

Introduction:

Tetanus is still a significant health hazard in developing countries, with high associated mortality. It is caused by *Clostridium Tetani*. This anaerobic bacillus produces Tetanospasmin which affects the central nervous system. It causes persistent tonic spasms. Spasms may be spontaneous or triggered by visual, auditory or emotional stimuli and can be severe enough to cause fractures or avulse tendons. Prolonged muscular action causes sudden, powerful, and painful contractions of muscle groups, which is called "tetany". The incubation period is the time from spore inoculation to the initial symptom of tetanus. The onset period - the time from the first symptom to the first muscular spasm - reflects the progression of neurological manifestations caused by the tetanus toxin. Tetanospasmin disables release of neurotransmitter from presynaptic vesicles (particularly the inhibitory neurotransmitters GABA and glycine). The toxin is transported in the neurons into the central nervous system, which takes from 10-14 days. Recovery period is about 4-6 weeks. The progression of clinical symptoms of tetanus, such as respiratory failure and autonomic instability, are associated with high morbidity and mortality in the early hospitalization period. Later in the course of the disease, death results from complications of autonomic instability or prolonged ICU exposure.

Case Study :

A 50 year old male k/c/o diabetes type 2 for 3 years on OHA presented in hospital with h/o road traffic accident on 14/6/17. He had lacerated injury to his knee. On examination, he had diaphoresis, slow tongue movements and was unable to open his mouth fully. CT brain, MRI cervical spine and wound pus culture including clostridium tetani (aerobic and anaerobic) were sent to rule out tetanus. While inserting Ryle's tube patient had cardiac arrest (which was most likely due to autonomic dysfunction). During the initial period of treatment patient was put on mechanical ventilation with volume control mode. Patient was handled with standard procedure for tetanus including debridement and care of lesion. Tetanus toxoid immunoglobulin 3000 units, inj. Metronidazole 500mg 6th hrly, inj Medazolam 5mg/kg as continuous infusion was started. Patient was shifted to isolation room with no lights to avoid any stimulation. Spasms were partially controlled by midazolam but additional muscle relaxant inj. Vecuronium 4mg/hr was added to the regimen for 2 days. Infusion of inj Magnesium Sulfate was started as an effective adjunct in relaxation, sedation and controlling the autonomic disturbance in tetanus. Tab Baclofen 10mg BD was started for better control of spastic movement. Further for better control of spastic movement, more 5000 units of tetanus toxoid immunoglobulin was given (3000 units IM & 2000 units S/C). Patient required 4 weeks of ventilator support, having tracheostomy done on 12th day. Intermittent

stopping of sedations allowed us to reaccess the sensorium. Slowly the sedations were tapered off as events of spasm were stopped and weaning process was initiated on day 25th. Gradually patient was put on T-Piece and weaned off from ventilator. Patient was shifted to wards on 30th day.

Specific treatment :

Three principles used while managing this case. 1) Prevent further toxin release. 2) Neutralise toxin present in the body outside the CNS. 3) Minimise the effects of the toxin already in the CNS.

Inj Metronidazole 500 mg iv 6 hourly was the drug of choice. Administration of tetanus toxoid immunoglobulin 150 mg/kg IM within 24 hours of diagnosis of tetanus. Further intermittent doses of TIG was given IM/SC to further neutralize the effect of toxin. Control rigidity and spasms with sedation, respiratory support given where necessary and control autonomic dysfunction achieved.

Discussion:

Benzodiazepines:

Benzodiazepines (GABA agonist) are the standard therapy for controlling muscle spasms in tetanus and have gained popularity over other agents due to their combined muscle relaxant, anticonvulsant, sedative and anxiolytic effects, which can be quite useful in managing a patient with tetanus. Midazolam, a relatively short acting benzodiazepine can be used as the continuous infusion @ 3mg/hr.

Muscle relaxant:

It should be added as the sedation alone is inadequate. Inj Vacuronium 0.1mg/kg IV should be used. Tab baclofen 10mg/kg BD can be added to control spasticity. Pancuronium may worsen autonomic instability by inhibiting catecholamine reuptake. Prolonged usage of muscle relaxants has been associated with critical illness neuropathy and myopathy.

Treatment of autonomic dysfunction:

Inj Magnesium Sulfate @ 2mg/kg should be started. It is a pre-synaptic neuromuscular blocker, reduces catecholamine release from nerves and the adrenal medulla, and reduces receptor responsiveness to released catecholamines. A loading dose of 5g should be given

over 20 minutes, followed by an intravenous infusion of 2g/hr. By antagonizing calcium metabolism, magnesium causes weakness and paralysis in overdose. Monitoring of serum magnesium levels is important to prevent this: the normal serum magnesium level is 0.7 - 1.0 mmol/l, whilst an acceptable therapeutic level is 2 - 3.5 mmol/l.

General management:

Enteral feeding should be started by nasogastric tube to prevent malnutrition and autonomic gastrointestinal dysfunction. Prevention of ventilator associated pneumonia due to prolonged ventilator support which was managed with appropriate antibiotic treatment. Care of wound should be taken to minimize bacterial growth. Measures to minimize the risks of thromboembolism, gastrointestinal haemorrhage and pressure sores should be applied.

Conclusion:

Tetanus is a dreaded disease with high mortality. But with a protocolized approach it can be treated effectively, which decreases the mortality and morbidity associated with it.

References:

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