fo

Relapsing tetanus (a case report).

Bhatt AD, Dastur FD

Year : 1981 | **Volume** : 27 | **Issue** : 3 | **Page** : 184-6

:: Introduction

Chronic tetanus, relapsing or recurrent tetanus are different terms which are used to describe conditions as presented by our patient. Recent knowledge[3] of the circulation and fate of tetanus toxin in the body has led to a better understanding of the disease process which justifies our case to be termed as one of relapsing tetanus.

:: Case report

A 60 year old female was admitted with inability to open the mouth and stiffness of the body. She had sustained an injury to the right heel with a fish bone 10 days prior to admission. She had never been immunised in the past against tetanus. Physical examination confirmed the findings; but there was no obvious scar over the injury site.
She was given 20,000 I.U. of anti-tetanus serum intravenously and 10 Lt of tetanus toxoid intramuscularly on admission. She was covered with benzathine penicillin. 1,200,000 U. intramuscularly and put on diazepam 50 mg, 2 hourly. She worsened after 48 hours and developed spasms, tachypnoea and chest secretions for which a tracheostomy was done. After tracheostomy she was given intermittant positive pressure respiration with a Bird Mark 8 Respirator and intravenous curare to abolish spasms. She improved and was taken off the respirator after 48 hours. She continued to make a good recovery. Her tracheostomy was removed 3 weeks after admission. During this period she was on changing antibiotics for a chest and urinary tract infection which totally recovered. She was able to walk by the end of one month. At this time she was given a booster injection of 10 L tetanus toxoid. She was kept in the ward for complete mobilisation and treatment of a bed sore, which subsequently healed. Her stiffness had totally disappeared by the end of 40 days.
On the 46th day, she complained of, stiffness in the right foot (the site of injury). She was again put on diazepam without significant effect. She continued to progress developing stiffness in the right lower limb, right side of the abdomen, followed by spread to the other side. Later on, she developed ascending stiffness of the whole body and generalised spasms. Despite a second tracheostomy and vigorous treatment of a chest infection she continued to worsen and expired on the 59th day after admission.
Investigations
Her tetanus antibody titre taken on the 54th day was 0.0092 I.U./ml, protective titre being 0.01 1-U./ml. Rest of the investigations were not significant except for polymorphonuclear leucocytosis compatible with her chest and urinary tract infection.

:: Discussion

Chronic tetanus, relapsing or recurrent tetanus are terms used to describe conditions as in the patient under discussion. One of the earliest case reports in the Military Medical Manual by Marie in 1918 (as quoted by Gordon et al[2]) describes a soldier who had 2 injuries in the thigh within a period of 6 months. He was given tetanus antitoxin intravenously as well as locally at the time of these injuries. After a lapse of 130 days, he developed paroxysms of painful contractures and intermittant jerks localised primarily in the abdomen and thoracic regions. The symptoms progressed and he developed rigidity. At this point he was given large doses of antitetanus toxin at an interval of a few days. Some days later a shell splinter was removed from the left thigh. From then on, slow progressive resolution of symptoms was observed with complete recovery two and a half months from the onset of "painful" contractures. This was termed "late, partial tetanus infection localised in the abdomino-thoracic muscles" by the author. Many other cases in the literature have been termed chronic tetanus, relapsing or recurrent tetanus. With the modern knowledge of electrophysiology,[2] these may, in fact, have been cases of Stiffman syndrome, Isaac's disease or McArdle's disease. The problem of seperating such cases from the atypical forms of tetanus continues, since most of the references on this are quite old and the diagnosis is difficult to confirm with the data supplied. Nevertheless, our experience includes isolated patients who have developed tetanus again after complete recovery and discharge from the hospital. It is difficult to give them any definite label but the case under discussion justifies the use of the term relapsing tetanus.
A brief idea of spread of tetanus toxin in the body will be helpful in understanding the pathogenesis of the case under discussion. To produce generalised tetanus, toxin gains access to the blood stream directly or through lymphatics.[1] It

gets carried to all parts of the body including muscles where it shows a strong affinity for nerve endings. Its further passage is along the nerves, which provide the final common pathway to the central nervous system. The length of the nerve determines the time required for ascent and explains why the disease is first seen in muscles supplied by the cranial nerves. Though the pathway of the toxin spread is ascending, the symptoms start with trismus and descend the body to cause generalised rigidity and spasms, producing what is known as tetanus descendans. **However, if the toxin in the blood is neutralised by anti-toxin, the toxin can only ascend to the brain or spinal cord by the local neural pathway.**[4] **There, it abolishes central inhibition. This leads to local muscle stiffness which slowly ascends to other parts of the body as the toxin spreads upwards in the spinal cord. This is local tetanus which leads to tetanus ascendans.**
It appears that in our patient the antitoxin level on the 54th day (0.0092 I.U./ml) was enough to prevent a generalised form of tetanus (protective titre being = 0.01 I.U./ml). Toxin production in deep wounds can continue for 9 days or longer,[5] which probably was the situation during her first episode of tetanus.

We would argue that the spores in the localised, deep wound on the sole of the right foot might have renewed toxin production when the patient started to walk on the 40eth day. The pressure exerted on the sole of the foot could have forced the spores deeper into an avascular area and provided the anaerobic conditions necessary for germination.

In the presence of circulating antitoxin, the toxin could take only the pathway of regional motor nerves. This contention is supported by the sequence of tetanus in the second episode. It started in the right foot spreading up the right lower limb, right side of the abdomen, then spreading to involve the opposite side and finally ascending to involve the rest of the body. This unfortunate woman had no obvious wound which would have permitted debridement to prevent this relapse which ended fatally.

:: References

1. Dastur, F. D. and D'Sa, J.: Tetanus-Present Knowledge and Experience, In, "Progress in Clinical Medicine in India." Editor: M. M. S. Ahuia, Third Series, Arnold-Heinemann, New Delhi, 1979, p. 71.
2. Gordon, E. E., Janusko, D. M. and Kaufman, L.: A critical survey of the stiffman syndrome. Amer. J. Med., 42: 582-599, 1967.
3. Kryzhanovsky, G. N.: The mechanism and action of tetanus toxin. NaunynSchmiedeberg's Arch. Pharmacol., 276: 247-270, 1973.
4. Kryzhanovsky, G. N.: Present data on the pathogenesis of tetanus, in, "Progress in Drug Research. Tropical Diseases II." Edited by Ernest Tucker; Birkhaiser Verlag, Basel, 1975, Vol. 19, pp. 301-302.
5. Smith J. W. G.: Tetanus and its prevention, In, "Progress in Drug Research. Tropical Diseases II". Edited by Ernest Tucker, Birkhaiser Verlag, Basel, 1975, Vol. 19, pp. 394-395.