Human *Streptococcus suis* Infection

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Human *Streptococcus suis* infection is a zoonosis caused by *Streptococcus suis*. Populations engaging in pig slaughtering and processing are high-risk groups. The bacteria gain their access into the human body via skin wound to cause the infection. Its clinical manifestations include common bacterial infection symptoms such as fever, chills, headaches, and poor appetite. In severe cases, it can be complicated by toxic shock syndrome (TSS) and streptococcus meningitis syndrome (SMS).

11.1 Etiology

Streptococcus suis was firstly isolated by De Moor in 1963 from specimens collected during the outbreak of piglet septicemia in the Netherlands. At that time, it was categorized into group R α-hemolytic streptococcus. In the year 1968, based on capsular typing, Elliot nominated it as capsular type 2 *Streptococcus suis.* Based on the polysaccharide antigen typing of *Streptococcus suis*, it can be further divided into 35 serotypes (1–34 and 1/2) with serotype 2 being the most virulent, followed by serotype 1. Serotype 2 is the most common pathogen isolated from infected pigs or patients. According to classification by Lancefield, *Streptococcus suis* can be categorized into group R, group S, and group T, with group R (serotype 2) and group S (serotype 1) being the most common. *Streptococcus suis* isolated from human specimens is virtually serotype 2. Therefore, specific antiserum against group R can be used for precipitation test. In such a way, the strain of serotype 2 *Streptococcus suis* can be identified.

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11.1.1 Physical and Chemical Properties

Streptococcus suis is categorized into the family of *Streptococcaceae*, which is a Gram-positive, spherical-shaped, or oval-shaped bacteria. The bacteria have no spore but have capsules, which grow into small colorless semi-transparent colonies on blood plate with raised regular smooth edges and with a diameter of 0.5–1.0 mm. Based on the hemolytic classification, *Streptococcus suis* is α-hemolytic on sheep blood plate but β-hemolytic on horse blood plate. The appropriate conditions for its culture include blood culture bottle and blood plate as the medium at a temperature of 37 °C for 18–20 h. The newly isolated *Streptococcus suis* has a typical morphology, with its chain length being up to above 20 thallus. After the second generation, its morphology is no longer typical and may even be Gram-negative coccobacillus without the shape of a chain. *Streptococcus suis* has poor intolerance to its surrounding physical and chemical factors and is sensitive to common disinfectants.

11.1.2 Factors Determining Its Virulence

Currently, it is believed that the important factors determining the virulence of *Streptococcus suis* includes the following.

11.1.2.1 Capsular Polysaccharide

So far, it is the only proved and the most important factor that determines the virulence of *Streptococcus suis*.

11.1.2.2 Lysozyme Released Protein and Extracellular Protein Factor

In addition to capsular polysaccharides, the two proteins are the more commonly used indicators to assess the virulence of *Streptococcus suis*.

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11.1.2.3 Hemolysin of *Streptococcus suis*

Hemolysin is believed to be a major factor indicating the virulence of several bacteria. It may facilitate *Streptococcus suis* to invade and decompose cells.

11.1.2.4 Protein of 44,000, IgG Binding Protein, and Other Factors

Protein of 44,000 is a protein in the bacterial cell wall of serotype 2 *Streptococcus suis*. IgG binding protein is heat shock protein, which is reported to be associated with its virulence. In addition, pili and adhesion factors are also common factors determining the bacterial virulence.

11.1.3 MLST Analysis

Multilocus sequence typing (MLST) was performed for the strains isolated during outbreaks of human *Streptococcus suis* infection in provinces of Jiangsu and Sichuan in China. The pathogenic strains are proved to be MLST type 7.

11.2 Epidemiology

11.2.1 The Source of Infection

Sick and deceased pigs with *Streptococcus suis* infection are the main source of infection. It has been reported that serotype 2 *Streptococcus suis* can be isolated from deceased deer, sheep, chickens, ducks, horses, cats, dogs, and ruminants. But the evidence is not sufficient to define them as the source of infection.

There has been no evidence to prove that human *Streptococcus suis* infection can spread from person to person.

11.2.2 Route of Transmission

Human *Streptococcus suis* infection spreads via contact with pigs and unprocessed pork products infected by *Streptococcus suis*. The bacteria gain their access through skin wound or conjunctiva to cause the infection.

11.2.3 Population Susceptibility

People are generally susceptible. Populations with direct contact to sick or deceased pigs or pork products are high-risk populations. In them, individuals with skin wounds are especially vulnerable to be infected. Most foreigner scholars believe that human *Streptococcus suis* infection is an important occupational disease with sick animals as its source of infection. Infection of *Streptococcus suis* by people with immune deficiency is more serious.

11.2.4 Epidemic Features

The epidemiological features of the disease are still not fully understood. It is highly sporadic along with the outbreak of swine *Streptococcus* infection, which is more commonly found in summers. The spread of swine *Streptococcus* infection in pigs may be closely related to the hot and humid environment. Therefore, hotness and humidity are most likely to indirectly contribute to the increased occurrence of human *Streptococcus suis* infection in summers. This disease is not necessarily related to gender or age, but has a close relationship to occupation. Populations engaging or involving in pig breeding, slaughter, processing, delivery, sales, and cooking are high-risk groups, especially those who slaughter sick and deceased pigs.

In 1968, scholars from Denmark firstly reported three cases of human *Streptococcus suis* infection that leads to meningitis and is complicated by septicemia. In 1975, sporadic cases were reported in the Netherlands. Thereafter, the cases of human *Streptococcus suis* infection have been continually reported in Chinese Hong Kong, the United Kingdom, Canada, Germany, France, the United States, Australia, Belgium, Brazil, Spain, Japan, Thailand, Sweden, and mainland China. By the year 2000, more than 200 cases of human *Streptococcus suis* infection had been reported across the world. Countries or regions with more reported cases are generally characterized by developed industry of livestock breeding or by a common lifestyle of eating pork, with highly sporadic prevalence. In China, human *Streptococcus suis* infection was reported in Jiangsu province in 1998, with 25 cases of occurrence and 14 cases of death. From June to August 2005, an outbreak of human *Streptococcus suis* infection occurred in Sichuan province, which has been the most serious outbreak of human *Streptococcus suis* infection in the world, with 204 cases of occurrence and 38 cases of death.

11.3 Pathogenesis and Pathologic Changes

11.3.1 Pathogenesis

Human *Streptococcus suis* infection mainly spreads via skin wound after direct contact. The *Streptococcus suis* gains its access into the human body via wounds of skin or mucosa, which rapidly grows and reproduces in the blood after spreading into the blood flow to cause septicemia. The bacteria then gain their access into organs and tissues along with

blood flow to cause pathological changes in multiple organs and tissues. Meanwhile, the bacteria release toxins to cause severe toxic reactions of the body, with following occurrence of toxemia. Under the effects of bacterial toxins, severe patients experience vascular endothelial lesions and disseminated intravascular coagulation (DIC) that leads to systemic microcirculatory disorder and multiple organ failure.

11.3.2 Pathological Changes

The severe clinical manifestations of human *Streptococcus suis* infection can be divided into two types: SMS and TSS.

The main pathological changes of SMS include purulent meningitis, obvious meningeal vascular congestion, infiltration of a large quantity of neutrophils, and slight pathological changes in other organs.

TSS is characterized by septicemic shock complicated by DIC. The pathological changes include cellular degeneration and necrosis of multiple organs and tissue parenchyma, varying quantities of neutrophil infiltration, interstitial vascular congestion and leakage hemorrhage, and formation of capillary microthrombus (hyaline thrombus). The involved organs have pathological changes of (1) petechiae and ecchymosis in the skin, mucosa (of gastrointestinal tract, respiratory tract, and urogenital tract), and serosa; bleeding of heart, liver, kidney, adrenal gland, esophagus, and intestines with incoagulable bright red blood; and varying quantities of microthrombus (hyaline thrombus) in capillaries of some organs (like lung and kidney) which are PTAH-positive fibrin thrombus; (2) pulmonary congestion and edema, focal and flakes of hemorrhage, and formation of microthrombus in capillaries; (3) acute pneumonia; (4) slight hepatomegaly as well as spots and focal or flakes of necrosis of hepatocytes; (5) renal congestion and hemorrhage and varying quantities of microthrombus in glomerular capillaries; (6) myocardial degeneration, spots of myocardial necrosis and infiltration of inflammatory cells, and interstitial vascular congestion with multifocal hemorrhage; (7) serous cavity effusion such as pleural, pericardial, and abdominal cavities; more lesions in the lung, kidney, and heart but not obvious lesions in the brain and cerebral meninges; and (8), in some cases, a skin wound that is commonly found on the arms and feet.

Severe human *Streptococcus suis* infection can be complicated by DIC. The pathological changes include intracapillary microthrombus in multiple organs and tissues and leakage hemorrhage, incoagulable blood, and secondary multiple organ failure and shock.

Patients with SMS have highly dilated and congested meningeal vascular vessels, widened subarachnoid cavity, and leakage of neutrophils, fibrin, and fluids in large quantities. Therefore, the patients sustain intracranial hypertension due to increased quantity of cerebrospinal fluid. And the patients complain of headache, projectile vomiting, and positive pathological signs. Due to the involved cranial nerves, patients may experience different degrees of hearing impairment or even permanent deafness.

11.4 Clinical Symptoms and Signs

The incubation period ranges from several hours to 7 days, commonly 2–3 days. The duration of the incubation period is related to the virulence and quantity of pathogenic bacteria as well as the immunity of the infected human. Generally, the shorter period of incubation predicts the more serious conditions. According to the clinical manifestations, it can be divided into the following types:

11.4.1 Common Type

It commonly has an acute onset, with manifestation of fever, chills, headache, dizziness, general upset, and fatigue. Some patients may experience nausea, vomiting, abdominal pain, and diarrhea. No shock or coma occurs in the common type of the disease.

11.4.2 Shock Type

Based on the systemic infection, the shock type has manifestations of hypotension with adult systolic pressure lower than 90 mmHg and pulse pressure lower than 20 mmHg, with accompanying two or more conditions: (1) renal dysfunction, (2) coagulation disorder or disseminated intravascular coagulation, (3) hepatic dysfunction, (4) acute respiratory distress syndrome, (5) petechiae and ecchymoses in skin and mucosa of the whole body or conjunctival congestion, and (6) soft tissue necrosis, fasciitis, myositis, and gangrene.

11.4.3 Meningitis Type

The clinical manifestations include fever, chills, general upset, fatigue, headache, and vomiting. In severe cases, coma occurs, with positive meningeal irritation sign and purulent changes of the cerebrospinal fluid.

11.4.4 Mixed Type

The manifestations of both shock type and meningitis type can be found in such type of cases.

11.5 Human *Streptococcus suis* Infection-Related Complications

11.5.1 Meningitis

About 85 % of cases with human *Streptococcus suis* infection have typical manifestations of meningitis. *Streptococcus suis* meningitis is characterized by higher incidence of hearing loss (54–80 %) than other bacterial meningitis, which commonly occurs within 24 h after the onset (1–14 days). In some cases, the onset of *Streptococcus suis* meningitis begins with occurrence of deafness. Bilateral deafness is more common than unilateral deafness. In some cases, the deafness is characterized by subclinical high-profile hearing loss. Other dysfunctions related to the 8th pair of cranial nerves are also common, with 30–50 % of cases with meningitis showing dizziness and ataxia. The patients may also experience palsy of the 3rd pair of cranial nerves, with unilateral or bilateral facial paralysis.

11.5.2 Toxic Shock Syndrome (TSS)

TSS is commonly caused by group A streptococcus and rarely caused by group B and group C streptococcus. *Streptococcus suis* is a rare pathogen of TSS, but commonly causes outbreak of TSS, with high rate of mortality. TSS has a short period of incubation, with an average incubation period of 2–3 days ranging from several hours to 7 days. The manifestations commonly include sudden high fever (in 100 % of TSS cases) with the highest body temperature of 42 °C and accompanying headache (in 56.25 % of TSS cases), diarrhea and other gastrointestinal symptoms (in 68.75 % of TSS cases), and skin petechiae and ecchymoses (in 81.25 % of TSS cases) that are commonly distributed in the face, limbs, and head with no protrusion higher than the skin surface and no ulceration. The outcomes of TSS include shock (in 100 % of TSS cases), oliguria (in 81.25 % of TSS cases), and death (in 81.25 % of TSS cases). There may be also blood coagulation dysfunction, renal insufficiency, hepatic insufficiency, acute respiratory distress syndrome, soft tissue necrosis, and fasciitis. By autopsy, the findings commonly include bleeding in multiple parts and multiple organs with different degrees and ranges and visceral capillary diffuse coagulation. The autopsy findings are similar to those findings by autopsy of dead pigs infected by *Streptococcus suis*.

11.5.3 Other Related Complications

Streptococcus suis has been isolated from patients with endocarditis and septicemia who have no diagnosis of meningitis and toxic shock. The patients may suffer from fulminant ecchymosis and rhabdomyolysis. Vilaichone et al. reported two cases of serotype 2 *Streptococcus suis* infection complicated by peritonitis. Robertson et al. reported their findings after detecting titer of antibodies against *Streptococcus suis* in pig-feeding workers, with a positive rate of 21 %, which indicated that subclinical infection of *Streptococcus suis* is common in certain vocations. They believe some cases of *Streptococcus suis* infection have only a manifestation of moderate fever.

11.6 Diagnostic Examinations

11.6.1 Laboratory Tests

11.6.1.1 General Laboratory Tests

Routine Blood Test

WBC count increases, but in some serious cases, WBC count may be normal or decreased in the early stage. The percentage of neutrophils increases. Some severe patients also have thrombocytopenia. The cases with secondary DIC have serious thrombocytopenia.

Routine Urine Test

There is positive finding of protein and positive finding of ketone in some cases.

Biochemical Detection

In some patients, ALT, AST, and TBil levels increase; albumin decreases; and Cr and BUN levels increase.

Cerebrospinal Fluid Examination

The cases of purulent meningitis have intracranial hypertension, significantly increased WBC count that is commonly above 0.5×10^6/L and mainly multinucleated cells, increased protein, and decreased glucose and chloride.

Blood Gas Analysis

The severe cases commonly have metabolic acidosis, respiratory alkalosis, and type I respiratory failure, with findings of decreased $PaCO_2$, decreased HCO_3, and decreased PaO_2. In the advanced stage, there are also respiratory acidosis and type II respiratory failure, with findings of increased $PaCO_2$, decreased HCO_3, and decreased PaO_2.

DIC Indicators

Patients with DIC have findings of positive 3P test, increased D-dipolymer, and thrombocytopenia.

11.6.1.2 Etiological Identification

The laboratory tests for *Streptococcus suis* include biochemical identification, serotyping, and specific gene detection

after isolation of the strain following bacterial culture. So far, there has been no mature examination for its specific antibody.

Biochemical Identification

Manual identification by Api20-step of API biochemical identification system can be applied for the isolated strain. Otherwise, VITEK 2 Compact or other biochemical identification system can be applied to identify the isolated strain. In such ways, the species of the isolated strain can be defined.

Serotyping

The defined species by biochemical identification can be further defined for its serotype by using 1–34 serotypes of *Streptococcus suis* or monoclonal antibody.

PCR Gene Identification

The isolated and purified colonies or suspected moist colonies on the plate should be firstly chosen. Specific primers are then applied for PCR amplification. The specific primers (tuf) of *Streptococcus* genus, specific *Streptococcus suis* species, type 2 capsular polysaccharide gene (cps2j) of *Streptococcus suis*, related protein encoding gene fragments released by *Streptococcus suis* lysozyme (mrp), and hemolysin gene of *Streptococcus suis* (sly) should be subsequently detected. For those patients who have given large doses of antibiotics, the collected specimens should be directly examined by PCR to detect the specific genes of swine streptococcus species (16SrRNA) as well as specific virulence genes. The cases with positive findings can be defined as human *Streptococcus suis* infection.

11.6.2 Imaging Examinations

11.6.2.1 Ultrasound

Color Doppler ultrasound of the heart is indicated for patients with infective endocarditis.

11.6.2.2 X-Ray

Chest X-ray is indicated for patients with secondary acute respiratory distress syndrome.

11.6.2.3 CT and MRI

Cranial and brain CT scanning and MR imaging are indicated for cases of human *Streptococcus suis infection* complicated by meningitis. Radiological examinations have limited value for the diagnosis and differential diagnosis. However, their applications clinically facilitate understandings about the range, location, and severity of the lesions as well as early finding of complications. Although the radiological examinations lack direct specificity to pathogens of intracranial infections, the imaging demonstrations facilitate to define the diagnosis of various complications. Especially for clinically suspected cases of brain abscesses, intracranial hypertension, and focal signs, MR imaging should be the examination of choice to define the diagnosis.

11.7 Imaging Demonstrations

11.7.1 The Nervous System

In the cases with the nervous system involved, typical purulent meningitis can be demonstrated.

11.7.1.1 CT Scanning

In the early stage, plain CT scanning demonstrates lesions in the interface between gray and white matters that are in low-density or low–high-mixed-density area with poorly defined boundary. However, the lesions have a higher density than their adjacent areas, with spots of bleeding foci inside them. Contrast CT scanning demonstrates no enhancement of the low density area. In some other cases, contrast CT scanning demonstrates irregular spots or gyrus-like enhancement. The adjacent sulci are demonstrated with faded color or to be absent, and there are cerebral edema and space-occupying effect in different degrees around the foci. In the cerebral abscess stage, plain CT scanning demonstrates low-density area with well-defined boundary, intact or not intact abscess wall, and regular or irregular ring-shaped shadows with equal or slightly higher density. Contrast MR imaging demonstrates intact abscess wall in ring-shaped enhancement with thinness and even thickness that are not specific. In the cases with small abscesses, nodular enhancement can be demonstrated. The distribution of foci is not consistent to that of blood vessels.

11.7.1.2 MR Imaging

In the acute inflammation stage of purulent meningitis, a large quantity of exudates fills up the subarachnoid space. MR imaging demonstrates abnormally increased T2WI signals of the cerebral convex and cistern as well as thickened meninges. The early meningeal lesions mainly involve the top cerebral convex and the anterior interhemispheric fissure. The meningeal inflammation often involves the dura mater to cause subdural effusions or empyema. In the advanced stage of meningitis, the subarachnoid space and basilar brain thicken and adhere to block the circulation of the cerebrospinal fluid. Therefore, communicating or obstructive hydrocephalus occurs. Purulent meningitis often involves adjacent brain parenchyma to cause meningoencephalitis. The early manifestations include vascular congestion, brain cells edema, and infiltration of perivascular inflammatory cells. At this time, MR imaging demonstrates plump gyrus, shallow or absent sulci, and local or diffuse long T1 and long T2 signals in the cortex and subcortex. In the

advanced stage of meningoencephalitis, there are capillary hyperplasia and aggravated extracellular edema. MR imaging demonstrates similar findings to tumor but with no tumor body. Therefore, the space-occupying lesions of tumor can be excluded. In the cases of meningitis, meningeal exudates accumulate in the basilar brain to cause vascular inflammation, fiber hyperplasia, luminal stenosis, or even occlusion. Therefore, cerebral infarction occurs. Cerebral abscess is commonly divided into two stages, encephalitic stage and capsular stage. The demonstrations of encephalitic stage include irregular long T1 and long T2 signals with poorly defined boundaries that fuse with the surrounding cerebral edema. The demonstrations of capsular stage include central liquefaction and necrosis of the abscess cavity in long T1 and long T2 signals and extensive edema in the peripheral cerebral parenchyma. Contrast imaging demonstrates ring-shaped enhancement with thin wall and well-defined boundary and no nodular shadows protruding inwards on the abscess wall.

11.7.2 The Respiratory System

In the cases with the respiratory system involved, secondary acute respiratory distress syndrome may occur. Chest X-ray demonstrates increased and blurry pulmonary markings in both lung fields, extensive interstitial infiltration, pleural reaction, and a small quantity of pleural effusion. In severe cases, pulmonary infiltrative shadows fuse into large flakes which may even develop into white lung.

11.7.3 The Circulatory System

In the cases with the circulatory system involved, infective endocarditis may occur. Color Doppler ultrasound of the heart demonstrates enlarged left ventricle and enlarged left atrium, suspected neoplasm on the mitral valve, as well as small quantities of regurgitation via the mitral valve and tricuspid valve. During hospitalization a reexamination was done after 8 weeks therapy, and findings include still enlarged left ventricle and left atrium, absence of neoplasm on the mitral valve, small to moderate quantity of regurgitation via the mitral valve, and small quantity of regurgitation via the tricuspid valve.

11.8 Diagnostic Basis

11.8.1 Human *Streptococcus suis* Infection

In combination with the epidemiological history, clinical manifestations, and laboratory tests findings, the diagnosis can be defined. Other diseases with similar manifestations should be excluded.

11.8.1.1 Diagnostic Evidence

Epidemiological History

The patients have a history of direct contact to diseased or deceased pigs or other livestock within 7 days before the onset. Especially, the patients have defective skin mucosa with a history of slaughtering diseased or deceased pigs, processing or selling pork products prepared from diseased or deceased pigs, and burying diseased or deceased pigs.

Clinical Manifestations

The disease has an acute onset, with chills, fever, and other systematic infectious toxic symptoms. Concurrent TSS and/or SMS can be found.

Laboratory Tests

The peripheral WBC count increases which is mainly neutrophils. Bacterial culture is positive or specific gene detection is positive.

11.8.1.2 Diagnostic Criteria

Suspected Cases

The patients who have a history of direct contact to diseased or deceased pigs or other livestock within 7 days before the onset and have manifestations of acute systemic infective toxicity should be suspected as the cases of human *Streptococcus suis* infection. Otherwise, the suspected cases should have an abovementioned epidemiological history as well as increased peripheral total WBC count and increased percentage of neutrophils.

Clinically Diagnosed Cases

The patients have an abovementioned epidemiological history, with manifestations of TSS and/or SMS.

Definitively Diagnosed Cases

Definitive diagnosis can be made after *Streptococcus suis* is isolated by bacterial culture of specimen collected from suspected or clinically diagnosed cases. Otherwise, the definitive diagnosis can be made based on a positive finding by a specific gene detection of specimen collected from suspected or clinically diagnosed cases.

11.8.2 Human *Streptococcus suis* Infection-Related Complications

11.8.2.1 Purulent Meningitis

The cases with diagnosis of human *Streptococcus suis* infection have clinical manifestations of headache, fever, irritation, consciousness disturbance, coma, and meningeal irritation sign.

Plain CT scanning demonstrates increased density of sulci and cisterns and poorly defined borderline between gyri.

Contrast CT scanning demonstrates enhancement of the brain surface. MR imaging demonstrates deformed subarachnoid space and high T2WI signal with enhancement. In the advanced stage of the disease, cerebral abscess may be demonstrated.

Cerebrospinal fluid examination following lumbar puncture demonstrates cerebrospinal hypertension, increased WBC count, and increased protein.

11.8.2.2 Acute Respiratory Distress Syndrome

The cases with human *Streptococcus suis* infection have respiratory symptoms.

Chest X-ray demonstrates increased and blurry pulmonary markings in both lung fields and extensive interstitial infiltration in both lungs. In severe cases, pulmonary infiltrative shadows fuse into large flakes which may even develop into white lung.

11.8.2.3 Infective Endocarditis

The cases with human *Streptococcus suis* infection have circulatory symptoms.

Color Doppler ultrasound of the heart demonstrates enlarged left ventricle and left atrium, neoplasm on the mitral valve, and regurgitations via the mitral valve and tricuspid valve.

11.9 Differential Diagnosis

Human *Streptococcus suis* infection should be differentiated from other diseases that have manifestations of fever, petechiae, ecchymoses, shock, and multiple organ dysfunction. It should especially be differentiated from the following diseases.

11.9.1 TSS Caused by Other Streptococcus

Group A streptococcus and its produced streptococcal pyrogenic exotoxins (SPE) such as SPE-A, SPE-C, and SPE-F can cause serious TSS. *Streptococcus mitis* infection caused by group B, C, or G streptococcus as well as *Streptococcus viridans* can also cause TSS. In combination with the epidemiological history of direct contact to diseased or deceased pigs within 7 days before the onset, other streptococcus-induced TSS can be differentiated from human *Streptococcus suis* infection. Detection of SPE-A by PCR or immunological assays facilitates the diagnosis. Identification of group A streptococcus or other streptococcus based on specimen collected from the infective position or based on blood culture can differentiate the diagnosis.

11.9.2 TSS Caused by Staphylococcus

TSS caused by staphylococcus is induced by *Staphylococcus aureus* which produces toxic shock syndrome toxin I (TSST-I) and enterotoxins. Its clinical manifestations are similar to those of human *Streptococcus suis* infection. It can be divided into menstrual-related TSS (mTSS) and nonmenstrual-related TSS (nmTSS). The manifestations of sudden onset of the symptoms during menstrual period in young women with a history of using vaginal tampon during menstruation facilitate the diagnosis of mTSS. The cases of TSS after focal infection, trauma, and invasive operations should be suspected as nmTSS. The differential diagnosis should be made based on successful isolation of *Staphylococcus aureus* from clinical specimens or a positive finding of specific TSST-I or enterotoxin A, B, C, D, or G.

11.9.3 Other Diseases

Human *Streptococcus suis* infection should also be differentiated from Gram-positive bacterial septicemia, septic shock, fulminant type of epidemic cerebrospinal meningitis, epidemic hemorrhagic fever, systemic inflammatory response syndrome, and other diseases. In its advanced stage, human *Streptococcus suis* infection should also be differentiated from other serious infections caused multiple organ dysfunction syndrome or multiple organ failure.

Suggested Reading

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