

Local Tetanus: a Case Report

AG Falade*, OJ Fatunde*, KO Ajayi*, OT Aluko*,
AA Adeyemo* and JB Familusi**

Summary

Falade AG, Fatunde OJ, Ajayi KO, Aluko OT, Adeyemo AA and Familusi JB. Local Tetanus: a Case Report. *Nigerian Journal of Paediatrics* 1994; 21: 63. Local tetanus following intramuscular injection into the gluteal muscle, in a ten-year old female patient is reported. The patient presented initially at a private clinic with fever and was treated for presumed malaria with intramuscular injection given into both gluteal regions. Later development consisted of stiffness of the left lower limb. Diagnosis of local tetanus was made, based on provocative spasms of the underlying left gluteal muscles. The diagnosis of tetanus became more obvious a few days later, when the spasms were spontaneous and generalized. During the recovery, the spasms became localized again to the left lower limb.

Introduction

LOCAL (or localized) tetanus, a rare condition, is defined as tetanus that affects mainly the region of the body bearing the contaminated wound.¹ To the best of our knowledge, no case of local tetanus has been previously reported in an African child, despite the high incidence of generalized tetanus throughout the continent.²⁻⁷ The present communication which concerns a 10-year old female patient, is reported because of the rarity of the condition, as well as the generalized tetanus which later manifested in the patient.

University College Hospital, Ibadan

Department of Paediatrics
* Senior Registrar
+ Lecturer
++ Professor

Correspondence: AG Falade

Case Report

RE, a 10-year old girl, presented at the Children's Emergency Ward, University College Hospital (UCH), Ibadan, with a history of fever for nine days and stiffness of the left lower limb for one day. At the onset of the fever, a provisional diagnosis of malaria was made at a private medical clinic where she received three intramuscular injections in the gluteal regions. Within 24 hours of the injections, she developed pains in the left lower limb and later had difficulty in walking. Eight days after the injections, she developed stiffness of the left lower limb and as the fever persisted, the parents brought her to the UCH for further management. Past medical history revealed that the patient was vaccinated with BCG during the first week of life and she had also received three doses of diphtheria, pertussis and tetanus

(DPT) vaccine and a dose of oral polio vaccine during infancy. No tetanus toxoid booster had been received.

Physical examination revealed an acutely ill and febrile child, (temperature, 38.3°C), weighing 29kg; she was mildly dehydrated. There was increased tonicity of the left lower limb with plantar flexion of the left foot. There were no swelling, sores or areas of tenderness on the gluteal regions. A provisional diagnosis of dystonia of the left lower limb was reached because the patient was reported to have received several doses of a phenothiazine (chlorpromazine) for restlessness prior to coming to UCH. Subsequent evaluation revealed spasms of the underlying muscles of the left buttock and lower limb. Because of this finding, a diagnosis of localized tetanus was made and therefore anti-tetanus therapy, comprising oral chlorpromazine (12.5mg), diazepam (10mg) and phenobarbitone (30mg) each eight hourly, was started. Anti-tetanus serum (ATS), 10,000 i.u. and tetanus toxoid, 0.5ml, *statim* followed by 300,000 i. u. intramuscular procaine penicillin once daily for one week, were added to the treatment.

Local spasms of the muscles of the left lower limb became more obvious during the following two days and these persisted despite repeated injections of intramuscular paraldehyde. Three days after admission, generalized spasms developed and other characteristic features of generalized tetanus, including trismus and risus sardonicus occurred. The patient developed profuse sweating, tachycardia and urinary retention that necessitated bladder catheterization. The dose of chlorpromazine and diazepam was increased to 15mg of each eight hourly, oral pyridoxine, 100mg thrice daily and propranolol 20mg twice a day. The spasms gradually lessened and became localized again to the left lower limb by the eighth day of admission, but completely disappeared

on the thirteenth day of admission. Fever however, persisted; a tender swelling of the left knee joint developed, but the knee joint aspirate was dry. Culture of a catheter specimen of urine grew *Escherichia coli* (*E. coli*) which was sensitive to ceftazidime and gentamicin. Blood culture was sterile. The fever persisted despite administration of cefotaxime 500mg six hourly for seven days, but it disappeared following administration of gentamicin given for thirteen days. She was discharged with a moderate contracture of the left foot after 37 days of hospitalization.

Discussion

Local tetanus is said to be uncommon during childhood^{8,9} and although its clinical presentation is variable, it produces pain and continuous rigidity and spasms of muscles around the site of initial injury in its most typical form. These features usually persist for weeks and resolve without sequelae, but may occasionally be followed by the development of generalized tetanus.⁹ The present case is thus, an example of a local tetanus that was followed by generalized tetanus. As the patient had received intramuscular injections on the buttocks nine days prior to the onset of symptoms, it is most likely that the portal of entry for the causative organism was the left gluteal region, while the early stiffness of the left lower limb was the first symptom of local tetanus.

The pathogenesis of local tetanus has been the subject of many theories.¹⁰⁻¹² The neurotoxin of tetanus (tetanospasmin) is said to travel by retrograde axon transport to reach the central nervous system, where it is bound to cerebral gangliosides. The principal effect of the toxin is to increase reflex excitability in the neurons of the spinal cord, brain stem and cortex by blocking functions of inhibi-

tory synapses. This results in the spasms of tetanus which can occur spontaneously, or as a result of external stimuli. Such a mechanism provides satisfactory explanation for the phenomenon of local tetanus and subsequent generalized tetanus. When local tetanus occurs, as in the present case, it results from the uptake and retrograde transport from the motor nerve terminals to the spinal cord and brain stem of tetanospasmin that has escaped from the vasculature into the extracellular fluid within the initially affected muscle.¹³ It is likely that the spasms became relocalized in the left lower limb during recovery because the concentration of the toxin would be highest and persist longest around the portal of entry of the causative organism.

A notable feature in our patient was the development of a tender swelling around the left knee-joint during the course of the disease. Aspiration of the joint space was dry, thus excluding septic and/or haemorrhagic effusion into the joint space. While the cause of the swelling remained undetermined, it is likely that trauma to the articular, or periarticular structures caused by intense and continuous spasms of the surrounding muscles might have been a contributory factor. In support of this suggestion is the known occurrence of compression fractures and intramuscular haematomas in severe generalized tetanus.⁹ The fact that the swelling resolved after two weeks makes myositis ossificans, another rare complication of tetanus, unlikely.^{14 15} The severity and prolonged period of spasms in our patient was probably related to the delayed diagnosis which allowed production of large quantities of tetanospasmin before therapy was commenced. Earlier diagnosis and prompt treatment would probably have shortened the course of the illness and prevented the urinary tract infection.

Acknowledgement

The authors thank all those who took part in managing the patient.

References

- 1 Simons ER. Local tetanus. *John Hopkins Med J* 1981; 149: 84-8.
- 2 Tompkins AB. Neonatal tetanus in Nigeria. *Br Med J* 1958; 1: 1382-5.
- 3 Idoko A. Neonatal tetanus in Benue-Plateau State. *Nig J Paediatr* 1975; 2: 47-51.
- 4 Hendrickse RG and Sherman PM. Tetanus in childhood: Report of a therapeutic trial of diazepam. *Br Med J* 1966; 2: 860-2.
- 5 Blankson JM. Problems of neonatal tetanus as seen in Ghana. *Afr J Med Sci* 1977; 6: 1-6.
- 6 Adeuja AOG and Osuntokun BO. Tetanus in adult Nigerians. A review of 503 patients. *E Afr Med J* 1971; 48: 683-6.
- 7 Ayim EN. The management of tetanus - A study of 51 cases treated at Muhimbili Hospital, Dar es Salaam. *E Afr Med J* 1972; 49: 170-6.
- 8 Weinstein L. Tetanus. *N Engl J Med* 1973; 289: 1293-6.
- 9 Vaughan VC, Mackay RJ and Behrman RE. Tetanus. In: Vaughan VC, Mackay RJ and Behrman RE, eds. *Nelson Textbook of Pediatrics*. Philadelphia: W B Saunders Company (publishers), 1979: 803-7.
- 10 Stockel K, Schwab M and Nelson Thoenen H. Comparison between the retrograde axonal transport of nerve growth factor and tetanus toxin in motor, sensory and adrenergic neurons. *Brain Res* 1975; 99: 1-16.
- 11 Price DL, Griffin J, Young A, Peak K and Stocks P. Tetanus toxin: direct evidence for retrograde intra-axonal transport. *Science* 1975; 188: 945-7.
- 12 Griffin JW, Price DL, Engel WK and Drachman DB. The pathogenesis of reactive axonal swellings: role of axonal transport. *J Neuropathol Exp Neurol* 1977; 36: 214-27.
- 13 Price DL and Griffin JW. Tetanus toxin. Retrograde axonal transport of systematically administered toxin. *Neurosci Lett* 1977; 4: 61-5.
- 14 Femi-Pearse D and Olowu AO. Myositis ossificans - a complication of tetanus. *Clinic Radiol* 1971; 22: 89-92.
- 15 Diop Mar I, Sow A and Alou A. On 12 cases of para-osteopathy noted in tetanus. *Bull Soc Med Afr Noire Langue Frse* 1973; 18: 95-104.