that the chlamydia antigen acted as a trigger but may not be the entire explanation for her vasculitis.

It is well known that HSP follows infections, particularly those due to streptococcus, varicella, hepatitis and yersinia. Symptoms suggesting infection are present in over half the patients in some series.⁵ Most commonly these consist of acute bronchitis, pharyngitis and flu-like symptoms, sometimes with a distinct interval between the infection and the onset of HSP. A seasonal variation has also been reported which is compatible with infection. The association with chlamydial infection has not been previously described, however.

Renal disease is a feature of HSP and many of the other vasculitides. Renal vasculitis is most often described following viral infections, particularly hepatitis B, but has not been reported previously in association with parvovirus B19 infection. Small vessel vasculitis (leucocytoclastic vasculitis) is the most common vasculitic manifestation associated with acute or chronic infections. The most frequently affected vessels are post-capillary vessels, capillaries and, less often, arterioles. It is generally believed that viral infections, particularly those caused by RNA viruses, seem to be associated with, or to cause, vascu-

litides confined to relatively small vessels. Bacterial infections, on the other hand, tend to involve a spectrum of blood vessels, from small blood vessels to large arteries, while fungal infections are associated with or cause vasculitis that manifests as erythema nodosum or that seems to be associated with large arteries or the aorta. It is noteworthy that several forms of vasculitis have been reported after respiratory infections.⁶

Treatment of systemic vasculitis usually requires systemic steroids, often in high dose. Our first patient responded to low-dose steroid but the second required no treatment. This again would suggest that infection played a significant role as our patients might have expected to have more prolonged and more severe disease if they had not been specifically associated with the infections.

We have, therefore, reported these two cases of infection-related systemic vasculitis; the first case, to our knowledge, has not been previously described in the literature and the second showed a rather unusual urinary sediment abnormality. We feel that chlamydial pneumonias should be added to the long list of microbes causing vasculitis, as misdiagnosis can have serious consequences.

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Ergotism related to a single dose of ergotamine tartrate in an AIDS patient treated with ritonavir

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Summary

We report a rare case of ergotism related to a single dose of ergotamine tartrate in a man with AIDS being treated with ritonavir. He was treated with a prostacyclin analogue and made a complete recovery.

Keywords: ergotism; ergotamine tartrate; AIDS; ritonavir; adverse drug reaction; HIV infection

Ergotism may occur when a low dose of ergotamine or dihydroergotamine is taken in association with another drug which inhibits its hepatic metabolism. These are mostly macrolide agents, dose-dependent inhibitors of the cytochrome P450 isoenzyme CYP 3A4. Troleandomycin and erythromycin are the most potent inhibitors; azithromycin and spiramycin

have not been incriminated.¹ Ritonavir is a potent HIV protease inhibitor.² Clinically significant drug interactions between ritonavir and many other medications have been predicted because of its pharmacokinetic profile.³ We report a case of severe ergotism related to the concurrent administration of a single dose of ergotamine tartrate and ritonavir.⁴

Case report

A 31-year-old HIV-1 infected homosexual man (stage C following *Pneumocystis carinii* pneumonia in December 1994) had been treated with stavudine (40 mg/12 h), lamivudine (150 mg/12 h) and ritonavir (600 mg/12 h) since August 1996, and with cotrimoxazole since December 1994. His CD4 cell count was 819/

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mm³ and plasma HIV-1 RNA measured by reverse-transcriptase polymerase chain reaction amplification was undetectable. He had long suffered from migraines. On 5 December 1997, he complained of pain, paresthesias and cyanosis in both feet. He was admitted to Cochin Hospital on 8 December with severe pain in both legs. Loss of tibial, peroneal, but also radial and ulnar pulses was noted. Achillean deep tendon reflexes were absent. Loss of sensation, pallor and decrease in skin temperature of feet were observed. The patient denied taking any drug. An arterial Doppler test revealed the absence of flow in both tibioperoneal arteries. Echography showed a uniform reduction of diameter of the femoral (2 mm) and popliteal (1 mm) arteries. Treatment with pentoxifyllin (300 mg/8 h, intravenously), calcium nadroparin (6150 U/12 h, subcutaneously) and nicardipin (20 mg/8 h, orally) was unsuccessful. Arteriography showed a uniform reduction of flow in the popliteal arteries of both legs without images of thrombosis or vasculitis. Femoral arteries were also narrowed. Treatment with an analogue of prostacyclin (iloprost, up to 50 µg/day intravenously for 7 days) was started on 12 December with immediate response.

The patient remembered taking a single dose (2 mg) of ergotamine tartrate 24 hours before symptoms appeared. Antiviral treatment including ritonavir was continued. The patient was discharged on 23 December. No recurrence occurred.

Discussion

All HIV protease inhibitors are primarily metabolised by the hepatic cytochrome P450

Learning points

- ritonavir is the only inhibitor of HIV protease to act as a cytochrome P450 inhibitor
- ergotamine and its derivatives must not be administered concomitantly with ritonavir
- ergotism due to the interaction between ritonavir and ergotamine derivatives can be treated with an analogue of prostacyclin (ritonavir can be continued)

isoenzyme CYP 3A4.⁵ Ritonavir is also metabolised by cytochrome P450 isoenzymes CYP 2D6 and CYP 2C9/10, and is the only HIV protease inhibitor to act as an inhibitor of these three cytochrome P450 isoenzymes. Ritonavir might therefore be expected to slow the metabolism of various drugs. This mechanism explains why ritonavir increases the bioavailability of other HIV protease inhibitors, especially saquinavir, when given concomitantly, and also explains the increase of ergotamine or dihydroergotamine concentrations to toxic levels in our patient.

Our case report is particularly interesting because ergotism appeared after the administration of only a single dose of ergotamine tartrate. Ritonavir was not stopped, unlike the case of Caballero-Granado,⁴ but the duration of symptoms was similar in both cases. The interaction with ergotamine is now described in the data sheet of ritonavir and co-prescription is contraindicated. Co-prescription with other HIV protease inhibitors is not recommended.

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Surgical emphysema and pneumomediastinum in a child following minor blunt injury to the neck

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Summary

Laryngotracheal and pharyngoesophageal tears following minor blunt trauma to the neck are uncommon. A child with such an injury is reported and the modes of diagnosis and management are discussed. Patients may initially present with minimal signs and symptoms, but their condition may deteriorate rapidly or insidiously. In the absence of respiratory compromise, conservative management is appropriate, but all patients with significant blunt neck trauma should undergo early direct laryngoscopy under a general anaesthetic.

Keywords: blunt injury; neck; emphysema; pneumomediastinum

A 4-year-old girl fell forwards onto the backrest of a wooden chair, causing minor pain to the neck and upper chest. Although initially well, 4 hours later she was taken to Mayday Hospital with neck swelling and hoarseness. On examination there was marked surgical emphysema over the neck and chest extending to the sixth rib (figure 1). There was no bruising or stridor and the trachea was central. The larynx was

normal on palpation and there was no dysphagia. Oxygen saturations remained normal on air. A chest X-ray demonstrated a pneumomediastinum (figure 2).

The child was transferred to St George's Hospital where flexible fiberoptic laryngoscopy revealed moderate swelling of the supraglottis without pooling of saliva. A thoracic opinion was obtained and the clinical findings were suggested to be compatible with barotrauma to the oesophagus resulting in a pinhole perforation. The child was monitored on the paediatric ITU overnight and kept nil by mouth. The following day she remained well and afebrile. There was some resolution of the surgical emphysema and she was commenced on clear fluids and later a soft diet. She was subsequently discharged without complication.

Discussion

Minor blunt trauma to the neck and upper chest in children is common, but significant injury to the pharynx, larynx, trachea or oesophagus is unusual. However, cases of laryngotracheal and pharyngoesophageal tears from relatively minor blunt trauma have been documented in the literature.

Children have a proportionally large mandible and short neck in comparison with adults. In addition, a child's soft and pliable laryngeal and tracheal cartilages can probably sustain greater temporary force than the adolescent or adult airway, a child's non-calcified larynx being more likely to recoil to its original position without fracturing. Despite protective mechanisms, however, with the neck extended, a midline blow can exert excessive force on the larynx and trachea, crushing the trachea

Figure 1 Lateral soft-tissue X-ray of the neck showing surgical emphysema

Figure 2 Chest X-ray showing pneumomediastinum

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