volume was slowly increased, bronchial rupture occurred after the injection of 15 ml of air.

In order to avoid potentially fatal complication, tracheal and bronchial cuffs should be inflated with the minimum volume necessary. The use of a 2 ml syringe to inflate the bronchial cuff may prevent accidental over-inflation. If more than 3 ml are required, the tube should be repositioned. The use of saline, or the ventilating gas to inflate the cuff will prevent N_2O diffusion, and if air is used the cuff should be checked frequently to prevent overdistension. The use of a cuff pressure limitation device, such as the Cardiff Cuff Controller[9], offers a further safeguard. Ideally, cuff pressure should not exceed 30 cm H_2O[7].

References

1 Blanc VF, Tremblay NAG. The complications of tracheal intubation: a new classification and review of the literature. *Anesth Analg* 1974;**53**:202-13

2 Guernelli N, Bragaglia RB, Briccoli A, Mastrorilli M, Vecchi R. Tracheobronchial rupture due to cuffed Carlens tubes. *Ann Thorac Surg* 1979;**28**:66-8

3 Foster JMG, Lau OJ, Alimo EB. Ruptured bronchus following endobronchial intubation. *Br J Anaesth* 1983;**55**:687-8

4 Heiser M, Steinberg JJ, MacVaugh H, Klineberg PL. Bronchial rupture, a complication of use of the Robert Shaw double-lumen tube. *Anesthesiology* 1979;**51**:88

5 Smith BAC, Hopkinson RB. Tracheal rupture during anaesthesia. *Anaesthesia* 1984;**39**:894-8

6 Burton NA, Watson DC, Brodsky JB, Mark JBD. Advantages of a new Poly Vinyl Chloride double-lumen tube in thoracic surgery. *Ann Thorac Surg* 1983;**36**:78-84

7 Seegobin RD, Van Hasselt GL. Endotracheal cuff pressure and tracheal mucosal blood flow: endoscopic study of effects of four large volume cuffs. *BMJ* 1984;**288**:965-8

8 Revenas B, Lindholm CE. Pressure and volume changes in tracheal tube cuffs during anaesthesia. *Acta Anaesth Scand* 1976;**20**:321-6

9 Willis BA, Latto IP, Dyson A. Tracheal tube cuff pressure - clinical use of the Cardiff cuff controller. *Anaesthesia* 1988; **43**:312-14

(Accepted 14 August 1991)

HIV infection in a patient with Meyer-Rokitansky-Küster-Hauser syndrome

P D Kell MRCOG[1] **S E Barton** MD MRCOG[1]
D K Edmonds FRCOG[2] **F C Boag** MRCP[1]
[1]*John Hunter Clinic, St Stephen's Clinic, 369 Fulham Road, London SW10 9TH and*
[2]*Queen Charlotte's Hospital, Goldhawk Road, London W6 0XG*

Keywords: HIV infection; Meyer-Rokitansky syndrome

Vaginal intercourse is the commonest route of transmission of HIV infection worldwide, according to the WHO[1]. The site of viral entry in the genital tract is poorly understood. This case report suggests that the vagina alone can be a route of HIV infection. Thus all women engaging in vaginal intercourse need to take precautions to prevent HIV transmission - even if they do not have a cervix.

Case report

A 31-year-old woman presented to a genitourinary medicine (GUM) clinic requesting an HIV antibody test. After counselling, a positive test was obtained and was confirmed by Western blot.

Seven months prior to this presentation she had had 10 episodes of unprotected vaginal intercourse with a man who subsequently had a severe chest infection. Two months later she developed a febrile illness of 2 weeks duration associated with a maculo-papular rash, a headache and buccal and vaginal candidosis. It is presumed that this was an HIV seroconversion illness. The patient denied oral or anal intercourse, injecting drug use, blood transfusions or injections abroad.

A gynaecological history revealed primary amenorrhoea, investigated when she was 18 years old. She had a female karyotype (46XX), a rudimentary uterus, normal fallopian tubes and ovaries (demonstrated by laparoscopy) and a normal intravenous urogram.

On examination the patient was a healthy caucasian female who was 5 feet 7 inches tall, weighed 59 kg, had female secondary sexual characteristics and normal external genitalia. On pelvic examination the vagina was short and blind ending with an absent cervix. Serum samples revealed FSH 7.1 mU/l, LH 26.1 mU/l, prolactin 348 mU/l and oestradiol 203 pml/l. A pelvic and a vaginal ultrasound could not identify a uterus and revealed only one ovary, which contained multiple small follicles. There were no clinical signs of HIV-related illness.

Discussion

This patient has the Meyer-Rokitansky-Küster-Hauser syndrome, which comprises vaginal atresia with other variable Mullerian duct abnormalities[2,3]. The fallopian tubes and ovaries are normal. In this case it is likely that the short vagina has been created by repeated sexual intercourse. The visualization of only one ovary during ultrasonography may have been because in such patients the ovaries tend to be abnormally high and are easily missed. The polycystic ovarian syndrome was also demonstrated by the ultrasound appearance and the LH : FSH ratio being greater than 3 : 1.

Transmission of HIV is presumed to have been through vaginal intercourse. This may have been facilitated by the short vagina being excessively stretched and traumatized during intercourse. Trauma during sexual intercourse can lead to epithelial disruption and thus to an increased susceptibility to infection. However, an accurate definition of what constitutes traumatic sex is lacking. A study of wives[4] of male HIV-infected transfusion recipients found that older women were more likely to seroconvert. A possible reason for this is microtrauma during sexual intercourse due to atrophic vaginitis. A similar mechanism has been suggested for the association between the use of vaginal tightening agents, such as stones, by multi-parous women in certain African tribes and an increased incidence of HIV infection[5]. Devices such as tampons might also abrade the vagina and facilitate transmission; this may explain the finding[6] that the seropositive wives of haemophiliac men had a higher rate of tampon use than did seronegative partners. Coitarche, which can be associated with trauma, has also been previously noted as a risk factor for HIV transmission[8].

This case represents the first patient in which hetero-sexual transmission of HIV has been associated with

Correspondence to: Dr Fiona Boag, Department of HIV/GU Medicine, St Stephen's Clinic, 369 Fulham Road, London SW10 9TH

vaginal atresia. The study of such cases will continue to shed further light on the mechanisms of HIV transmission. The message of safer sex must be delivered to all, including women without a cervix who may erroneously consider themselves impervious to HIV.

References

1 Chin J. VII International Conference on AIDS. Florence, June 1991
2 Meyer CAJ. *Jahrb Gebur Augenh* 1829;13:525
3 Kuster H. Uterus bipartitus solidus rudimentarius cum Vagina solida. *Zschr Gebur Gynak* 1910;67:692-718
4 Peterman TA, Stoneburner RL, Allen JR, Jaffe HW, Curran JW. Risk of human immunodeficiency virus transmission from heterosexual adults with transfusion-associated infection. *JAMA* 1988;259:55-8
5 Dallabetta G, Miotti P, Chiphangwi J, Liomba G, Saah A. Vaginal tightening agents as risk factors for acquisition of HIV. V International Conference on AIDS, Abstract Th C 574. Montreal 1989
6 Goedert JJ, Eyster ME, Ragni MV, Biggar RJ, Gail MH. Rate of heterosexual HIV transmission and associated risk with HIV antigen. IV International Conference on AIDS, Abstract 4019 Stockholm 1988
7 European Study Group. Risk factors for male to female transmission of HIV. *BMJ* 1989;298:411-15
8 Bouvet E, DeVincenzi I, Ancelle R, Vachon F. Defloration as risk factor for heterosexual HIV transmission. *Lancet* 1989;1:615

(Accepted 30 August 1991)

Spinal cord compression in polyarteritis nodosa

H Rodgers MRCP[1] **D Veale** MD MRCP[2]
P Smith MD MRCP[3] **P Corris** FRCP[2]
[1]*Royal Victoria Hospital,* [2]*Freeman Hospital and* [3]*Newcastle General Hospital, Newcastle upon Tyne*

Keywords: polyarteritis nodosa; subdural haematoma; spinal cord compression

We describe a rare but potentially remediable cause of spinal cord compression.

Case report

A 55-year-old man presented with a dry cough, wheeze, headache, malaise and weight loss. Four months previously he had undergone a nasal septoplasty for recurrent epistaxis. He had been otherwise well apart from a chronic duodenal ulcer for which he was taking ranitidine. On admission he was pyrexial (38°C) with a vasculitic rash, there were signs of right lower lobe consolidation. The initial examination was otherwise normal.

Investigations showed a haemoglobin level of 13.7 g/dl, white cell count of 14.7×10^9/l (no eosinophilia) and an ESR of 79 mm/h. Platelet count and clotting studies were normal. Renal function was impaired with a serum creatinine concentration of 129 μmol/l and urea concentration of 15.1 mmol and urine microscopy revealed haematuria and cellular casts. A chest X-ray confirmed the clinical findings and at bronchoscopy blood was seen in the right middle lobe. No organisms cultured from sputum or blood. Antinuclear antibody, rheumatoid factor, antineutrophil cytoplasmic antibody and hepatitis B serology were negative.

A clinical diagnosis of systemic vasculitis was made and he was treated with pulses of intravenous methylprednisolone (500 mg daily) whilst awaiting a tissue diagnosis from renal and skin biopsies. He initially showed marked symptomatic improvement; in view of this response cyclophosphamide was withheld.

Two days later he developed left monocular blindness associated with a central retinal artery occlusion. He then complained of sudden onset of low back pain radiating down both legs and he rapidly became paraplegic with a sensory level at T9. Urgent myelography showed spinal cord compression at the level of the first lumbar vertebral body. Platelet count and clotting studies were again normal. At operation a subdural haematoma was removed from around the conus and cauda equina. Postoperatively the patient was unrousable with marked neck stiffness and a CT scan showed an intraventricular haemorrhage. He died 12 h postoperatively.

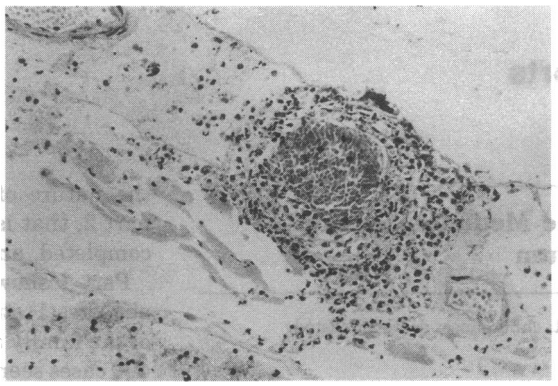

Figure 1. Histology of leptomeningeal vessels around the lumbar cord showing fibromoid necrosy of the vessel walls with infiltration of the adventitia predominantly with neutrophils

At postmortem there was extensive haemorrhage over the brainstem associated with intraventricular haemorrhage and subarachnoid haemorrhage around the lumbar spinal cord. Histology demonstrated extensive vasculitic changes in the small dural and leptomeningeal vessels of the lumbar cord and showed fibrinoid necrosis of the vessel walls with infiltration of the adventitia, predominantly by neutrophils. No atypical infiltrates were seen (Figure 1). Similar changes were seen in the histology of the lungs, kidneys, small bowel and adrenal gland.

Discussion

This case is an example of fulminant systemic vasculitis resulting in multiple small vessel subdural and subarachnoid haemorrhages.

The diagnosis of polyarteritis nodosa was based upon the histological findings of extensive focal acute vasculitic infiltrates and by the absence of granulomatous changes. Careful evaluation of the renal and pulmonary pathology in particular showed no features typical of lymphomatoid or Wegener's granulomatosis. Although lung and nasal involvement in polyarteritis are rare, being more readily associated with the Churg-Strauss (eosinophilic) variant, we conclude that polyarteritis was the most likely diagnosis[1,2].

Correspondence to: Dr Helen Rodgers, Department of Neurology, Royal Victoria Infirmary, Queen Victoria Road, Newcastle upon Tyne NE1 4LP