A drug addict suffering from tetanus is described in order to alert physicians into recognizing this life-threatening complication of parenteral drug abuse. This young man had used all accessible veins and started injecting heroin subcutaneously. This resulted in numerous cutaneous ulcers and abscesses—the probable site of Clostridium tetani infection. Prompt recognition and appropriate management resulted in complete recovery after a prolonged stay in the intensive care unit.

Tetanus has been a well recognized entity since antiquity. Although most cases are reported from developing countries, industrialized nations still encounter this disease. Tetanus is preventable by immunization and can be treated by modern day supportive care. However, it still carries a significant mortality. A case of generalized tetanus presented to our hospital as a complication of illicit subcutaneous drug injections. Prompt recognition and appropriate management resulted in complete recovery. We describe this case to alert physicians, as this complication of parenteral drug abuse may be confused with signs of drug withdrawal or drug reaction.

Case Report

A 31-year-old male, was admitted to a drug rehabilitation centre on the night of 14 October 1991. He had been an intravenous heroin addict for the previous 6 years and had recently begun injecting it subcutaneously having used all accessible veins. He had noticed pain in his jaw and back for one day. Following admission, he started to develop increasing generalized stiffness, difficulty in swallowing and profuse sweating. He was at that time transferred to King Fahad National Guard Hospital (KFNGH) for further management.

On arrival at our hospital his blood pressure (BP) was 150/80 mmHg, pulse 110/min, respiratory rate 40/min, and temperature 36°C. He was conscious, alert and oriented. His pupils were 4 mm wide and reactive to light. Cardiac and respiratory systems were unremarkable, except for tachycardia and tachypnea. The abdomen was rigid and extremities were undergoing repeated spasms. There were several subcutaneous ulcers and abscesses. He had typical manifestations of tetanus which included trismus, ophthohotonus and profuse sweating. His chemistry showed sodium, 149 mmol/litre; potassium, 5.0 mmol/litre; chloride, 111 mmol/litre; bicarbonate, 20 mmol/litre; BUN, 44 mmol/litre; creatinine, 86 μmol/litre; glucose, 6.5 mmol/litre; total bilirubin, 6 μmol/litre; alkaline phosphatase, 187 U/litre; albumin, 21 g/litre; and calcium 2.13 mmol/litre. CBC revealed WBC 14.9 x 10⁹/litre with a normal differential. Hgb was 125 g/litre and the platelet count 493 000/mm³. Hepatitis B surface antigen and HIV serology were negative. Urine was positive for barbiturates and opiates. On room air, arterial blood gases showed pH 7.45; PO₂ 33 mm Hg; and P0₂ 72 mm Hg. He was admitted to the intensive care unit for further management. He received 50 mg of diphenhydramine for possible acute dystonic drug reaction, with no relief. His treatment included...
intravenous penicillin, intramuscular human tetanus immunoglobulin, tetanus toxoid, and local wound debridement. For his repetitive and severe spasms, which were not fully controlled by diazepam, he was paralysed with pancuronium and placed on mechanical ventilation for a total of 36 days. He had a tracheostomy done in the third week of his stay. His course was complicated by central line-related infection and pneumonia, which were treated with appropriate antibiotics.

He manifested labile hypertension, tachycardia, mild fever and profuse sweating. This hyperactivity of the sympathetic nervous system was controlled with labetolol and morphine. He also developed transient episodes of bradycardia which reverted to sinus rhythm without any intervention. Paralysis and sedation were withheld intermittently throughout the stay on the ventilator for neurological assessment. Physiotherapy was started and continued when he was transferred to the ward. The patient was discharged on the 61st hospital day and sent back to the drug rehabilitation centre.

Discussion

Since tetanus is a clinical diagnosis, physicians dealing with such patients should be well versed in the characteristic clinical picture. The earlier stages of tetanus are more difficult to diagnose than the full blown picture. Drug withdrawal syndromes, strychnine poisoning, acute dystonic drug reactions, and hypocalcaemic tetany may mimic generalized tetanus. The presence of a clear sensoritis is important in differentiating it from meningitis and drug withdrawal syndromes.

Tetanus as a complication of parenteral drug abuse has been well recognized. Parenteral drug abusers accounted for about 2% of tetanus cases in the USA during the period 1975–1984. In the Kingdom of Saudi Arabia, this condition has not been previously described in the drug addict population. Non-immunized drug addicts develop tetanus when they have used all accessible veins and start injecting drugs intravenous or subcutaneously. This practice is commonly known as ‘skin popping’ among drug addicts.

The intra- or subcutaneous injection of drugs with contaminated needles provides a nidus of anaerobic environment which is conducive to the growth of Clostridium tetani.

The management of tetanus is optimally carried out in an intensive care unit. The principles of treatment include supportive care, control of spasms, neutralization of toxin, eradication of the organism and active immunization. Adequate control of spasms should be obtained with the use of diazepam. If the respiration becomes compromised secondary to excessive requirement of diazepam, mechanical ventilation should be instituted. The airway should be secured as early as possible in generalized tetanus, and early tracheostomy considered in view of the anticipated protracted recovery.

The hypersympathetic state is controlled by combined alpha- and beta-blockade. Morphine has also been used for this state. Those patients who develop parasympathetic overactivity resulting in bradycardia, should be considered for temporary cardiac pacing. Adequate attention should be given to fluid balance, nutritional support, prevention and treatment of infections, decubitus ulcers, pulmonary embolism and contractures. The circulating toxin should be neutralized as soon as possible with intramuscular administration of human anti-tetanus immunoglobulin. Intrathecal human anti-tetanus immunoglobulin has been used in severe cases but its use is not recommended routinely. Local debridement and drainage of cutaneous ulcers and abscesses needs to be done diligently to eliminate the source of exotoxin production by the tetanus organisms. Penicillin also needs to be given for this reason. Active immunization should be started and completed as immunity does not develop from an attack of tetanus.

The mortality rate of tetanus has decreased from 40% to 10–20% with appropriate treatment carried out in intensive care units. The quality of intensive care is the most important factor affecting the prognosis.

References