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Lesson of the week

Wegener's granulomatosis presenting as a pleural effusion

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Wegener's granulomatosis is one of the pauci-immune small vessel vasculitides. It classically presents with the triad of upper and lower respiratory tract granulomas and necrotising focal segmental glomerulonephritis. It is associated with the presence in the serum of autoantibodies against components of neutrophil cytoplasm—antineutrophil cytoplasmic autoantibodies (ANCA). The illness can develop at any age but is more common in patients in their 50s and 60s and in men. The incidence of vasculitis is increasing, with about 10-20 people per million affected. We present a case that in retrospect had many clues at the initial time of admission, but it took five months and six different hospital teams to make the diagnosis.

Case report

A 64 year old woman, who had had breast carcinoma that had been treated with wide local excision and radiotherapy six years previously, was admitted to her local hospital at the end of March 2001. She presented with a two week history of an influenza-like illness, including a blocked nose and right ear, dry cough, and intermittent sweats. She was feverish and had a left pleural effusion, which was confirmed radiologically. Her inflammatory markers were raised (total white cell count $13.1 \times 10^9/l$, C reactive protein 322 mg/l (normal range <5 mg/l)). Treatment was started with intravenous antibiotics for a possible empyema. Despite three different antibiotics, her symptoms failed to improve over the next two weeks. Ultrasound scanning of the chest confirmed a fluid collection, but several attempts at aspiration and drainage were unsuccessful. She was transferred to a teaching hospital under the care of the cardiothoracic surgeons, but she became increasingly breathless and developed atrial tachyarrhythmias and presumed acute pulmonary oedema. At this time there was evidence of renal impairment (serum creatinine concentration 130 $\mu\text{mol/l}$) and she was deemed unfit for surgery, so she was transferred back to the referring centre.

A computed tomogram of the thorax showed bilateral pleural effusions, and transthoracic echocardi-

graphy showed a pericardial effusion. Owing to persisting fever and raised inflammatory markers, her antibiotic regimen was again altered and she was transferred to a different tertiary centre for a respiratory opinion. Soon after admission she developed respiratory failure and needed intubation and ventilation. She was found to have no empyema. She recovered slowly and was transferred back to her original team at the beginning of June without a uniform diagnosis. Repeat echocardiography at this time showed resolution of the pericardial effusion and her creatinine concentration was 124 $\mu\text{mol/l}$.

At the end of June she was transferred to a community hospital for rehabilitation. Over the next month she had recurrent episodes of syncope and bradycardia. Her serum potassium concentration was persistently raised and her renal function deteriorated markedly (creatinine concentration 618 $\mu\text{mol/l}$). She had a cardiac arrest, from which she was successfully resuscitated. She was subsequently transferred to the intensive care unit of our hospital, where she needed ventilation support and continuous venovenous haemofiltration for acute renal failure. She was found to be strongly seropositive for cytoplasmic ANCA (cANCA) (titre >2560 units) for antibodies to proteinase 3 with enzyme linked immunosorbent assay (ELISA), and was treated with pulsed intravenous methylprednisolone, followed by oral prednisolone and cyclophosphamide. Two weeks later she developed pulmonary haemorrhage and needed reintubation and treatment with plasma exchange for two weeks. She improved slowly over the next few weeks and was discharged at the end of October on a combination of prednisolone and azathioprine; her creatinine concentration at this time was 200 $\mu\text{mol/l}$.

Discussion

Vasculitis can be categorised by the size of vessel affected (small, medium, or large). Wegener's granulomatosis is a small vessel vasculitis classically involving the upper and lower respiratory tracts and kidneys. A limited form

Check ANCA (antineutrophil cytoplasmic autoantibodies) urgently in patients with respiratory symptoms and unexplained renal impairment

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involving the upper respiratory tract has been described in a quarter of cases.¹ The most common presenting symptoms include most upper airway respiratory symptoms, but particularly rhinorrhoea, oral ulcers, nasal discharge, polyarthralgias, myalgias, and sinus pain. Lower airway complaints such as cough, dyspnoea, haemoptysis, and pleuritic pain are also early features. More rarely, Wegener's granulomatosis can present with tumour-like masses distant from the lung. The renal disease often presents with haematuria, red cell casts, proteinuria, and renal failure. Other systems that may be involved include joints, eyes, skin, nervous system, heart, and gastrointestinal tract.¹

Serological testing for ANCA is a useful diagnostic aid for small vessel vasculitis,² although caution is needed as the sensitivity may be as low as 66% when the disease is not severe.³ The diagnosis should be suggested from clinical as well as laboratory findings and where possible confirmed with a tissue biopsy.

Treatment in Wegener's granulomatosis can be life saving. It is usual to obtain a tissue diagnosis before starting treatment, but if the patient is too unwell, as in this case, treatment should not be delayed. Untreated Wegener's granulomatosis has a poor prognosis, with most patients dying within two years.¹ With cytotoxic treatment, this has improved, and the eight year survival is about 80%.¹ Up to 90% of patients respond to cyclophosphamide, and three quarters have a complete remission,⁴ but pulmonary haemorrhage remains a potentially life threatening complication.

Induction treatment is usually given as combined treatment with cyclophosphamide and corticosteroids. Use of corticosteroids alone is associated with a higher relapse rate.⁵ The specifics of induction regimens vary with the drugs used, doses, routes of administration, and duration. Patients with severe disease are treated with either plasma exchange or pulsed intravenous methylprednisolone. The results of a randomised study

comparing these two treatments when used as an additional early treatment in patients with severe kidney damage are awaited (MEPEX trial, European Vasculitis Study Group). Recovery from dialysis dependence is common; 55% to 90% of patients recover their renal function, and 40% to 70% manage without dialysis for three years or more.⁶

This case illustrates the importance of considering vasculitis in differential diagnosis of patients with multi-system disease. The clinical manifestations of small vessel vasculitis can be very varied because they are influenced by the sites of involvement and disease activity. ANCA testing is highly sensitive for detecting pauci-immune small vessel vasculitis, although its specificity depends on the patient population tested. Early diagnosis allows appropriate treatment to be started before serious and potentially life threatening complications have developed.

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The greater man, the greater courtesy

It was another busy admission day. I was working as a surgical senior house officer in Calcutta Medical College and was part of an eight member team, coping with the usual 30 or so admissions. It was the middle of the night when I saw a 9 year old boy who had just been brought to us by his father. The boy seemed to have developed generalised peritonitis and was not looking good.

"Why did you bring him so late?" I asked rather abruptly.

The boy's father, a softly spoken man, tried to explain his difficult social circumstances, lack of transport at this time, etc. Then he suddenly paused and said, "I understand his condition is not good. Please do whatever you can." I was taken aback by the polite resoluteness of the man.

We started the treatment. The child needed a laparotomy for what turned out to be a perforated small bowel due to typhoid. By the time I left next morning, his condition was slightly better but still far from good.

The next day I went away on holiday for 10 days. On my return to Calcutta, I faced a tremendous downpour of rain as I left Howrah railway station. And, guess what, I had just missed the 44 bus that would take me close to my college.

As I was cursing my luck, something strange happened. The bus reversed, and a friendly voice addressed me: "Doctor-Sahib, please get up quickly." I thanked the driver for his kindness, but it

was only after I had left the bus that I realised that the driver was the father of the boy whom I met in the hospital.

The next day, I proudly boasted to my colleagues: "Has a bus ever reversed just for you? It happened to me yesterday."

After sharing my story, I asked about the boy. "He died after a few days," came the reply.

I was humbled twice in two weeks. Firstly, by the determination of a man who kept his head cool at a time when his son was most unwell. Secondly, when he unreservedly showed his appreciation by picking me up in bad weather, perhaps even risking his job.

What did I learn? Since then, I have never asked any parents or guardians why they have brought their child or dear one late. No one does, not by choice anyway. As to the courtesy, well, I think I had a lesson or two from the humble bus driver.

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We welcome articles up to 600 words on topics such as *A memorable patient, A paper that changed my practice, My most unfortunate mistake*, or any other piece conveying instruction, pathos, or humour. If possible the article should be supplied on a disk. Permission is needed from the patient or a relative if an identifiable patient is referred to.