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SHORT REPORTS

Bilateral ulnar and radial nerve palsies as a complication of childbirth

Iatrogenic bilateral ulnar nerve palsies have resulted from the use of walking frames¹ and operating tables.² With the former the injury is at the wrist and with the latter at the elbow. Theoretically, iatrogenic bilateral radial nerve palsies could occur in the axillae owing to incorrectly fitted crutches, but to our knowledge this has never been reported. We report a case of bilateral radial and ulnar nerve palsies in a patient as a result of childbirth using a birthing stool.

Case report

A 31 year old pregnant woman was admitted to the labour ward at 39 weeks' gestation having had uterine contractions for seven and a half hours. She had had one previous full term normal vaginal delivery with an uneventful labour and there was no relevant medical or family history. About two hours after admission she had a fully dilated cervix and a vertex presentation. She chose to use our three legged wooden birthing stool (supplied by Crewdys Wood Products) during the second stage with her husband sitting in a standard office chair behind her to provide support. Instead of passing his hands under her axillae so as to support her with his forearms, he placed his hands in her axillae. Throughout the second stage, which lasted 20 minutes, she noticed numbness and tingling in both her

Comment

To our knowledge this is the first reported case of both bilateral radial and ulnar nerve palsies and a maternal nerve palsy as a complication of childbirth. Teres major and latissimus dorsi form the posterior axillary wall over which the arm's neurovascular bundle passes directly anteriorly. Within this neurovascular bundle the radial nerve lies posteriorly, and the ulnar nerve is the most medially placed structure and lies posteriorly to the axillary vessels and median nerve. Apparently, her husband's finger tips compressed the radial and ulnar nerves against the posterior axillary wall and pushed the other, more anteriorly placed, neurovascular structures superiorly into the axilla. Fortunately, her husband did not compress the neurovascular bundle against the posterior axillary wall 2 cm more proximally, as he may then have also damaged the axillary nerve causing weakness of shoulder abduction.

Since bilateral ulnar and radial nerve palsies can occur with this method of delivery, delivery suite staff need to be aware of this danger.

We thank Mr D McCoy, consultant obstetrician, for allowing us to report this case.

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(Accepted 15 April 1983)

Details of sensory and motor deficits of arm muscles due to bilateral radial and ulnar nerve palsies

	Left arm	Right arm
<i>Sensory deficit</i>		
Dorsum of first web space		
Ulnar border of hand and little finger	Tingling Numbness	Tingling Numbness
<i>Motor deficit</i>		
Biceps	5	5
Triceps	1	1
Flexor carpi radialis	5	5
Flexor digitorum superficialis	5	5
Flexor carpi ulnaris	2	1
Flexor digitorum profundus:		
Index and middle finger	5	5
Ring and little finger	2	1
Flexor palmaris longus	5	5
Extensor carpi radialis	1	1
Extensor digitorum communis	1	1
Extensor carpi ulnaris	1	1
Brachioradialis	1	1
Thenar eminence	5	5
Other intrinsic hand muscles	2	1

Muscle power grading: 0 = total paralysis, 1 = barely detectable contraction, 2 = not enough power to act against gravity, 3 = strong enough to act against gravity, 4 = still stronger but less than normal, 5 = full power.

hands but ignored it so as to concentrate on pushing. After a normal vaginal delivery of a normal birthweight baby girl the numbness and paraesthesia persisted and was accompanied by weakness of both arms. On examination she was found to have sensory and motor deficits attributable to bilateral radial and ulnar nerve palsies; the details are shown in the table. She also complained of slight tenderness in the lateral wall of both axillae. Over the next five days there was a slight improvement in the power of all her muscles, and the sensory symptoms and signs in the radial nerve distribution resolved bilaterally. Three weeks later her condition was virtually unchanged.

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Oxidative haemolysis after administration of doxorubicin

Deficiency of glucose-6-phosphate dehydrogenase (G6PD) is the most common enzyme deficiency in red blood cells and has been found in 11.2% of black men in hospital.¹ Infections and certain drugs may induce haemolysis in patients with this defect. We describe a patient with G6PD deficiency who developed acute haemolytic anaemia after treatment with doxorubicin.

Case report

A 58 year old black man was admitted to hospital for investigation of an enlarging thigh mass and increasing shortness of breath. Seven years previously G6PD A⁻ deficiency had been diagnosed, although he denied any knowledge of haemolytic episodes. On examination he had a firm mass measuring 16 × 20 cm on the right thigh. An enlarged right inguinal node was also present. Laboratory results showed a haemoglobin concentration of 14.8 g/dl (1.2% reticulocytes); chest x ray examination showed multiple

bilateral pulmonary nodules. Histological examination of a biopsy sample of the thigh mass suggested synovial sarcoma.

Because of the metastatic pulmonary lesions 150 mg doxorubicin was given by intravenous injection. Three days later the haemoglobin concentration fell suddenly to 10.6 g/dl (reticulocytes 4.1%). Results of Coombs tests were negative. Supravital staining (cresyl blue) of a blood film showed numerous Heinz bodies. In addition, there was evidence of both haemoglobinuria and haemoglobinemia. Over the next few days the haemoglobin concentration dropped to 9.2 g/dl (9.8% reticulocytes), but three weeks later it had returned to 14.0 g/dl (2.1% reticulocytes). At the patient's request no further chemotherapy was given, and he died three months after his initial presentation.

Comment

The sudden decrease in haemoglobin concentration after administration of doxorubicin strongly suggests that this drug was causally related to the acute haemolytic episode. Although a wide variety of treatments may induce haemolysis in patients with G6PD deficiency, I was unaware that antineoplastic agents might do so. None the less, metabolic insults to red blood cells have been described after exposure to cytotoxic drugs *in vitro*²⁻⁵; doxorubicin generates reactive oxygen compounds³ and methaemoglobin² in normal human red blood cells *in vitro*. Furthermore, in red blood cells deficient in G6PD doxorubicin poses a potent oxidant stress, as shown by the production of hydrogen peroxide and depletion of reduced glutathione.⁴ These findings support the suggestion that doxorubicin acts as an oxidant stress to both G6PD deficient and normal red blood cells. Thus patients with G6PD deficiency might be susceptible to haemolysis after receiving doxorubicin. Moreover, the possibility of oxidative haemolysis should be considered in any patient who shows an unexplained drop in haemoglobin concentration after treatment with this agent.

I thank Mrs G Congleton for her technical help in preparing the manuscript.

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³ Henderson CA, Metz EN, Balcerzak SP, Sagone AL Jr. Adriamycin and daunomycin generate reactive oxygen compounds in erythrocytes. *Blood* 1978;52:878-85.

⁴ Sagone AL Jr, Burton GM. The effect of BCNU and adriamycin on normal and G6PD deficient erythrocytes. *Am J Hematol* 1979;7:97-106.

⁵ Sponzo RW, Arseneau JC, Canellos GP. Procarbazine induced oxidative haemolysis: relationship to *in vivo* red cell survival. *Br J Haematol* 1974;27:587-95.

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Abnormalities of lymphocyte subsets in polymyositis

Polymyositis (dermatomyositis if the characteristic rash is present) is an inflammatory myopathy of unknown aetiology. Considerable circumstantial evidence suggests that immune mechanisms play a part in its pathogenesis.¹ The functions of the different cellular components of the immune system have recently become easier to study because of the availability of monoclonal antibodies that recognise single antigenic determinants. These antibodies are used widely to identify lymphocyte subpopulations, and analyses of the different subgroups with a fluorescence activated cell sorter have shown changes in the ratio of helper to suppressor cells in several diseases.² A balance between these lymphocytes is presumably necessary for immune homeostasis, and it has been postulated that immunologically mediated disorders may be due to aberrations in T cell immune regulation. We therefore studied the lymphocyte subpopulations in patients with polymyositis.

Patients, methods, and results

We studied 18 patients with proved polymyositis; 11 had a concomitant rash. As there is no evidence that the presence of skin lesions is due to a different aetiology we did not distinguish between dermatomyositis and polymyositis in this study. The patients were divided into three groups as follows: five patients had acute disease of less than three months' duration and were not receiving treatment; eight had had chronic active polymyositis for from nine months to 10 years and were taking 5-10 mg prednisolone on alternate days; and five had made a complete recovery from the disorder six months to 11 years previously and were not receiving treatment. In addition we studied 15 control subjects with various neuromuscular disorders—for example, the Guillain-Barré syndrome, myasthenia gravis, alcoholic myopathy, seven of whom were taking prednisolone 5-10 mg on alternate days, and 20 normal healthy volunteers.

Mononuclear cells were isolated from heparinised peripheral blood using Ficoll-Hypaque (Pharmacia Fine Chemicals) gradient density centrifugation and then stained with Ortho-mune monoclonal antibodies (OK) T3, T4, T8, or Ia1 (Ortho Diagnostic Systems Ltd, Buckinghamshire) followed by fluorescein conjugated antimouse IgG (sheep F (ab)2 antimouse Ig, fluorescein conjugated, New England Nuclear, Boston, Mass, USA) as described.³ Lymphocytes were analysed with a fluorescence activated cell sorter model IV (Becton Dickinson, Mountain View, California) using logarithmic amplification of the fluorescence signal⁴ and with 90° scatter to eliminate all contaminating monocytes. The Ia1 percentage obtained was considered to represent only B cells and activated T cells.

Analysis of lymphocyte subpopulations in 18 patients with polymyositis (group results expressed as means (SEM))

Case No	% reactivity with monoclonal antibodies				T4:T8 ratio
	T3	T4	T8	Ia 1	
<i>Acute polymyositis (n = 5)</i>					
1	53	49	10	27	5
2	78	70	10	16	7
3	41	39	4	53	10
4	61	57	13	25	4.4
5	56	43	27	19	1.6
<i>Chronic active myositis (n = 8)</i>					
6	87	82	6	5	14
7	93	82	10	6	8
8	76	68	14	13	5
9-13	78 (2)	60 (3)	18 (3)	14 (3)	2.8 (0.1)
<i>Recovered from polymyositis (n = 5)</i>					
14-18	71 (4)	52 (5)	24 (1)	19 (2)	2.1 (0.1)
<i>Neuromuscular disorders (n = 15)</i>					
	70 (3)	45 (2)	24 (1)	19 (2)	2.0 (0.1)
<i>Healthy controls (n = 20)</i>					
	75 (2)	49 (2)	25 (2)	12 (1)	2.1 (0.1)

The table shows the results. Patients with acute and chronic active disease had gross abnormalities in their lymphocyte subgroups: suppressor/cytotoxic cells were conspicuously reduced in both groups, total T lymphocytes and helper cells were decreased in patients in the acute phase, and helper cells were increased in patients with chronic active disease. The abnormalities persisted in patients with chronic active disease who were studied serially for three months.

Comment

These results show that lymphocyte subpopulations are disturbed in patients with acute and chronic active polymyositis, with a conspicuous decrease in the numbers of suppressor/cytotoxic cells. These findings are similar to those reported in other immunologically mediated disorders.² Some patients with multiple sclerosis show a similar loss of suppressor cells before a clinical relapse occurs, with reappearance of the cells when the disease remits.² In our cases the number of suppressor cells was reduced for at least three months in the patients tested serially. The findings documented here were not secondary to steroid treatment because the abnormalities were detected whether or not the patients were taking the drugs and no changes were present in control subjects taking a similar prednisolone dosage.

In a study of two children with dermatomyositis Bresnan *et al* reported a fall in circulating suppressor cells similar to the one shown here.⁵ Muscle biopsy specimens taken at the same time contained a mixed mononuclear cell infiltrate in which inducer (helper) cells, not suppressor cells, were most prominent. Our preliminary studies, on the other hand, have shown one case in which T8 cells were prominent in the inflammatory infiltrate between muscle fibres.

These findings confirm that there is a gross disturbance of immunoregulation in polymyositis.