normal on serial testing. A review of the recent medical literature yielded only one case in which hypercortisolism disguised symptoms and signs of an abdominal surgical crisis in a patient who died of an unrecognized gallbladder perforation.1 Presumably in both these cases the well-documented anti-inflammatory and immunosuppressive effects of hypercortisolism2,3 masked the development of infection and reduced the host’s response once it has become established. Cushing’s syndrome is known to predispose to opportunistic infection, in the past up to 46% of patients with Cushing’s syndrome died of sepsis, many following major surgery.4 In the modern era opportunistic infection is less commonly seen. This case illustrates the need for continuing vigilance in patients with Cushing’s syndrome. It reaffirms the commonly held belief that hypercortisolism, whether due to Cushing’s syndrome or exogenous steroids, can mask severe abdominal pathology.

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References


Trimethoprim-induced vasculitis

Sir,

Adverse drug reactions associated with co-trimoxazole may be caused by either trimethoprim or sulphamethoxazole. However, the skin rashes are so similar to those caused by sulphonamides alone that most are attributed to these.1 We report a case of vasculitis which was induced by trimethoprim.

An 83 year old woman was admitted with severe anaemia (HB 3.8 g/dl) secondary to vitamin B12 deficiency, attributed to a previous vagotomy and pyloroplasty and a coliform septicemia secondary to a urinary tract infection. She was treated with intravenous cefuroxime for 11 days. She then suffered a right cerebro-vascular accident. Following this she made a good recovery. During her rehabilitation, 2 months later, she developed a further urinary tract infection. A mid-stream urine grew coliforms, which were sensitive to trimethoprim, but resistant to ampicillin and nalidixic acid. She was given a week’s course of trimethoprim 200 mg twice daily. Nine days later she developed a florid leucocytoclastic vasculitis on her hands, tongue, shins and feet. This was associated with acute arthritis of her interphalangeal joints and haematuria. Previous urinalysis for blood had been negative.

Her daughter then remembered that her mother had had a similar rash 2 years previously when she had been treated with co-trimoxazole for a urinary tract infection. A case summary was obtained and this confirmed that our patient had had a vasculitis, secondary to co-trimoxazole.

Other causes of a vasculitic rash were looked for: a full blood count showed a white cell count of 3.3 x 10⁹/l, haemoglobin of 14.9 g/dl and platelets of 120 x 10⁹/l. Blood cultures, rheumatoid factor and auto-antibodies were negative. Routine viral titres showed no recent infection. Serum electrophoresis showed a double M band in the gamma region but there was no Bence–Jones protein. Serum complement and cryoglobulins were in the normal ranges. An ultrasound of her abdomen was normal.

She was treated with hydrocortisone (200 mg 4 times daily) and intravenous antibiotics (to cover for a possible diagnosis of endocarditis). She responded dramatically to treatment over a week but developed a chest infection and died 4 weeks later.

We are convinced that the vasculitis was caused by trimethoprim because of the recurrence of the rash on subsequent exposure and because of the lack of another explanation. The Committee on the Safety of Medicines has a number of reports of various types of rashes being associated with the use of trimethoprim. These include maculo-papular, morbilliform, erythematous, angio-derma and purpuric. There was only one previous reported case of a vasculitis from Holland (unpublished, Wellcome Foundation Ltd files) which was described as purpuric and is distributed over the trunk and lower limbs.

Patients who have adversely reacted to co-trimoxazole should receive neither trimethoprim nor sulphamethoxazole in future.

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References


Listeria rhombencephalitis in a previously healthy adult

Sir,

We report a case of listeria rhombencephalitis (brain stem encephalitis) in a healthy adult. An 85 years old previously well woman was admitted with a 10 day history of right seventh cranial nerve paresis. She was pyrexial and slightly confused. The neurological examination revealed nystagmus towards
Trimethoprim-induced vasculitis.

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doi: 10.1136/pgmj.68.799.391

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