Tetanus

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This potentially fatal condition, also called ‘lockjaw’, is caused by Clostridium tetani , a Gram-positive spore-forming bacillus occurring naturally in the intestines of humans and in the soil. It enters the body through a wound and replicates, thriving on the anaerobic conditions present in devitalised tissues. It produces tetanospasmin, an exotoxin that binds to Hans Christian Joachim Gram , 1853–1938, Professor of Pharmacology (1891–1900) and of Medicine (1900–1923), Copenhagen, Denmark, described this method of staining bacteria in 1884. the neuromuscular junctions of the central nervous system neurones, rendering them incapable of neurotransmitter release. This leads to failure of inhibition of motor reflex tion and generalised contractions responses to sensory stim ula of agonist and antagonist muscles produce tetanic spasms. The median incubation period is 7 days, ranging from 4 to 14 days. Early symptoms are painful spasms of the facial muscles, resulting in risus sardonicus (Figure 33.15). The spasms spread to involve the respiratory and laryngeal m usculature. Spasms of the paravertebral and extensor limb musculature produce opisthotonus, an arching of the whole body . Laryngeal muscle spasm leads to apnoea and, if prolonged, to asphyxia and respi - ratory arrest. The spasms can be brought on by the slightest of sensory stimulus. The diagnosis is obvious once it is fully manifest. There are three aspects of management: /uni25CF Prevention . Wounds contaminated with soil can har - bour tetanus spores, and active immunisation is indicated by administering 0.5 /uni00A0 mL of tetanus toxoid intramuscularly . Patients with gross contamination of cavitating w ounds should also receive 250–500 /uni00A0 U of human anti-tetanus globulin (ATG) intramuscularly to provide passive immuni - sation and to neutralise the circulating toxin. In full-blown

(d)

clinical tetanus, 3000–10 /uni00A0 000 /uni00A0 U of ATG should be admin - istered. Wound manipulation should be avoided for 2–3 hours after ATG administration to minimise tetanospas - min release. /uni25CF Local wound care . This includes a thorough wound debridement to eliminate the anaerobic environment. 6 Intravenous administration of 10–24 /uni00A0 ×/uni00A0 10 U per day /uni00A0 of penicillin G should be continued for 10–14 days. The wound should be closed using the delayed primary or sec - ondary closure techniques. /uni25CF Supportive care for established disease . These patients are nursed in an intensive care unit (ICU) environ - ment, free from strong sensory stimuli. Diazepam is use - ful in preventing the onset of spasms but, if these become sustained, the patient is paralysed, intubated and placed on a ventilator. The patient is then gradually weaned o ff the ventilator under cover of anticonvulsants. The o verall mortality rate is around 45%, prognosis being determined

(c) Figure 33.14 (a–d) Late-presenting facial injury with gross contami nation. A thorough debridement followed by delayed primary closure has yielded good results. Figure 33.15 Risus sardonicus of ‘lockjaw’ (courtesy of Dr Samira Ajmal, FRCS). (d)

tom to the first tetanic spasm. In general, shorter intervals indicate a poorer prognosis. Recently , intrathecal antitoxin administration has been used for spasm control to avoid ventilatory support. Nev ertheless, without access to me chanical ventilation the mortality remains high and even those who survive may require several weeks of hospital isation. Summary box 33.7 Tetanus /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF

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