and nail scrapings for fungi and hyphae were negative. Serum zinc concentration was 14 $\mu\text{mol/l}$ (92 $\mu\text{g}/100\text{ ml}$), zinc clearance 3.5 $\mu\text{mol/l}$ (229 $\mu\text{g/l}$), and creatinine clearance 95 ml/min.

Comment

These are the only three cases of onycholysis due to cytotoxic drugs to be reported without associated dermatopathy. This is surprising as our experience indicates that it is not a rare phenomenon: these three cases occurred over nine months. Two of the patients had low serum zinc concentrations and all three had reduced urinary zinc clearance, which with normal creatinine clearance implies zinc deficiency.

The precise importance of these findings is uncertain, though the importance of zinc in growth and nutrition is well known and possibly the combination of zinc deficiency and cytotoxic treatment led to nail loss. Thus zinc supplementation might conceivably have prevented the onycholysis. The cytotoxic agents common to all patients were doxorubicin and vincristine. Doxorubicin causes alopecia in virtually all patients, and the developmental similarity between hair and nails is readily apparent. Vincristine has no such effect on hair. Of the other cytotoxic drugs, methotrexate suppresses growth of finger nails³ and cyclophosphamide and bleomycin cause pigmentation and banding of nails.^{4,5} Therefore, although doxorubicin is most likely to have been the cause, the contribution of the other cytotoxic agents or zinc deficiency cannot be dismissed.

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Cortical blindness complicating Wegener's granulomatosis

Wegener's granulomatosis is characterised by a necrotising vasculitis of the respiratory tract and an associated glomerulonephritis. The condition often includes a generalised vasculitis affecting various organs,¹ sometimes producing neurological and ocular complications such as cranial nerve abnormalities and mononeuritis multiplex, and conjunctivitis, retinal vasculitis, and optic neuropathy.²

We report on a patient with classical Wegener's granulomatosis who suffered bilateral infarction within the occipital cortex with resultant cortical blindness. This complication has not been reported previously.

Case report

A 35 year old woman with a three year history of nasal stuffiness and a chronic clear nasal discharge developed polyarthralgia in early 1983. In August 1983 she developed scleritis and was treated with prednisolone 30 mg and azathioprine 150 mg daily. The scleritis, however, persisted and in January 1984 a chest x ray film showed a cavitating lesion in the upper lobe of the right lung. Despite a negative Mantoux test result and sputum culture, rifampicin 600 mg and isoniazid 300 mg daily were started for presumed pulmonary tuberculosis. There was no response and one month later her polyarthralgia deteriorated and a blistering skin eruption developed. A dipstick urine test showed protein and blood in large amounts and renal function began to deteriorate to the point where haemodialysis became necessary. Skin and nasal septum biopsy specimens showed necrotising vasculitis and a renal biopsy specimen showed focal segmental proliferative glomerulonephritis with crescents in 64% of the glomeruli. Immunofluorescence showed traces of IgG and C3, but no IgA, IgM, C1q, or C4. The crescents stained with high intensity for fibrinogen.

Treatment with cyclophosphamide, prednisolone, and plasma exchange

produced dramatic improvement. Her blood pressure was well controlled and renal function began to recover rapidly (see figure). Nevertheless, her progress was interrupted by two generalised seizures over four days. An

Effect of treatment on renal function. Under vision, episode 1 was transient loss of vision and episode 2 permanent loss of vision.

isotope brain scan after the first seizure was normal. Just before the second seizure she had transient blindness, but after she recovered from the convulsion her vision was normal. A third seizure followed, after which a repeat isotope brain scan was again normal. Nevertheless, two days later she developed sudden blindness, which this time persisted. Direct and consensual reactivity of the pupils was normal but she could not see finger movement at a few inches' distance and a rotating drum test could not induce nystagmus. Computed tomography showed segmental areas of low attenuation consistent with small focal areas of infarction near both occipital poles and in the high parameian parietal regions. Although renal function subsequently improved steadily, she recovered very little vision. Perimetry showed generalised constriction of the peripheral visual fields consistent with bilateral cortical infarction.

Comment

Cortical blindness has been described in association with hypertension secondary to glomerulonephritis.^{3,4} In both these cases complete recovery of vision occurred and blindness was probably secondary to arteriolar spasm (of the branches of the posterior cerebral arteries), which occurs in severe hypertension.³

Our patient had transient blindness preceding a convulsion while her blood pressure was well controlled. The permanent blindness which developed subsequently occurred when the renal and systemic manifestations of her disease were improving, and the two episodes are therefore unlikely to have been due to spasm or to progressive disease. Although combination therapy proved effective against the arteritis in the kidneys, and brought about definite improvement in renal function, the most likely explanation for the blindness is that thrombosis occurred in branches of the posterior cerebral arteries affected by the original disease. If this is so then in Wegener's granulomatosis and other forms of arteritis antiplatelet agents may be important in minimising tissue damage once the original inflammatory reaction has been controlled by steroids, cyclophosphamide, and plasma exchange.

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