Maternal and neonatal tetanus are the important causes of maternal and neonatal mortality claiming about 180,000 lives annually globally. Tetanus is caused by neurotoxin produced by Clostridium tetani, a gram positive, obligate anaerobic, rod-shaped, spore forming bacterium. Tetanus is characterized by muscle rigidity and painful muscle spasms caused by tetanus toxin blockade in neurons. Diagnosis of tetanus is done by analysis of clinical records. Maternal and Neonatal Tetanus causes are predominant in poor, remote and disenfranchised communities where unhygienic obstetric and postnatal practices prevail, and access to maternal tetanus toxoid immunization is poor. The only reliable immunity against Maternal and Neonatal Tetanus is that induced by vaccination with tetanus toxoid. Prevention relies on avoidance of unsafe delivery, unsafe abortions, and umbilical cord care practices which can predispose to tetanus infection; and promotion of maternal tetanus immunization.

**Keywords:** Clostridium Neonatal Tetanus Maternal Mortality

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**INTRODUCTION**

Maternal and Neonatal Tetanus are important causes of maternal and neonatal mortality, claiming about 180,000 lives worldwide every year, almost exclusively in developing countries. Tetanus in the first 28 days of life (neonatal tetanus) was long recognized by clinicians in resource-poor setting as an important cause of neonatal death. However, since babies affected by this disease usually are born at home and die there without registration of either event, the true burden was unknown. In the 1970s and 1980s community based surveys about neonatal tetanus from more than 40 countries showed that fewer than 10% of tetanus-related cases and death routinely reported in most countries: in some regions, the reporting fraction was as low as 2-5%. This disease accounts for 5-7% of worldwide neonatal mortality, compared with 14% in 1993. Estimates suggest that these deaths have been reduced, but that still some 130,000 babies died around the year 2004 from this very preventable disease (WHO, 2004). In developed countries, tetanus is now little more than a medical curiosity; maternal and neonatal tetanus are exceedingly rare (Rosenhlatt et al., 2005).
CAUSATIVE AGENT OF MATERNAL AND NEONATAL TETANUS (MNT).

Tetanus is caused by a neurotoxin produced by Clostridium tetani, a grampositive, obligate anaerobic rod-shaped, spore forming bacterium as shown by Feingold (1998) in Figure 1. Tetani spores worldwide are constituent of soil and in the gastrointestinal tracts of animals (including humans), and can contaminate many surfaces and substances. The spores are extremely hardy; destruction requires autoclaving or prolonged exposure to iodine, hydrogen peroxide, formalin or gluteraldehyde (Feingold, 1998). Clostridium tetani stains gram positive in fresh cultures; established cultures may stain gram negative. During vegetative growth, the organism cannot survive in the presence of oxygen, is heat-sensitive and exhibits flagella mortality. Their endospores, a dormant form, are indifferent to oxygen, and can survive for long periods by withstanding measures of heat, desiccation, chemicals, and irradiation that would kill all vegetative bacteria. When the appropriate conditions are renewed, these endospores germinate, and the resulting vegetative bacteria may once again multiply (Feingold, 1998).

TOXINS

Toxins are poisonous substances produced by living cells or organisms although humans are technically living organisms, man-made substances created by artificial processes usually are not considered toxins by this definition.

Classes of Toxins

Endotoxins: Endotoxins are toxins associated with certain bacteria. An endotoxin is a toxin that is a structural molecule of the bacteria that is recognized by the immune system.

Exotoxins: Exotoxins are toxins excreted by a microorganism including bacteria, fungi, algae, and protozoa. They are highly potent and can cause major damage to the hosts.

<table>
<thead>
<tr>
<th>Exotoxins</th>
<th>Organisms</th>
<th>Toxin produced</th>
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<tr>
<td>1 A-B toxins:</td>
<td></td>
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<tr>
<td>Neurotoxins</td>
<td>Clostridium tetani</td>
<td>Tetanospasmin</td>
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<tr>
<td>Enterotoxins</td>
<td>Vibrio cholera</td>
<td>Cholera toxin</td>
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<td>Cytotoxins</td>
<td>Corynebacterium diphtheria</td>
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<td>Membrane damaging toxins</td>
<td>Clostridium perfringens</td>
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<td>3 Superantigens</td>
<td>Staphylococcus aureus</td>
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<tr>
<td>4 Other toxic protein</td>
<td>Microorganisms</td>
<td>Proteases, lipases and other hydrolases.</td>
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</tbody>
</table>
Tetanus Toxins (Tetanospasmin)

The endotoxin responsible for tetanus is one of the worst potent toxins ever identified with a minimum lethal dose of less than 2.5 ng/kg in humans. This high potency is caused by the toxin’s absolute neuro-specificity and enzymatic action. Tetanospasmin is synthesized as an inactive polypeptide chain during the bacterial growth phase. The genes for the neurotoxin and its transitional regulator, T ox R which is needed for toxin production, are located in an intracellular plasmid. At autolysis, after death of the bacterium, the toxin molecule is released and transformed by bacterial or tissue protease into its active form a 1000 KDa heavy chain and a 50 KDa light chain. The heavy chain is necessary for binding to and entry into the neuron. The light chain is responsible for the toxic properties (Lalli et al., 2003)

MECHANISM OF ACTION OF TETANUS TOXIN

The complex mechanism for binding of tetanus toxin to peripheral neurons and its absorption into these cells, transport to the Central Nervous System, and toxic activity is shown in Figure 2. After its release, tetanus toxin diffuses to adjacent muscle tissue, where it binds to specific glycoprotein in lipid-raft constituents needed for the effective binding of tetanus toxin are not fully understood. Free tetanus toxin also enters the lymphatic system and the bloodstream, disseminating widely before entering motor neurons at disparate sites. Inside motor neurons, tetanus toxin is transported via acetylcholine to the Central Nervous System at 3-13 mm/h by a specific retrograde axonal transport system. At the spinal cord and brain stem, the toxin diffuses across the synaptic spaces to enter glycineric and gabinergic inhibitory interneurons (Schiavo et al., 2000).

Inside inhibitory interneuron’s, the disulphide bond converting the heavy and light chains of the toxin is broken. The free light chain is a zinc-endopeptidase that cleaves synaptobrevin proteins in synaptic vesicle membranes. The action of inhibitory neurons is thereby impeded, leaving motor neuron excitation unopposed, and resulting in the muscle rigidity and long-lasting painful spasms which are characteristics of tetanus. In addition to its action on the motor system, tetanus toxin can have profound and life threatening effects on the autonomic nervous system by interrupting spinal inhibitory sympathetic reflexes, resulting in a hyperadrenergic state. In action of tetanus toxin within the neurons persist for several weeks; the mechanism of functional recovery remains unclear (Feingold 1998; Schiavo et al., 2000).

SYMPTOMS

Tetanus is characterized by muscle rigidity and painful muscle spasms, caused by tetanus toxins.
blockade of inhibitory neuron that normally oppose and modulate the action of excitatory motor neurons. Maternal and Neonatal Tetanus are both forms of generalized tetanus (the most common manifestation of the disease), and have similar courses. Tetanus muscle rigidity usually begins in the masseter muscle, resulting in trismus (lockjaw). Dysphagia and neck, shoulders, back, or abdominal muscle stiffness and pain are other early symptoms (Patel et al., 1999). In neonatal tetanus, trismus and up muscle rigidity interfere with normal sucking and feeding, which is the hallmark of disease on set. As disease severity increases, muscle rigidity extends throughout the body and muscle spasms begin, first in response to sensory stimuli but later progressing to spontaneous long-lasting excruciating spasms of many muscle groups (Figure 3). The onset period, or time from first symptom to first spasms, is typically 1-3 days, ranging from hours to 5 days (Patel et al., 1999). The average incubation period for Maternal Neonatal Tetanus cage (age at first symptom) is shorter than that on non neonatal tetanus. About 90% of neonates with tetanus develop symptoms in the first 3-14 days of life, mostly on day 6-8, distinguishing neonatal tetanus from other causes of neonatal mortality which typically occur in the first two days of life (Farrar et al., 2000)

**DIAGNOSIS**

The diagnosis of Maternal and Neonatal Tetanus is made strictly on clinical grounds. Cultures of tetanus patients wounds frequently fail to detect growth of *C. tetani*; moreover, the organism occasionally grows in cultures from patients without tetanus. The neonate tetanus form can be diagnosed, if the patient shows signs of uncontrollable irritation, and the inability to take in fluids. As this type of tetanus infection is found only in infants, they are found to have a poor sucking ability. Diagnosis is a step by step process. The doctor has two options, either to go for the laboratory test to diagnose the patient or to option for analysis of clinical records. Though laboratory testing is done in some special cases, most of the diagnosis for the tetanus infection is done based on recent and previous clinical records. Here diagnosis involves four steps:

- Confirmation of an infection
- Checking symptoms of various infections
- Diagnosing the infection
- Stages and type of diagnosed infection

Once the doctor diagnoses the patient case as a tetanus infection he has to classify the type of tetanus which has affected the patient. This is important as proper treatment is possible only if the correct type of infection and all symptoms are perfectly known (Wassilak et al., 2004). Laboratory testing for the tetanus infection is used in determining the presence of the toxins in the blood sample, which will help in diagnosing the presence of tetanus bacteria (Akina et al, 2004).

**EPIDEMIOLOGY**

Maternal and Neonatal Tetanus causes are clustered in poor, remote and disenfranchised
communities where unhygienic obstetric and postnatal practices prevail, and access to maternal tetanus toxoid immunization is poor. Home delivery assisted by untrained birth attendants are the main reasons behind the widespread of Maternal Neonatal Tetanus in many developing countries, especially in rural areas, and bring together many factors that confers, a high risk of tetanus to both mother and child. The following are the reasons behind the widespread of Maternal and Neonatal Tetanus:

- Home delivery
- Untrained birth attendants
- Poverty
- Lack of maternal and paternal education
- Young maternal age
- Cultural restriction in women’s access to health services
- Low antenatal care attendance
- Inadequate vaccination with tetanus toxoid
- Unsafe abortion (Omoigberale and Abiodun, 2005; Ogunlesi, 2007).

**TREATMENT**

The specific objectives of tetanus treatment are to stop the production of toxin at the site of infection with appropriate wound care and antibiotic use; to neutralize circulating toxin with antitetanus immunoglobulin; and to provide effective management of muscle spasm, respiratory failure, autonomic dysfunction, and complications that arise during the cause of illness. Therapeutic approaches depend on the resources available in the facility to which the patients present. The only reliable immunity against tetanus is that induced by vaccination with tetanus toxoid. Newborn babies and young infants born to mother with antitetanus antibodies are protected against tetanus by acquired maternal antibody.

**PREVENTION AND CONTROL**

Maternal and Neonatal Tetanus prevention relies on avoidance of unsafe delivery, abortion, and umbilical cord care practices, and promotion of maternal tetanus immunization. The use of topical antimicrobials to replace traditional substances applied for cord care could have an important effect on neonatal tetanus in communities where high risk cord care practices persists, for example in rural Pakistan (Darmstadt et al., 2005). Control involves the Maternal and Neonatal Tetanus elimination initiative. Tetanus toxoid vaccination of pregnant women to prevent neonatal tetanus was included in WHO’s EPI a few year after its inception in 1974 and only 27% of pregnant women received at least two doses of tetanus toxoid in the 1980s.

Sustaining elimination of Maternal and Neonatal Tetanus will be a challenge, especially in places where the high risk approach is needed. Routine immunization with tetanus toxoid has been stagnant over the past decade with only 50-54% of pregnant women worldwide receiving adequate immunization, a situation largely unchanged since the late 1980s. Many countries still striving to achieve elimination have approved tetanus toxoid coverage in most districts and are close to meeting the objective (Figure 4) (WHO, 2005). Even before tetanus vaccine was available neonatal tetanus became increasingly rare in most of Europe and North America through hygienic childbirth practices and cord care. (Rossenhlatt et al, 2005). WHO recommends that at least five doses of tetanus toxoid vaccine be
given over 12-15 years, starting in infancy; a sixth dose given in early adulthood is encouraged, to ensure long lasting protection (WHO, 2005).

**CONCLUSION**

Maternal and Neonatal Tetanus still contribute considerably to neonatal mortality in world, even in Nigeria. This work has been examined some of the possible causes of the persistently high incidence of the disease over the years, and has suggested that attempts to eliminate the disease be carried in a wider context of meeting millennium development goal 4. This approach is suggested on the ground that reduction is neonatal mortality, and in countries with high Maternal Neonatal Tetanus mortality, its reduction equally becomes an imperative. The focus here is on improving health care delivery at the grass root levels to reduce or if possible eliminate the scourge of *Clostridium tetani* in the environment.

However, Nigeria as a case study is a large country with 774 LGA. Therefore, it would be wise to start implementation of these suggestions in carefully selected LGA as pilot centers, and gradually expand to other communities over time.

**REFERENCES**


