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Case report

TETANUS TRISMUS IN A 2 YEAR OLD CHILD: CASE REPORT

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ABSTRACT

Tetanus is still a major cause of mortality and morbidity in developing countries. It occurs in children mainly in the unimmunized, due to parental ignorance and objection to vaccination. This potentially fatal disease caused by a neurotoxin, tetanospasmin released from wounds infected with *Clostridium tetani*, an anaerobic gram–positive bacillus. As tetanus becomes less common, cases are likely to be misdiagnosed or go unrecognized. In this case report, we present a case of tetanus in a partially immunized 2 year old girl who presented with trismus. She was treated with the recent recommendations and adequate supportive care. Detection of tetanus at a very early stage can favor lifesaving interventions. Trismus, infected wound and partially immunized/unimmunized status of a child were the key features leading to the prompt diagnosis and early treatment.

Keywords: Tetanus, trismus, tetanospasmin, tetanus toxoid

INTRODUCTION

Tetanus is a preventable neurologic disease caused by the bacterium *Clostridium tetani*, spores of which live in the soil, dust, and environment and infect via a cut or puncture in the skin or mucosa. Unfortunately, cases of tetanus are still common in many parts of the world. The World Health Organization estimates that 58,000 newborns died of tetanus in 2010.[1] It is found commonly in warm climates and highly cultivated rural areas [2] and remains a life-threatening disease that continues to have a high prevalence in developing countries due to social problems such as poor education of the parents when parents are not ready to immunize their children. *C. tetani* spores germinate to produce an exotoxin, tetanospasmin, which causes rigidity and spasms of voluntary skeletal muscles [2]. The different forms of tetanus are neonatal, generalized, localized and cephalic. The most common forms are generalized and neonatal tetanus.[2] It usually occurs in neonates and persons aged older than 65 (as a result of waning immunity) whereas, the incidence occurs equally in male and female persons.[3] Neonatal tetanus remains a significant problem in developing countries due to poor umbilical stump hygiene and lack of maternal antibody as a result of inadequate immunization.[4] Prevention is possible with immunization. The regimen varies depending on patient's age and prior exposure to tetanus vaccine.[5] Incidence varies with level of immunization within a population. The highest rates are in resource-poor countries with non-universal immunization practices and in economically deprived nations due to poor immunization and unhygienic practices whereas, it is a forgotten disease in developed countries since many practicing primary care physicians have not seen a single case in their career. The diminished incidence in the developed world may probably due to the introduction of primary vaccination.[5] The management of tetanus aims at removing the source of tetanospasmin, neutralising circulating toxin, and providing adequate

supportive care for muscle spasms, respiration and autonomic instability and timely recognition of this serious disease helps in better outcome.[5,6]

CASE REPORT

A 2-year-old girl, first sibling from a poor socioeconomic family of a non-consanguineous couple, admitted in the Department of Paediatrics, Amala Institute of Medical Sciences, Thrissur, Kerala, with difficulty in opening of her mouth since 3 days. History revealed that she was partially immunized; only BCG and 0 dose of OPV were taken. On examination, she had lockjaw (Fig.1), low grade fever and healing pyoderma on her upper and lower limbs.

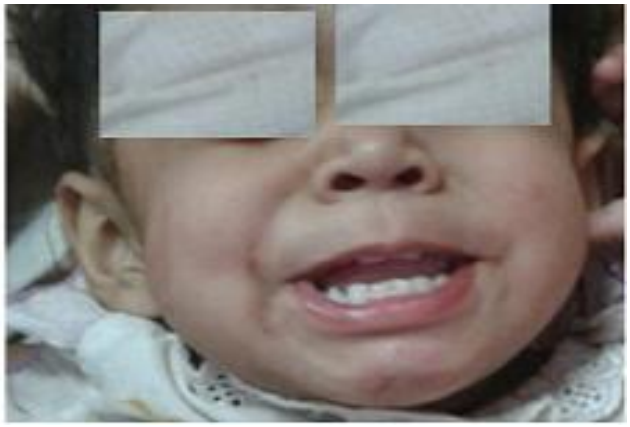

Fig.1: Child on admission with trismus and sick

She was anxious, irritable, tonic bite present; spatula test was positive, deep tendon reflexes were exaggerated whereas, plantar response and sensorium were normal. Systemic examinations were normal. Tetany which usually manifest as Chvostek sign and Trousseau sign were absent in our child. Laboratory investigations showed hemoglobin (6.7 g/dl) with low indices, total leucocyte count (11,550/cumm), neutrophils (75%), lymphocytes (22%), platelets (210000/µl), ESR (35mm at1 hr); lumbar puncture was done and CSF study was normal; and study on blood culture and sensitivity was sterile. The diagnosis of tetanus was made based on the trismus and infected wound and the history of partial immunization. She was treated with human tetanus immunoglobulin (TIG), tetanus toxoid–containing vaccine, wound cleaning and injection of crystalline penicillin for 10 days. Oral diazepam was given for muscle relaxation. The patient did not progress to severe generalized tetanus with autonomic instability. She was given excellent supportive care, Ryle's tube feeds started on 3rd day and also packed red blood cell transfused on 3rd day. After completion of 10days of intravenous antibiotics, she was discharged on day 11 with improvement in clinical conditions (Fig.2) on multivitamins, hematinics and deworming drugs with an advice to follow-up for catch up vaccination.

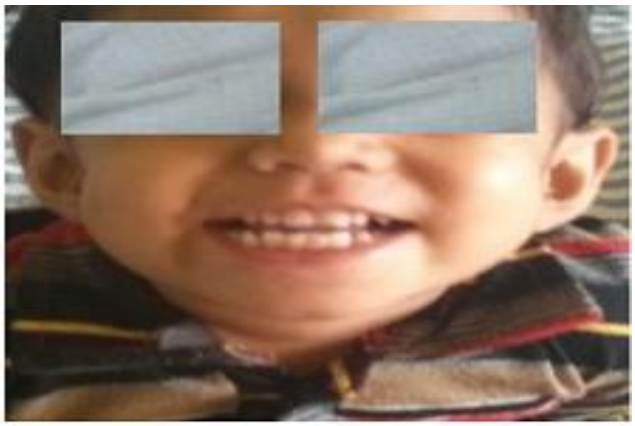

Fig 2: Child after 10 days of treatment.

DISCUSSION

Tetanus is caused by a neurotoxin, tetanospasmin released from wounds infected with *Clostridium tetani*, a Gram–positive bacillus. [2,3] The bacteria enter the body through cuts and abrasions to the skin, but will multiply and transform into vegetative forms only in an environment that is oxygen-free. Deep puncture wounds and wounds with a lot of dead tissue provide an oxygen-free environment for the bacteria to grow, especially in the presence of a foreign body, crush injury and suppurative infections. Among the two exotoxins such as tetanolysin and tetanospasmin produced by *C. tetani*, tetanospasmin is the main toxin that gains access to the blood stream directly or through lymphatics and ascends along the nerves to central nervous system. The intial symptom such as trismus can be ascribed to the ascending spread of the toxin and its action on muscle supplied by the cranial nerves. At the neuronmuscular junction mainly at the presynaptic nerve terminal it prevents the release of inhibitory neurotransmitters glycine and gamma amino butyric acid that can also lead to uncontrolled contraction of muscles. The descending of the toxin explains the mechanism of generalised rigidity and spasms.[7] The diagnosis is based entirely on clinical presentation and immunization history. Symptoms such as trismus and risus sardonicus appear 4 to 20 days after wound contamination. Trismus is a firm closing of the jaw due to tonic spasm of the muscles of mastication from disease of

the motor branch of the trigeminal nerve. It is usually associated with tetanus, also called lockjaw. Risus sardonicus or rictus grin is a highly characteristic, abnormal, sustained spasm of the facial muscles that appears to produce grinning. The name of the condition derives from the appearance of raised eyebrows and an open "grin" - which can appear sardonic or malevolent to the lay observer - displayed by those suffering from these muscle spasms.[2,3]

Depending on the severity of the disease, the painful contractions can, in a few days or even hours, spread to the whole body. Death can follow due to respiratory failure.[8] The differential diagnosis includes meningitis, encephalitis and rabies (see these terms), as well as peritonsillar abscess, medication (phenothiazine, metoclopramide) -induced dystonic reactions, subarachnoid hemorrhage, hypocalcemic tetany and acute strychnine poisoning.[2-,4] The complications of tetanus are laryngospasm, aspiration pneumonia, nosocomial infections (common because of prolonged hospitalization), fractures of the spine or long bones (from sustained contractions and convulsions), acute renal failure (due to rhabdomyolysis), pulmonary embolism, hypertension and/or an abnormal heart rhythm (due to hyperactivity of the autonomic nervous system) and sudden cardiac death.[3,6]

Treatment of tetanus aims at airway maintenance, prevention of further toxin absorption, relieving clinical features like spasms, controlling autonomic instability and antibiotics.[3,9] The main method of prevention of tetanus is by adequate immunization using tetanus toxoid.[10, 11] Measures such as cleansing of new bites, burns, and wounds and prophylaxis with antibiotics and tetanus immune globulin (TIG) should be instituted if an asymptomatic, newly injured patient is not adequately immunized.[9,10] The side effects of immunization with tetanus toxoid adsorbed intramuscularly such as mild fever, joint pain, muscle aches, nausea, tiredness, or pain/itching/ swelling/ redness at the injection site may occur. Rarely, other side effects such as tingling of the hands/feet, hearing problems, trouble swallowing, muscle weakness and urticaria or neurologic complications. [3]

Pharmacological eradication of *C. tetani* bacilli can be achieved by either penicillin or metronidazole based regimens.[9] Treatment is symptomatic and aims to control contractions with high doses of myorelaxant drugs or even prolonged curarization until elimination of the toxin.[2,6] The role of Benzodiazepine derivatives in the sedation and muscle relaxation in the ICU during the course of generalized tetanus has prevented rapid progression.[12,13] All patients should receive a complete course of immunization with tetanus toxoid once recovered, as the disease does not induce protective antibodies. Bhatt et al described a case of relapsing tetanus in a 60 year old female.[7]

For wounded individuals and with infected wounds with an uncertain vaccination status, systematic administration of specific gamma globulins prevents the disease. [10,11] The incubation period of tetanus may be up to several months, but is usually about eight days.[14] Symptoms usually occur within 8 to 12 days. The risk of wound infection is constant. Prognosis is variable. The disease lasts 2 to 4 weeks. With mortality varying between 20 and 80%, depending on disease severity, patient age, and availability of intensive care facilities.[2]

Amornpol et al.[15] identified tetanus in a 73–year–old man with symptom of locked jaw for one day. All standard treatments were given. However, the patient eventually progressed to severe generalized tetanus with autonomic instability as TIG does not neutralize toxin that has already bound to nerve endings. At this stage, the main treatment is supportive care; early protection of the upper airway, adequate ventilation, control of muscle spasms, and limiting the consequences of autonomic dysfunction which is the most common cause of death in ventilated patients with severe tetanus. Magnesium sulfate was postulated by the team as the drug of choice to control cardiovascular instability.[12,15]

In our case, clinical findings of trismus and infected wound and unimmunized status of the child were key features leading us to prompt diagnosis and emergent treatments, including Tetanus immunoglobulin, tetanus toxoid–containing vaccine, wound cleaning and antibiotics. Fortunately, the child did not progress to severe generalized tetanus with autonomic instability as is seen sometimes[8,15] requiring further advanced treatment.[12]

CONCLUSION

Tetanus is still a major cause of mortality and morbidity in developing countries and occurs in children mainly in the unimmunized, due to parental ignorance and objection to vaccination. Since tetanus

is a very rare case, and a child with trismus is vulnerable to progress, if missed. Physician education is vital in detecting tetanus at a very early stage, so further lifesaving interventions can be done and prevent rapid clinical deterioration.

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Conflict of interest: No

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