of an intrauterine contraceptive device without any other obvious predisposing factor, and in which the interval between insertion and first symptoms was greater than two months.^{14,15} A further fatality was due to pelvic actinomycosis.¹⁶ So far as we are aware no death due to pelvic actinomycosis has been reported from the United Kingdom, nor have we found reports of fatalities due to infected second trimester abortion in the presence of an intrauterine contraceptive device, as were described in the United States in the early 1970s.¹⁷ These should now be avoidable if the current advice to remove devices in early pregnancy is followed routinely.¹⁸

ADDENDUM—A further death associated with intrauterine contraceptive devices has been reported in the United Kingdom.¹⁹ A woman who used a Dalkon shield had mitral stenosis and a bicornuate uterus and died of renal and cardiac failure after *Streptococcus pneumoniae* peritonitis and salpingitis.

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