PP133 TRISMUS LED BY PALM OIL THORN PRICK

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INTRODUCTION

Tetanus are caused by tetanus toxin(tetanospasmin), produced by Clostridium tetani, whose spores survive in soil and cause infection by contaminating wounds. Often manifesting as trismus/lockjaw, opistotonus, or rigidity and spasms of respiratory, laryngeal, and abdominal muscles. Incubation period ranges from 3 to 21 days, averaging about 10 days. The following is a case of patient with history of pricked by palm oil thorn who was diagnosed with tetanus.

CASE REPORT

61 years old Chinese gentleman presented with difficulty swallowing solids, unable to open mouth, hypersalivation and slurred speech for two days. Works as rubber tapper, had history of herbicide usage (unsure content) without PPE. On arrival patient afebrile, no gastrointestinal symptoms, vital signs stable, limited mouth opening, no excessive sweating, no limb weakness but there was a puncture wound lateral aspect of left knee, which patient claims had been pricked by palm oil thorn two weeks ago. Blood parameters were normal and was referred to dental and ORL team but no abnormalities detected. Clinical diagnosis of tetanus was made in view persistent trismus, history of pricked by palm oil thorn, and unsure history of last tetanus immunization. Patient was treated with intravenous Diazepam, tetanus immunoglobulin 3000 unit, antitetanus toxoid, intravenous Metronidazole, Magnesium Sulphate and Baclofen. Day 2 of admission, patient developed generalised tetanus with persistent jaw stiffness, body pain and spasm every 15 minutes and was referred to anesthetic team. Patient was intubated in operation theatre anticipating difficult airway since patient had limited mouth opening and receding chin. Patient was then ventilated in dark room in ICU sedated with Midamorphine infusion. Day 11 post intubation percutaneous tracheostomy was performed. Patient was spasm free from day 17 of admission, however delayed weaning due to patient developed nosocomial infection and was discharged well after 30 days.

DISCUSSION AND CONCLUSION

Tetanus is a clinical syndrome without confirmatory laboratory tests. Differential diagnosis was confusing in view patient had herbicide exposure and only trismus during presentation which later developed into generalised tetanus. Early diagnosis and treatment are essential to avoid fatal outcome. Treatment includes intravenous metronidazole, 500 mg three times daily. Intramuscular Tetanus antitoxin; doses of 500 IU, 3000 IU, to inactivate any free tetanus toxin, benzodiazepines, which augment the effect of GABA on the GABAA receptors of lower motor neurons and Baclofen, which acts on GABAB receptors. Magnesium, a calcium antagonist that acts both by reducing acetylcholine release and by reducing the muscle response to acetylcholine [8,9,10]. Tetanus patients should be in a calm environment to avoid the triggering of spasms by noise or other sensory stimulation.