Antiphospholipid antibody-related Bell's palsy in young women

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ABSTRACT The present report describes three young women aged 25, 20 and 15 years who presented with Bell's palsy. Two of the patients had a past history of the disease, which responded to steroid treatment. All three patients were positive for antiphospholipid antibody (APLA). In addition, one of the patients tested positive for antinuclear antibodies; however, there was no clinical evidence of systemic lupus erythematosus. All three patients responded well to steroid therapy. We conclude that Bell's palsy may be one of the manifestations of APLA syndrome, and thus, APLA testing should be done in such cases.

Keywords: antiphospholipid antibody, APLA syndrome, autoimmune neuropathy, Bell's palsy, facial palsy Singapore Med J 2012: 53(3): e55-e56

INTRODUCTION

Bell's palsy, an idiopathic disorder, is the most common form of facial paralysis. It is a common condition that affects both males and females of all age groups. The disease begins with pain around the ear, followed by loss of movement on one side of the face, accompanied by numbness, hyperacusis and loss of both taste and salivation. The role of autoantibodies in cases of Bell's palsy has not been described in the literature. Therefore, we performed tests of antiphospholipid antibody (APLA), antinuclear antibody (ANA) and anti-double-stranded deoxyribonucleic acid (dsDNA) antibody in order to examine their roles in three cases of Bell's palsy.

CASE REPORTS

Case 1

A 25-year-old Indian woman presented in December 2007 with complaints of deviation of the angle of the mouth and an inability to close the right eye for the past five days. The problem had started about a week following a full-term normal delivery. Clinical examination revealed right-sided facial nerve palsy. There was no history pertaining to joint pain, facial rash, recurrent abortion, oedema or any other features of systemic autoimmune diseases. Laboratory investigations of total leukocyte count, differential leukocyte count, erythrocyte sedimentation rate (ESR), routine and microscopic urine examination were within normal limits. Immunological profile was positive for ANA and APLA (30 GPL/ ml) but negative for rheumatoid factor, C-reactive protein and anti-dsDNA antibody. Nerve conduction velocity was lower in the right facial nerve as compared to the left facial nerve. The patient was treated with prednisolone and recovered completely in three months.

Case 2

A 20-year-old Indian woman presented in March 2008 with

complaints of deviation of the left side of the mouth, along with numbness and inability to close her left eye. In November 2006, she had had a similar episode. She was diagnosed with Bell's palsy and administered steroids, to which she responded well. Laboratory examinations of total leukocyte count, differential leukocyte count, ESR and urine examination were within normal limits. Immunological tests revealed that her rheumatoid factor and anti-dsDNA antibodies were negative; ANA was equivocal, but APLA was positive (20 GPL/ml). The patient was lost to follow-up, and hence, her progress could not be monitored.

Case 3

A 15-year-old Indian girl presented to the neurology outpatient department in March 2008 with recurrent episodes of deviation of the angle of the mouth to the right side and inability to close the right eye. The first episode had occurred 18 months ago, and she had recovered after 20–25 days of treatment with steroids. At presentation, she had similar complaints, which started gradually one day ago. She also complained of recurrent ulcers and erosions of the buccal mucosa. Laboratory findings showed that haemoglobin, total leukocyte count, differential leukocyte count, ESR, and urine routine and microscopic examinations were all within normal limits. Immunological profile was negative for ANA, anti-dsDNA antibody and rheumatoid factor. However, the patient tested positive for APLA (15 GPL/ml).

DISCUSSION

Many conditions could be considered in the differential diagnosis of Bell's palsy. Among these are tumours of the carotid body, cholesteatoma, acoustic neuroma, dermoid tumour, herpes zoster of the geniculate ganglion causing vesicular eruptions, facial palsy and eighth nerve palsy (Ramsay Hunt syndrome). Cerebral infarct, multiple sclerosis, sarcoidosis of the uvea and parotid (Heerfordt's syndrome), Lyme disease and Melkersson-Rosenthal syndrome

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(recurrent facial palsy, labial and facial swelling and plication of the tongue) are the other conditions that cause facial palsy. (1,2) The literature contains very few reports of Bell's palsy due to APLA. APLA is typically associated with recurrent abortions, arterial and venous thrombosis, myocardial infarction, peripheral neuropathy, cerebral infarct and lupus endocarditis.(3) Adverse pregnancy in the form of recurrent pregnancy loss and intrauterine death with maternal complications including thrombosis, thrombocytopenia and pregnancy-induced hypertension is very common in APLA patients; these antibodies cause thrombosis in the placental blood vessels and impairment of embryonic implantation. (4,5) Recently, one report described a case of multiple mononeuropathy of the lower limbs associated with anticardiolipin antibody, vasculitis and thrombus formation.⁶⁰ In our patients, blood vessels near the facial nerve may have had ischaemia due to thrombosis resulting from APLA, hence leading to facial nerve

palsy. Thus, the estimation of APLA titre is suggested in every case of Bell's palsy.

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