after an illness of 3 weeks. We feel that the ear abscess and the chloroquine treatment had no influence on the autonomic neuropathy. A literature search failed to reveal any reports of autonomic neuropathy associated with chloroquine therapy.

In this case the 8-fold fall in Widal titres of O and H antigens can be considered to be meaningful. It is possible that the patient had an attenuated typhoid fever because he had received vaccine. An anamnestic reaction to the antigens of *S. typhi* at the exposure documented, however, could also explain the titres of the O and H antigens as a result of the preceding immunisation. The relation between the auto-immune neuropathy and the titres of antibody in the absence of any viral infection is striking, however.

The first recorded case of acute autonomic neuropathy was described in 1969 by Young *et al.*,[2] since then very few cases have been reported.[3,4] The aetiology and pathogenesis of the condition remains obscure; it has been associated with infectious mononucleosis,[4] an illness in which auto-immune mechanisms[3] are said to be operative. A raised cerebrospinal fluid protein level[2] has been noted in certain cases.

We were unable to find any references relating to pure autonomic dysfunction in typhoid infections, although typhoid has been associated with numerous neurological complications,[5] e.g. coma, semi-coma, confusional states, meningitis, convulsions, generalised myoclonus, focal neurological signs (e.g. deafness, hemiplegia, facial palsy), parkinsonism, spasticity, hypotonicity, schizophrenia, symmetrical sensorimotor polyneuropathy and mononeuritis.

No explanation has been forthcoming for the relative bradycardia and constipation during the first week of typhoid fever and it is possible that an autonomic neuropathy may account for these features.

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Eosinophilic cystitis associated with Glanzmann's thrombasthenia

A case report

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Summary

Eosinophilic cystitis is a rare condition, only 41 cases having been recorded in the literature. Glanzmann's thrombasthenia has been documented more than 100 times. The presence of these two conditions in one patient has, to our knowledge, not yet been reported in the English-language literature.

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Since 1959 at least 41 cases of eosinophilic cystitis have been described.[1] Its association with the uncommon Glanzmann's disease has not yet been reported.[2]

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A patient with eosinophilic cystitis generally presents with urgency, frequency, dysuria, bladder pain and, not uncommonly, haematuria. The symptoms usually show remission within a few weeks.[1,3] Rapid regression of the vesical mucosal lesions has also been described.[3] The clinical condition must be differentiated from interstitial cystitis, tuberculosis and bladder neoplasms.

Glanzmann's thrombasthenia is inherited as an autosomal recessive trait and consanguinity is frequently present in affected kindred. The basic abnormality seems to be that the platelets are refractory to the aggregating stimulus of adenosine diphosphate (ADP). This leads to the formation of a haemostatically inadequate platelet plug and deficient clot retraction. There is no specific treatment, but blood and platelet transfusions are beneficial. Exchange thrombocytophoresis should be considered in cases of life-threatening haemorrhage.[2]

Case report

A 52-year-old white woman presented with a 6-week history of frequency, dysuria and macroscopic haematuria. She denied previous episodes of haematuria, but gave a history of easy bruising and repeated epistaxis. She had received 9 units of blood after a caesarean section, and also required blood transfusions after tooth extractions. Her paternal uncle had died at the age of

12 years from an unspecified haemorrhagic incident, and her sister at the age of 2 years as a result of uncontrolled epistaxis.

Physical examination was unremarkable except for multiple bruises ascribed to minor trauma. The patient's haemoglobin value was 10,0 g/dl, the platelet count 27,8 x 10^9/l, and the prothrombin and partial thromboplastin times were normal. The bleeding time was normal at 2½ minutes. A peripheral blood smear revealed no abnormality, and the eosinophil count was normal. Urine cultures were negative for urinary pathogens, including *Mycobacterium tuberculosis*. Serological tests for schistosomiasis were negative.

Screening tests for collagen disease, including antinuclear factor, were negative, and routine intradermal allergy tests evoked no reaction. The ristocetin test was normal, and the ADP test was non-responsive up to 1 000 μg/ml. Platelet aggregation could not be induced by the addition of adrenaline or collagen.

An intravenous pyelogram was normal. At cystoscopy multiple haemorrhagic areas were seen in the bladder and several specimens were taken. Histological study of the specimens revealed the typical picture of eosinophilic cystitis (Fig. 1).

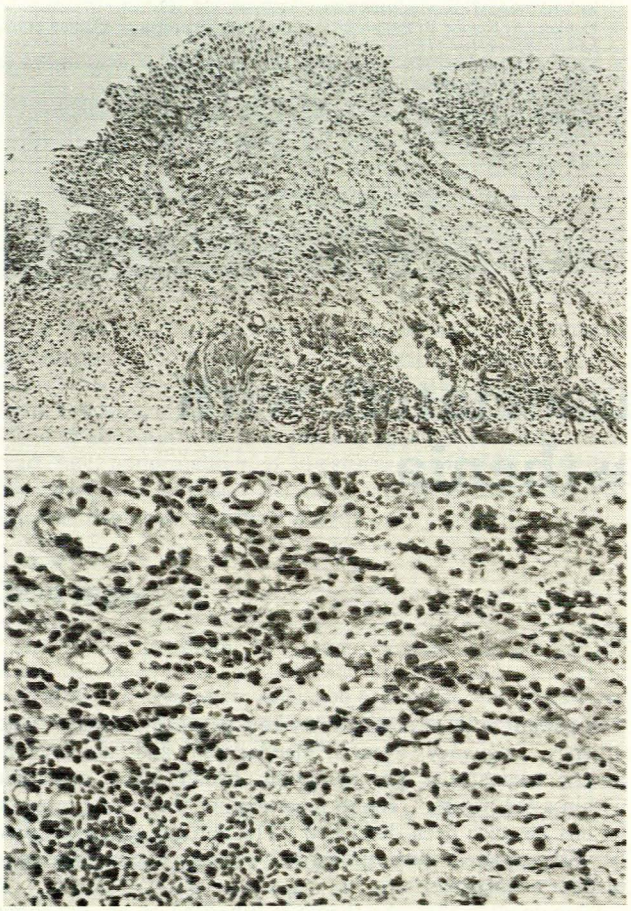

Fig. 1. Severe oedema of the lamina propria, with an extensive eosinophil infiltrate and vascular congestion (top: **H and E x 100;** bottom: **H and E x 400).**

The patient had severe postoperative bleeding from the bladder, which necessitated several blood transfusions and endoscopic fulguration of the biopsied areas. One mega-unit of platelets was administered, after which the bleeding abated.

The patient was initially treated with prednisone 40 mg/d, the dose being tapered off to zero over 14 days. She also received promethazine hydrochloride 25 mg 3 times a day for the same period.

Repeat cystoscopy after 2 weeks revealed a virtually normal bladder mucosa. Bladder wall biopsy at this time showed chronic inflammatory changes with a very scant eosinophilic infiltrate. At a 6-month follow-up examination the patient was symptom-free and the bladder was endoscopically normal.

Discussion

The aetiology of eosinophilic cystitis is unclear, but bacteria and foreign proteins are believed to act as antigenic stimuli. Immune complexes cause the release of lysosomes, which results in an inflammatory reaction with an eosinophilic infiltrate. Parasitic infestation of the bladder has been proposed as an aetiological factor, but only 1 proven case has been reported.[4]

In classic cases the patients present with severe symptoms of cystitis and suprapubic pain, and they may have eosinophilia, pyuria and haematuria.[1] The diagnosis is established by means of a biopsy, the specimen showing eosinophilic infiltration of the bladder wall.

Treatment includes the removal of known allergens such as oranges, tomatoes, chocolates, tea, coffee, spices and in 1 case milk.

Drugs such as warfarin have been implicated, and contact allergens such as condoms, vaginal tampons and spermicidal jellies are also suspect.[1]

Systemic antibiotics are used to control secondary infection; steroids and antihistamines are also of proven benefit.[1] Fulguration of lesions has been successful, but 3 cases requiring cystectomy and urinary diversion have been reported.[5]

In our patient no causative agent could be identified, and the significance of the associated Glanzmann's thrombasthenia is unknown.

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