unicef
Gemeinsam für Kinder

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Sehr geehrte Frau Goldfinger,

vielen Dank für Ihr Schreiben vom 2.1.2010, das uns von Frau Kalweit weitergeleitet wurde. Gerne erläutern wir Ihnen nochmals die Position von UNICEF zum Thema Impfen. Als Kinderhilfswerk der Vereinten Nationen ist es unsere Aufgabe, die Lebensbedingungen für Kinder in Entwicklungsländern langfristig und nachhaltig zu verbessern. Bei der Ermittlung von Krankheiten, Präventionsmöglichkeiten und Behandlungstherapien richtet sich UNICEF nach den Erkenntnissen und Vorgaben der Weltgesundheitsorganisation WHO.

Die Existenz von bakteriellen Erregern (Mikroben) und viralen Erregern (Viren) ist wissenschaftlich belegt. Jeder kann sich in der einschlägigen Fachliteratur darüber informieren. Für uns steht außer Frage, dass diese Erreger Krankheiten verursachen. Tetanus (Wundstarrkrampf) wird durch das Bakterium Clostridium tetani ausgelöst. Durch Schutzimpfungen sind Tetanus und andere Krankheiten wirksam zu bekämpfen.

In Deutschland und anderen Industrieländern ist Impfen heute ein Mittel unter anderen, um Krankheiten vorzubeugen. Daher besteht in Deutschland die Impffreiheit. In Entwicklungsländern hingegen sind umfassende Schutzimpfungen nach wie vor unverzichtbar. Denn nicht nur die mangelhaften hygienischen Verhältnisse, sondern auch der insgesamt schlechtere Gesundheitszustand der Kinder führen dazu, dass Infektionen schneller entstehen und sich verheerend auswirken.

Neuartige Impfseren sowie neue Erfahrungen und Erkenntnisse mit Impfungen haben das Schadensrisiko beim Impfen erheblich verringert. In der Regel sprechen nur sehr schlimme akute Erkrankungen eines Kindes gegen eine Impfung. In jedem Einzelfall wird jedoch geprüft, ob ein Kind geimpft werden kann oder nicht. Gegner von Impfkampagnen kritisieren oft, dass in einigen Impfseren Thiomersal als Konservierungsstoff verwendet wird. Thiomersal enthält Quecksilberethyl - kein Quecksilbermethyl. Im Gegensatz zu Quecksilbermethyl wird Quecksilberethyl nicht im Körper eingelagert, sondern ganz normal durch den Darm ausgeschieden. Auch ist die Halbwertszeit von Quecksilberethyl mit nur einer Woche sehr gering. Ein von der WHO eingesetztes Beratungskomitee für Impfsicherheit hat daher den Einsatz von Thiomersal als Konversierungsstoff genehmigt.

Anbei senden wir Ihnen zwei Artikel zum Thema Tetanus. Für weitere Fragen, Berichte und Quellennachweise können Sie sich an die Weltgesundheitsorganisation WHO oder auch an das Robert-Koch-Institut wenden.

Die Adressen lauten:

- WHO-ECEH Bonn, Görresstraße 15, 53113 Bonn, Tel.: (0228) 2094-0, Fax: +(0228) 2094-201, info@ecehbonn.euro.who.int, http://www.euro.who.int/ecehbonn.
- Robert-Koch-Institut, Postfach 65 02 61, 13302 Berlin, Tel.: 030/18 754-0, Fax: 030/ 18 754-2328, www.rki.de

Mit freundlichen Grüßen

Wiebke Eckau
Grundsatz und Information

gus: CDC "Pink Book" - Centers for Disease Control and Prevention. Epidemiology and Prevention of Vaccine-Preventable Diseases. Atkinson W, Wolfe S, Hamborsky J, McIntyre, eds., Washington DC 2009

Chapter 27

Tetanus Toxoid

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Tetanus is unique among diseases for which immunization is routinely recommended because it is not communicable. *Clostridium tetani*, the causative agent of tetanus, is widespread in the environment; many animals in addition to humans can harbor and excrete the organism and its spores. When spores of *C. tetani* are introduced into the anaerobic/hypoaerobic conditions found in devitalized tissue or punctures, they germinate to vegetative bacilli that elaborate toxin. The clinical presentation results from the actions of this toxin on the central nervous system (CNS). Many animal species besides humans are susceptible to the disease.

The clinical characteristics of tetanus were recognized as distinct early in human history because of the constancy and severity of the symptoms in animals and humans. Although the first medical description appears in the writings of Hippocrates, the etiology of tetanus was unknown until 1884. Carle and Rattone[1] demonstrated that, when the contents of a pustule from a fatal human case were injected into sciatic nerve in a rabbit model, the typical symptoms of tetanus resulted; the disease could subsequently be passed to other rabbits from infected nervous tissue. Inoculation of soil samples into animals also resulted in tetanus. Gram-positive bacilli often were noted in the exudate at the inoculation site but generally not in nervous tissue, leading Nicolaier[2] to hypothesize that a poison produced at the site of inoculation led to the nervous system symptoms. In 1886, spore-forming bacilli were observed in the exudate obtained from a human case.[3] In 1889, the spores of *C. tetani*, in contrast to the vegetative organisms, were shown to survive heating and to germinate under anaerobic conditions; injection of pure cultures reproducibly caused the disease in animals.[4] After identification and purification of the toxin in 1890, repeated inoculation of animals with minute quantities of toxin led to the production in survivors of antibodies that neutralized the effects of the toxin.[5] Preparations of antibodies derived from animal sera, particularly from horses, became the first means to prevent and treat tetanus. The culmination of these efforts was in the preparation of "anatoxin"—chemically inactivated toxin, now termed a *toxoid*—in 1924.[6] Toxoid induced active immunity against the disease before exposure.

The impetus to prevent tetanus through immunization originated from the striking and highly fatal disease in both industrialized and developing nations, predominantly associated with injuries to otherwise healthy persons, and particularly during military conflicts. In the developing world, the continuing health burden from tetanus is largely among neonates. Prevention of tetanus is now almost universally achievable by use of highly immunogenic and safe toxoid-containing vaccines. Tetanus also can be prevented or modified by use of exogenous antibody.

Background

Clinical Description

Although the incubation period for tetanus has been reported to vary from 1 day to several months following a wound, the majority of cases occur within 3 days to 3 weeks after inoculation of spores. In the United States during 1998 to 2000, the median interval between the injury and onset of tetanus was 7 days (range 0 to 112 days) for 89 non-neonatal cases with reported information. The time between injury and the onset of symptoms was 30 days or less for 94% of the cases, and 2 days or less for 12% of the cases.[7]

There is a direct relationship between the site of inoculation and the incubation period, with the longest intervals occurring after injuries farthest from the CNS; injuries of the head and trunk generally are associated with the shortest incubation periods.[8,9] As historically noted, the incubation period is inversely related to severity of illness,[10–16] and the incubation period has been considered one of the best prognostic indicators.[17,18] Incubation periods of 10 days or more tend to result in mild disease, whereas incubation periods within 7 days of injury tend to result in more severe disease.

Three clinical syndromes are associated with tetanus infection: (1) localized, (2) generalized, and (3) cephalic.[8] Localized tetanus, which is unusual in humans, consists of spasm of muscles in a confined area close to the site of the injury.[19,20] Painful contractions may persist for several weeks to months before gradually subsiding. Localized disease is thought to occur when transport of toxin produced at the

Vaccines Textbook 2004

Tetanus

Tetanus is an acute, often fatal, disease caused by an exotoxin produced by the bacterium *Clostridium tetani*. It is characterized by generalized rigidity and convulsive spasms of skeletal muscles. The muscle stiffness usually involves the jaw (lockjaw) and neck and then becomes generalized.

Although records from antiquity (5th century BCE) contain clinical descriptions of tetanus, it was Carle and Rattone in 1884 who first produced tetanus in animals by injecting them with pus from a fatal human tetanus case. During the same year, Nicolaier produced tetanus in animals by injecting them with samples of soil. In 1889, Kitasato isolated the organism from a human victim, showed that it produced disease when injected into animals, and reported that the toxin could be neutralized by specific antibodies. In 1897, Nocard demonstrated the protective effect of passively transferred antitoxin, and passive immunization in humans was used for treatment and prophylaxis during World War I. Tetanus toxoid was developed by Descombey in 1924. It was first widely used during World War II.

Tetanus

- First described by Hippocrates
- Etiology discovered in 1884 by Carle and Rattone
- Passive immunization used for treatment and prophylaxis during World War I
- Tetanus toxoid first widely used during World War II

Clostridium tetani

C. tetani is a slender, gram-positive, anaerobic rod that may develop a terminal spore, giving it a drumstick appearance. The organism is sensitive to heat and cannot survive in the presence of oxygen. The spores, in contrast, are very resistant to heat and the usual antiseptics. They can survive autoclaving at 249.8°F (121°C) for 10–15 minutes. The spores are also relatively resistant to phenol and other chemical agents.

The spores are widely distributed in soil and in the intestines and feces of horses, sheep, cattle, dogs, cats, rats, guinea pigs, and chickens. Manure-treated soil may contain large numbers of spores. In agricultural areas, a significant number of human adults may harbor the organism. The spores can also be found on skin surfaces and in contaminated heroin.

C. tetani produces two exotoxins, tetanolysin and tetanospasmin. The function of tetanolysin is not known with certainty. Tetanospasmin is a neurotoxin and causes the clinical manifestations of tetanus. On the basis of weight, tetanospasmin is one of the most potent toxins known. The estimated minimum human lethal dose is 2.5 nanograms per kilogram of body weight (a nanogram is one billionth of a gram), or 175 nanograms for a 70-kg (154lb) human.

Clostridium tetani

- Anaerobic gram-positive, spore-forming bacteria
- Spores found in soil, animal feces; may persist for months to years
- Multiple toxins produced with growth of bacteria
- Tetanospasmin estimated human lethal dose = 2.5 ng/kg