November, 1970
Eighth Series. Vol. 3. No. 11
I. J. Med. Sc.

# TOXOPLASMIC POLYMYOSITIS

By B.McNICHOLL, M.D., F.R.C.P., D.C.H. and
D. UNDERHILL, M.B.

*Departments of Paediatrics, Regional Hospital and University College, Galway*

POLYMYOSITIS as a manifestation of acute acquired toxoplasmosis has been described in reports of two patients, by Rowland and Greer (1961) and Chandar, Mair, and Mair (1968). We believe that the present case is most probably of similar aetiology, on the basis of the serological findings.

*Case Report*: A 9-year old boy had an illness with a productive cough and an itchy rash on the trunk in the second week of February 1969. Two weeks later limb pains commenced and were accompanied by mild rigors, sweating, headache and occasional vomiting. Soon the pains became mainly muscular in nature, being at first most noticeable after walking and greatest in the calves and groins. At the peak of illness he found difficulty in raising his arms above his head, walked with difficulty and showed marked generalized weakness. During the week before admission some improvement occurred. He literally waddled into the out-patients consulting room on 13th March, when he showed marked generalized muscular weakness and tenderness, the latter being greatest in the calves, which were slightly swollen and reminiscent of the appearance in pseudo-hypertrophic muscular dystrophy. Weakness of the legs and pelvic musculature was clearly shown by his having to "climb" up on his legs with his hands when put sitting on the floor and by his inability to stand on one leg. Tendon jerks were absent, apart from feeble responses at both knees, but sensation appeared normal. Lymph-glands in the anterior cervical triangle, axillae and groins were slightly enlarged, whereas the liver and spleen were not; the optic fundi were normal then and subsequently.

*Investigations*

Haemoglobin was 13g./100 ml., W.B.C. 8,500 with 69% polymorphs, 20 %lymphocytes, 6% eosinophils and 5% monocytes; E.S.R. was 30 mm. in 1 hour (Westergren). Serum enzymes: creatine-phospho-kinase 20 international units (normal 0-60), lactic dehydrogenase 100 Wacker units (normal 30-140), glutamic-oxaloacetic-transaminase 25 Karen units (normal 15-45), alkaline phosphatase 30 King Armstrong units (normal 4-17). Serum proteins were 8.0g., globulins 4.0g. Negative serology included anti-streptolysin 0 titre, antinuclear factor and latex fixation. No serological or skin tests for toxocara or trichinella spiralis were made. Urinary creatinine was 450 mg. in 24 hours; creatine was omitted. Biopsy of the left gastrocnemius on 27th March showed no abnormality. The dye test titre for toxoplasma on 2nd April was 1/16,000, in August 1/4,096 and in November 1/1,024.

*Progress*

Four weeks after admission muscle power had improved markedly, he was walking normally and all tendon jerks were present but weak. He remained afebrile throughout and gained 1.3 Kg., being discharged in mid. April. No treatment was given, primarily because the result of the dye test was not received until two weeks after his discharge and then because of his apparent recovery. By August recovery appeared complete.

*Discussion*

Toxoplasmosis as the cause of the polymyositis can only be presumed on the evidence of the high initial dye test titre and the subsequent fall. Proof would require demonstration of toxoplasma gondii either in the patient's tissues or in those of innoculated animals. Beattie (1967) found antibodies to toxoplasma only rarely in children between 6 months and over 10 years and considered that a dye test titre over 1/1,000 was indicative of active or recent infection; Fleck and Ludlam (1965) regarded a titre of over 1/512 as probably significant. The negative muscle biopsy could be explained by its not being done until the recovery phase or else due to sampling an uninvolved area. The normal creatine-phospho-kinase, which was not estimated until just after the biopsy, would suggest that the myositis had become inactive at that time. The muscle biopsy in the case of Rowland and Greer (1961) did not show the organism, whereas in that of Chandar, Mair and Mair (1968) a terminal colony was found in one multinucleate cell. Involvement of muscle in toxoplasmosis is however probably frequent, judging by the frequency of myalgia in reported cases and by frequent findings of the organism in muscle in other cases, as reviewed by Rowland and Greer (1961).

The finding of antibodies to toxoplasma in about 30-40% of the adult population (Fleck and Ludlam 1965) suggests that infection with this organism is common and usually mild, or sub-clinical. (Attempts to investigate the family and surroundings of the present case were unsuccessful due to parental reluctance; serum obtained from the mother in November 1969 gave a dye test titre of 1/16). The incidence in the West of Ireland has not been studied as far as we know and we have recognized only 2 probable cases of acquired toxoplasmosis in recent years, since the dye test has been available to us for selected cases. In 1966 a titre of 1/512 was found in the terminal stages of fatal encephalitis in a boy of 12 years; autopsy was not permitted. In 1967 a titre of 1/16,000 coincided with a severe illness in a girl of 4 years, comprising marked cervical adenopathy, stomatitis, rash, pyrexia, myocarditis and encephalitis: she made a complete recovery; initial treatment was with penicillin and streptomycin followed by a course of pyrimethamine and sulphadiazine during convalescence when the positive dye test was known.

Acquired toxoplasmosis is most likely to present with lymphadenopathy, pyrexia, malaise, myalgia, rash and enlargement of liver and spleen. Beverley and Beattie (1958) found that toxoplasmosis was the probable cause of 7% of cases of "glandular fever" with negative Paul Bunnell reaction. Less common clinical pictures include myocarditis, pneumonia, encephalitis and

choroido-retinitis. It would also appear advisable to consider the diagnosis in cases of acute polymyositis and in acute illnesses with prominent muscular symptoms.

*Summary*

A nine year old boy who showed the clinical picture of polymyositis with marked weakness was found to have a toxoplasma dye-test titre of 1/16,000, which had had fallen to 1/1024 nine months later. He made a complete recovery without specific treatment. Muscle biopsy done after the acute phase was normal. It seems probable that the polymyositis was due to toxoplasma infection. Consideration of this cause in instances of polymyositis or severe muscular pain is advised.

*Acknowledgment*

We wish to thank Dr. G. B. Ludlam for the dye tests and much helpful information, Professor J. D. Kennedy for the histological report, and Dr. T. O'Shea for endeavouring to investigate the family.

*References*

Beattie, C. P.: 1967. Recent advances in Medical Neurobiology, edited by A. P. Waterson, London.

Beverley, J. K. A. and Beattie, C. P.: (1958). Lancet, 2, 379.

Chandar, K., Mair, H. J., and Mair, N. S.: (1968). Brit. Med. J., 1, 158.

Fleck, D. G. and Ludlam, G. B.: (1965). Brit. Med. J., 2, 1239.

Rowland, M. D. and Greer, M.: (1961). Neurology, 11, 367.