Cerebellar syndrome complicating *Mycoplasma pneumoniae* pneumonia

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Acute self-limiting cerebellar syndrome is a poorly recognized complication of *Mycoplasma pneumoniae* infection[1].

CASE HISTORY

A woman aged 60, who 27 years earlier had had her mitral valve replaced with a prosthesis, developed 'flu-like symptoms and a productive cough for which her general practitioner prescribed amoxycillin. On day 6 he found signs of consolidation at the right lung base with continued fever, so clarithromycin was added. She had well-controlled atrial fibrillation and was on warfarin and digoxin; the prosthetic valve was functioning normally.

On day 8 the international normalized ratio (INR) was checked and had risen to 8.2. On the advice of a haematologist she was given intravenous vitamin K. Because of concern over the anticoagulation, together with continuing fever, anorexia and persistent signs at the right base, she was admitted to hospital on day 9. On admission the chest X-ray confirmed right basal consolidation. The INR was 2; clarithromycin was stopped because it was thought to be interfering with anticoagulant control. White cell count showed a slight lymphopenia (0.6×10^9/L). She was treated with intravenous ceftriaxone for 7 days and improved clinically and radiologically. On day 17 she was switched to oral co-amoxiclav and was discharged.

On day 18 she was readmitted with relapse of her productive cough, fever and continued anorexia and weight loss. White cell count was 13.7×10^9/L (neutrophils 12.2, lymphocytes 0.4), erythrocyte sedimentation rate was 40 mm/h and C-reactive protein 97 mg/dL. Chest X-ray now showed left basal consolidation with resolution of the right basal consolidation. Increased antibody to *M. pneumoniae* was detected by the complement fixation and gelatin particle agglutination tests, confirmed by the presence of specific IgM by enzyme-linked immunosorbent assay in all samples apart from that collected on day 8. No significant antibodies were detected by the complement fixation test to adenovirus, influenza virus type A, herpes simplex, *Coxiella burnetii* and/or *Chlamydia psittaci*, and specific IgM to Epstein–Barr virus was absent. *Pseudomonas aeruginosa* was grown from a sputum sample. She was treated with intravenous ceftazidime and gentamicin and with oral azithromycin.

On day 20 she required catheterization for urinary retention and on day 21 she was noted to be very weak with slurred speech and complaining of double vision. Examination revealed bilateral horizontal nystagmus, upbeat nystagmus, slurring dysarthria, bilateral upper and lower limb ataxia and bilateral dysdiadochokinesis; the central and peripheral nervous systems were otherwise normal. A computed tomographic head scan and magnetic resonance imaging of the brain and cerebellum showed nothing abnormal. Thyroid function tests were normal. C-reactive protein peaked at 135 mg/dL. A lumbar puncture was not done because it seemed unlikely to help diagnostically and her INR was 4.6. On day 22 she no longer exhibited dysdiadochokinesis and on day 24 her nystagmus was less pronounced and she was able to walk aided. On day 27 she was walking unaided and had nystagmus to the left only, and on day 28 she was well enough to be discharged, taking azithromycin for 5 days. At follow-up on day 35 she had made a full recovery from her pneumonia and was neurologically normal.

COMMENT

M. pneumoniae infection has neurological complications in some 7% of patients requiring hospital admission, manifesting as focal or diffuse encephalitis, meningitis, psychosis, transverse myelitis, polyradiculitis, peripheral neuropathy, cerebellar syndrome or combinations of these. It can develop as long as three weeks after the onset of the respiratory illness, and in up to 20% of cases with neurological features there is no history of respiratory symptoms. Most patients make a full neurological recovery[2,3].

Reviewing 50 cases with neurological involvement Lerer and Kalavsky[2] reported that all 7 of the patients with cerebellar signs also had encephalitis—non-focal in 5 and focal in 2. Three mechanisms have been postulated for the neurological effects. *M. neurolyticum* and *M. gallisepticum* both produce a neurotoxin causing neurological disease in animals, but there is no evidence for a similar mechanism with *M. pneumoniae*. *M. pneumoniae* has been isolated from the cerebrospinal fluid (CSF) in man[4], but the organism is difficult to culture so it is not known whether direct infection arises in all cases. An autoimmune mechanism might explain the delay often seen between the respiratory

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and neurological manifestations. Autoantibodies to several host tissues, including brain, have been identified in infected patients with and without neurological complications[5]. CSF findings in *M. pneumoniae* neurological disease have included a raised protein, normal to low glucose and pleocytosis with lymphocytes predominating[2,3].

Brain scans are usually normal but may show evidence of oedema[3]. In our patient the differential diagnosis was cerebrovascular accident, possibly secondary to erratic anticoagulation. It is noteworthy that in this case the neurological complication occurred during relapse after an incomplete course of appropriate antibiotics, and coincided with the most severe phase of her respiratory illness. In published series the proportion of patients with neurological complications who have received appropriate antibiotic treatment is not clear.

This report highlights two important points. First, the potentiating effect of macrolide antibiotics on oral anticoagulants may discourage some physicians from prescribing the appropriate agents in anticoagulated patients with atypical pneumonia. Conversion to heparin may be an option in such cases, to allow proper treatment. Second, *M. pneumoniae* infection should be thought of in all patients with cerebellar syndrome, whether or not they have had antecedent respiratory symptoms.

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Pulsatile varicose veins

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Tricuspid regurgitation is a rare cause for unilateral pulsatile groin swelling that continues to be missed in clinical practice[1,2]. A pulsatile vein may be mistaken for an arteriovenous fistula.

CASE HISTORY

A woman aged 78 reported that her legs had suddenly become painful and cool. The symptoms resolved overnight after treatment with heparin. On examination she was in atrial fibrillation; she had bilateral femoral pulses with no pulses distally. The legs were of equal size but the right leg showed a pulsatile varicosity of the entire right long saphenous vein extending to the ankle, present even when the limb was elevated. In view of her history of coronary angioplasty performed via the right groin 14 years ago, an iatrogenic arteriovenous fistula was suspected, and a duplex scan done by an inexperienced operator supported this diagnosis. On angiography, however, there was no evidence of either arteriovenous fistula or acute embolism to the limbs. The patient was therefore re-examined. No machinery murmur was present in the long saphenous vein and Branham's sign (relative bradycardia occurring with occlusion of a fistula) was absent. There were classic signs of tricuspid regurgitation, including a pansystolic murmur loudest on inspiration at the left sternal edge, raised jugular venous pressure with prominent cV waves, pulsatile hepatomegaly and distended abdominal veins. A further duplex scan by an experienced operator confirmed regurgitant flow extending to the ankle (Figure 1), with a biphasic waveform typical of tricuspid regurgitation present also in the inferior vena cava and hepatic veins. The iliac venous valves on the right were undetectable. Echocardiography showed a dilated right atrium and confirmed tricuspid regurgitation. Right ventricular function was normal.

COMMENT

The signs of tricuspid regurgitation derive from 'ventricularization' of venous flow[3] both proximally and distally. If there is concomitant saphenofemoral junction incompetence then lower-limb varicosities will pulsate throughout the course of the long saphenous vein. In the patient reported here, absence of iliac vein valves on the right enhanced back pressure on the saphenofemoral junction, producing the striking phenomenon of venous pulsatility at the ankle. Venous pulsatility has previously been described to the popliteal fossa[4] but not, to our knowledge, to the ankle.

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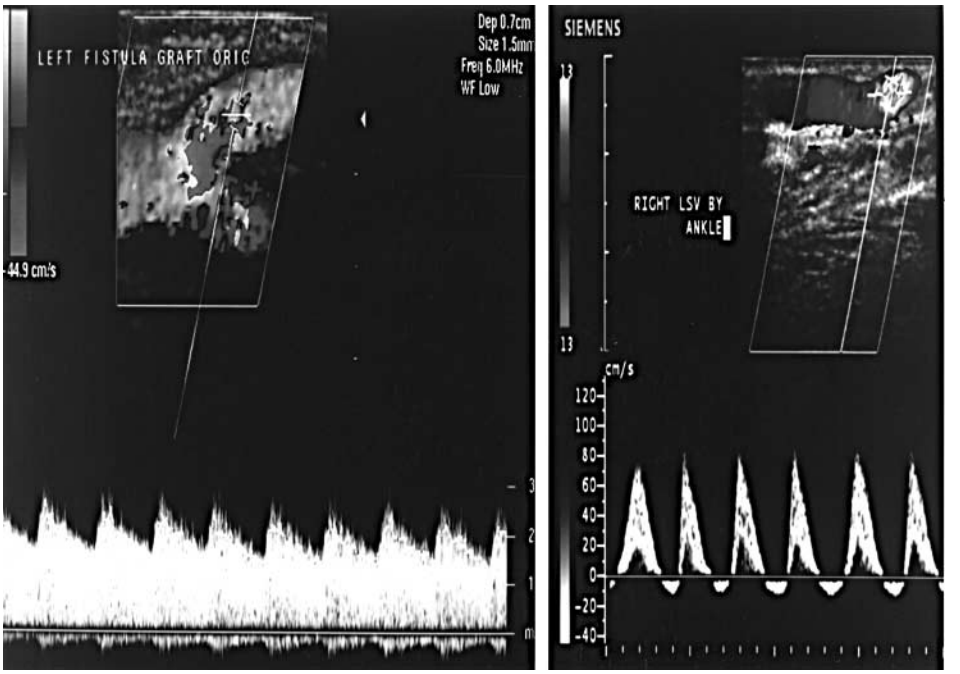

Figure 1 **Colour doppler flow curves of tricuspid regurgitation (right) and a hypothetical arteriovenous fistula (left).** The right hand display shows a biphasic pulsatile jet form at the ankle of 80 cm/s towards the transducer (conventional orientation, with head of patient to left of picture).

The features in this patient were compatible with a high output fistula following cardiac catheterization, though in retrospect none of the signs pathognomonic of an arteriovenous fistula were present. A central difficulty was that the clinical features were not consonant with the principal clinical problem. A possible explanation of events is that her atrial fibrillation led to arterial embolization that resolved on early heparinization (though echocardiography did not demonstrate any source of cardiac emboli). A cause of both tricuspid regurgitation and atrial fibrillation might be rheumatic heart disease, but the patient had no history of rheumatic fever.

Clinical diagnosis of tricuspid regurgitation is not always easy. The accentuated cV-waves in the neck veins can be missed if the upper limit of the venous column cannot be seen to pulsate, and pulsation of a distended liver may be misinterpreted as transmitted from the abdominal aorta. With a duplex scan an experienced operator should always be able to differentiate the biphasic flow of tricuspid regurgitation from the low impedance monophasic flow of an arteriovenous fistula.

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