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Case Report

Tetanus after a minor injury leading to death in a previously non-immunized, elderly, Norwegian woman

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ABSTRACT

Tetanus vaccination is part of the Norwegian childhood vaccination program. An elderly woman injured her arm and leg after a minor fall on her outdoor stairs. Two weeks later she presented with trismus. This developed into tetanic spasms, obstructed airways and the need for a tracheostomy. She died 14 days later due to pneumonia and multi-organ failure. ELISA for tetanus toxoid IgG was negative, probably because the patient was born before the introduction of tetanus vaccination in the Norwegian childhood vaccination program. Lack of adherence to the vaccination programs should be considered in patients presenting with symptoms resembling diseases they normally would be protected from. Although the patient presented with typical symptoms the diagnosis was not suspected initially, probably due to the rareness of this disease in Norway.

Introduction

The tetanus vaccine has been available in Norway since 1945 but has only been a part of The Norwegian Childhood Immunization Program since 1952. All children residing in Norway are offered participation in this program, which includes immunization against tetanus starting at the age of 3 months and finishing at the age of 16 years. Vaccination is not compulsory. The vaccination coverage is 91–95% [1].

Case report

The patient was a 94-year old woman with macular degeneration, hypertension and depression. She used hydrochlorothiazide–amilorid and escitalopram and received monthly intraocular injections of bevacizumab. The patient lived at home and cared for herself but had home based nursing help once a week.

She first contacted her dentist 1 week prior to admission to hospital due to trismus and suspicion of a dental infection. No dental infection was found and no further dental care was given. The patient then developed difficulties speaking and a left sided spasticity. She was admitted to her local hospital with a suspicion of a cerebrovascular event.

At her local hospital a computer tomography scan of the brain and facial skeleton was performed but no intracerebral bleeding or infarction was found and no abnormalities of the facial bone structure was seen. CT revealed chronic dental infection of the teeth 14, 26 and 45.

Under the suspicion of a neurological disease due to trismus and spasticity in her facial muscles, the patient was transferred to the Neurological Department at the Oslo University Hospital. At the University Hospital the patient was seen by an oral surgeon but he was not able to open the patient's mouth for further examination. Diazepam did not relax the patient's facial muscles and evaluation was done by exterior observation, evaluation of the CT-scans and palpation of the face and jaw.

A neurologist was also not able to open the patient's mouth but had the impression the patient was awake, alert and oriented for time and space. During the examinations the patient suffered seizures with neck and facial cramps, and experienced difficulties swallowing and breathing. Tetanus was suspected and the patient was moved to the Intensive Care Unit (ICU).

In the ICU, the patient communicated by nodding and shaking her head and her responses were appropriate. Laryngeal spasms and airway compromise intervened and a tracheostomy was

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performed. Shortly after the tracheostomy, the patient experienced a spastic seizure (opisthotonus) involving the whole upper part of her body.

After the tracheostomy, the patient was easier to ventilate and monitor so her previous history could be assessed more thoroughly. It appeared that 1–2 weeks prior to the start of her symptoms she had fallen on her outdoor stone steps and hurt/grazed her left arm and right leg. The wounds were quite superficial and she had not sought medical attention for this. We therefore contacted the plastic surgeon who performed wound excision. Samples of the excised skin were sent for culturing. Benzylpenicillin was started and tetanus immune globulin (3000 IU) was administered. The patient was sedated and kept on a ventilator. She was kept in a dark room with as few stimuli as possible.

The next day the patient had another seizure with extension of the neck, trismus and spasticity in all extremities after minor stimulation. A chest X-ray showed possible infiltration in her right lung and C-reactive protein (CRP) increased from 7 to 16 mg/L. Pneumonia possibly caused by aspiration was suspected and metronidazole was added to penicillin. EEG did not show any epileptic activity.

During the next 2 days her CRP increased to 80 mg/L and she became febrile. Lung physiotherapy was not possible due to increased spasms during all manipulation of the patient. Sedation was increased. Culturing of samples from her wounds on her extremities showed *Staphylococcus aureus* and clindamycin was added to the antimicrobial regimen.

Upon admission to the ICU when tetanus was suspected, serological testing was performed. Specific IgG antibodies to tetanus toxoid were measured by an ELISA technique [2]. The result of the analysis showed that the tetanus toxoid IgG antibody level was <0.1 IU/ml – i.e. the patient had no immunity to tetanus.

Eight days after admission, the CRP values increased to 274 mg/L and the frequency of the spasms increased. The antibiotic regimen was changed to piperacillin–tazobactam and the patient was given more sedation. Pneumonia was still suspected as the main complicating infection. After this the situation rapidly deteriorated and she died 12 days after admission from multiple organ failure.

Discussion

Tetanus is a rare disease in Norway. From registration started in 1977 to the current case in 2012 only 49 cases had been reported to the Norwegian Surveillance System for Communicable Diseases (MSIS) at the Norwegian Institute of Public Health (FHI), of which three cases were fatal (this case included). A major contributor to this low number is probably the immunization program, which was introduced in Norway in 1952. The incidence of tetanus has dropped dramatically after the introduction of the tetanus toxoid vaccine. The US Centers for Disease Control and Prevention (CDC) reported for the period 2001–2007 an overall annual incidence of 0.10 and 0.23 cases/million among individuals >65 years of age [3]. The incidence of tetanus in European countries is also low and has declined due to vaccination programs [4,5]. Until the 1950s 10–15 Norwegians died annually from tetanus [1]. Today the disease is most common among older people with penetrating skin injuries and where the wound surface has come in contact with infected soil. Most patients born before 1950 have not been primary vaccinated against tetanus. A recent published article from the United Kingdom highlights the importance of tetanus risk management during wound management [6].

The diagnosis of tetanus is mainly based on clinical criteria. A serum IgG antibody level to tetanus toxoid of more than 0.1 IU/ml measured with ELISA technique is considered protective. However, tetanus disease has occurred in individuals with antibody levels of 0.15 IU/ml measured with ELISA [7]. There is no specific test to diagnose tetanus and the organism is most often not recovered from the site of infection. Our diagnosis of tetanus seems convincing, with the absence of antitoxin antibodies and a typical history and symptoms.

Although the diagnosis of tetanus is usually obvious there are several differential diagnoses:

1. From an epidemiological point of view a cerebrovascular event would be much more likely and it would be natural to look for cerebral infarcts or bleeding in a patient with hypertension presenting with (what was considered to be) aphasia and spasticity of one part of the body. Although a CT-scan of the head was performed a recent cerebral infarction is often not visible the first days after the event. In this case the patient contacted the hospital more than 1 week after the presentation of symptoms and signs of infarction (and bleeding) would probably have been visible on CT-imaging. Cerebral infarcts/bleeding are common diagnoses in the elderly patient with hypertension and recent development of neurological symptoms. And the suspicion of this should probably be one of the first diagnoses to be evaluated.
2. Trismus due to dental infection was the original diagnosis and may be confused with (cephalic) tetanus. Muscle spasms would normally not progress in a patient with dental infection.
3. Malignant neuroleptic syndrome can present with muscular rigidity. Patients with this condition will normally have a history of recent intake of a neuroleptic drug, many will have a fever and an altered mental status.

Our patient's symptoms were bilateral with a typical facial expression, trimus and spastic seizures. The diagnosis of tetanus was still not obvious probably due to the low prevalence of this condition in Norway. The recent history of trauma was also delayed since the injuries were small and relatively innocent. Interestingly the primary site of entry of the infection, as in this case, might be quite superficial and the wound might have healed at the time of tetanus development. The incubation period is between 1 day and several months with most cases occurring within 8 days [8–12] depending on the amount of the inoculum and the distance from the introduction of spores through the skin and the central nervous system. Our patient had wounds on her arm and leg which were consistent with an incubation period of 1–2 weeks prior to symptom development. Her skin injuries were superficial but they did penetrate the skin and left the tissue beneath open for microbial invasion. Clostridium tetani will not grow in healthy tissue and penetrating injuries or devitalized tissue is necessary for tetanus toxin to be released in the human body. It is not the clostridial bacterium but the released toxin (tetanospasmin) that causes the symptoms. In many cases the wound has healed at the time of presentation of the muscular symptoms.

The wound debridement was probably done too late since the wounds had more or less healed at the time of presentation of the symptoms. The role of antibiotics is also probably small in the management of tetanus but in the absence of better alternatives most patients receive penicillin and/or metronidazole.

One of the major pharmacies in Oslo has at all times an emergency supply of human tetanus immune globulin (HTIG). This was administered within 2 h of the clinical diagnosis of tetanus being made. HTIG binds to unbound tetanus toxin and neutralizes this. The already bound fraction of toxin is irreversibly bound and is not available for neutralization [8,13].

One of the major problems in the treatment of our and many other tetanus patients is the obstruction of the airways, and airways management should always be a top priority. In our case

the patient needed tracheostomy to secure the airways. Normal intubation was not possible and not feasible due to her trismus.

In developed countries most patients survive if intensive care facilities and modern supportive treatment is available [14]. In developing countries the fatality rate is 8–50% for non-neonatal tetanus [15]. The challenge is to keep the patient stable and free of complications during the recovery phase which requires growth of new axonal nerve terminals.

Conclusion

Tetanus can still occur among non-immunized patients in countries where immunization is available. Patients without a history of receiving a full series of tetanus vaccination, and who experience a skin penetrating injury with potential introduction of C. tetani spores are at risk and should be considered for vaccination and treatment. A typical clinical picture of tetanus could be tetanus, even in Norway.

Conflict of interest

None declared.

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