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Nursing care of Patients with Tsutsugamushi Disease (Scrub Typhus)

Ms.Shanthi.K[1], Ms. Mercy Jesudoss[2]
[1](MSN Tutor, Christian Medical College, Vellore, Tamil Nadu.)
[2] (Professor, Christian Medical College, Vellore, Tamil Nadu.)

Abstract
Scrub typhus is a vector borne Zoonotic disease. It is caused by the bite of a trombiculid mite larva (Chiggers) which is infected and carrying Orientia tsutsugamushi (O.tsutsugamushi) bacteria. [1] Globally one billion people are at risk of acquiring scrub typhus. [2] It is a common acute febrile illness prevalent in India causing severe morbidity and mortality. Scrub typhus is endemic and also resurgent disease in southern and eastern parts of Asia. Early diagnosis and proper antibiotics will decrease the morbidity and mortality among patients with scrub typhus.

Keywords: *Scrub typhus, Zoonotic disease, Chiggers, Orientia tsutsugamushi, Endemic, morbidity, mortality*

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I. Introduction

Scrub Typhus is also known as tsutsugamushi disease and Bush typhus. It is a potentially life threatening vector-borne, Zoonotic, acute febrile illness caused by O.tsutsugamushi (Formerly Rickettsia). Bush typhus is caused by after bite of an infected mite vector of the Trombiculidae family (Leptotrombidium delinase and L. akamushi). It affects irrespective of age and both sex with male dominance and it also affects children. Tsutsugamushi is a most common re-emerging infection caused by Rickettsiae, which is prevalent in India and many other South Asian countries.

Scrub means the type of vegetation (Terrain between woods and clearings), that harbors the vector. The word typhus means "fever with stupor" or smoke in Greek. Scrub typhus first described in Japan in 1899. Japanese word Tsutsuga" means small and dangerous and mushi means insect or mite. [3]

Molecular Characteristics of O.tsutsugamushi

O. tsutsugamushi is the causative agent of scrub typhus in India. It differs from other rickettsiae through its antigenic structures. Orientia is a gram negative coccobacillus measures 0.5 - 0.8 × 1.2-3micron. Initially they were categorized under genus rickettsia, but now classified in a separate genus known as Orientia. It is an obligate intracellular gram negative rod shaped bacterium; it grows freely in the cytoplasm of infected cells because it does not have vacuolar membrane. The organisms are highly virulent and exhibit extensive genomic and antigenic heterogeneity. It has specific protein (56-kDa protein) it is unique. O.tsutsugamushi lacks major component of outer membrane called lipopolysaccharides and has low levels of unclassical peptidoglycan. [4, 5]

Serotypes of Orientia tsutsugamushi [6]

More than 30 serotypes of O. tsutsugamushi are prevalent in endemic areas. Following are the habitual serotypes of O.tsutsugamushi

- Gilliam
- Karp
- Kato
- Kawasaki
- Boryon

Epidemiology [7, 8]

- Scrub typhus was first described in China in 313 AD and first isolated in Japan in 1930.
- During World War II, 18,000 cases were observed in Allied troops stationed in rural or jungle areas of the Pacific theatre.
- According to WHO Scrub typhus is probably one of the most under diagnosed and under reported febrile illness requiring hospitalization

- It accounts for up to 23% of all febrile episodes
- 1 million cases occurring annually in endemic areas.
- Mortality rate 30-35% (if not treated early).

Incubation period

- 6 to 21 days after the initial bite

Agent

- O. tsutsugamushi is a causative agent of scrub typhus.

Reservoir

- Larval (Chigger) stage of trombiculid mite act as a primary reservoir and small rodents are secondary reservoir.

Host factors

- It is an occupational disease among rural workers particularly adults involved in agriculture and forest occupations and solders living in temporary camps.

Environmental factors

- Scrub typhus is found in areas with a suitable climate, plenty of moisture and scrub vegetation

Life cycle and mode of transmission [3]

Scrub typhus will not spread directly from person to person, it is spread from one person to another by the bite of an infected larval trombiculid mite. Figure: 1 depicts the four stages of the life cycle of adult mites which are egg, larva, nymph and adult. The larva (Figure: 2) is the only stage that can transmit the disease to humans and other vertebrates, since the other life stages (nymph and adult) do not feed on vertebrate animals. Both the nymph and the adult live freely in the soil. Mites in their larva stage contracts the disease organism by biting rodents.

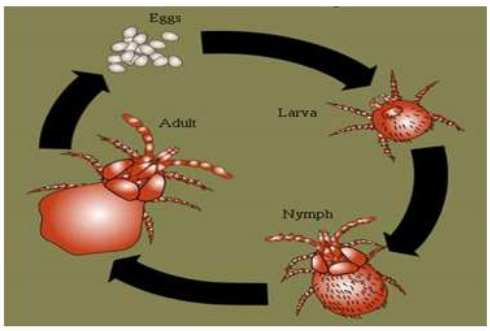

Figure: 1lifecycle

Figure: 2 Larval stage (Chigger)

Season of transmission

- Scrub typhus occurs throughout the year in the tropical areas, whereas in the temperate zones, transmission is seasonal. Outbreaks are seen in the southern parts of India during the cooler season.

Indian scenario [3]

Bush disease is also prevalent in areas like equatorial rain forests, sandy beaches and mountain deserts. The following areas are favorable for the infected mites to flourish in the riverbanks, forest clearings, and grassy region.

Scrub Typhus Triangle [3, 9]

Scrub typhus is an endemic in the "Tsutsugamushi triangle" where it covers 13 million km2, the current reviews indicates that the following countries are covered in the tsutsugamushi triangle, which includes Russia, China, Korea, Japan, and Taiwan (northeast), Australia, Papua, New Guinea, Indonesia, and the islands of the southwestern Pacific (south), Pakistan, Afghanistan, Tajikistan, Nepal, India, Bangladesh, Sri Lanka, and Maldives (west), Myanmar, Thailand, Laos, Cambodia, Malaysia, Vietnam and Philippines (middle).[Figure:3]

Figure: 3 Tsutsugamushi triangle

Pathogenesis [10, 11]

The severity of the disease independently depends on the strains of organism and also on the host

Infected mite passes the infection O. tsutsugamushi (Bacteria) to humans by feeding on the fluid in the skin cells
It affects and infects the dentritic cells in the dermis layer of the skin
Bacteria multiply at the inoculation site, and the papule forms, that ulcerates and becomes necrotic and evolving into an eschar.
O. tsutsugamushi spreads through blood circulation and lymphatic system.
It causes regional and generalized lymphadenopathy through lymphogenus spread
Bacteria enter into blood circulation and continue to proliferate in the endothelium of small blood vessels
Endothelial cells of most organs including skin, heart, lung, brain, kidney, and pancreas are target cells of O. tsutsugamushi.
Interaction between bacteria and endothelial cells which releases cytokines by endothelial and non endothelial cells
Inflammatory cytokines cause destruction of endothelial cells and its leads to fluid leakage, platelet aggregation and proliferation of polymorph and monocytes this also leads to venous thrombosis due to micro infarction.
O.tsutsugamushi attacks endothelial cells, causing local and systemic inflammation by oxidative mechanism resulting in vaso-occlusion.

Clinical manifestation [2]

Ranges from mild to fatal begins with in 7-10days of being bitten

- Eschar: lesion measures 1cm in diameter and looks flat, then it becomes elevated and filled with fluid, when it ruptures it will be covered with black scab. (Localized cutaneous necrosis at the site of mite bite), often found in groin, axilla, genitalia and neck (Figure:4)

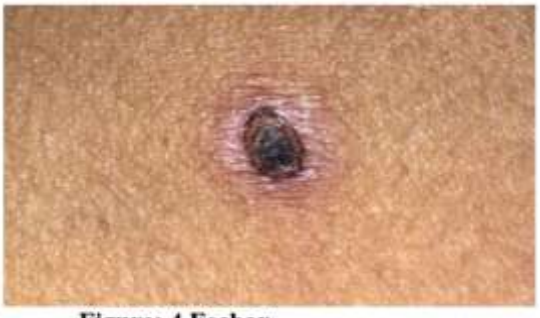

Figure: 4 Eschar

- Regional and Generalized lymphadenopathy
- Fever
- Headache
- Myalgia
- Cough
- Gastrointestinal symptoms like vomiting, abdomen pain
- Transient hearing loss

- Conjunctival infection
- Mental Changes, ranging from confusion to coma
- Maculopapular rash

In severe cases

- Tachycardia
- Hypotension
- Muscular twitching
- Acute respiratory distress syndrome (ARDS)
- Myocarditis
- Acute kidney injury
- Aseptic meningitis
- Splenomegaly
- Pneumonitis
- Delirium
- Stupor
- Loss of consciousness

Scrub typhus in pregnancy

It may cause spontaneous abortions, stillbirths, preterm birth and low birth weight

Diagnostic evaluation [2, 12]

- Chest X ray
- Renal function test – Raised creatinine
- Liver function test – Elevated transaminases
- EKG: Show nonspecific ST and T wave changes, arrhythmias and heart block
- CSF: Show lymphocytic pleocytosis and increased protein and normal sugar
- Weil Felix
- Indirect immunofluorescence assay (IFA)
- Enzyme linked Immunosorbent Assays (ELISA)
- Rapid lateral flow assay
- KpKtGm r56 Western blot
- ImmunochromatographicTest (ICT)
- The indirect Fluorescent Assay
- Indirect Immunoperoxidase (IIP)
- PCR amplication of Orientia genes
- Bacterial Culture: O.tsutsugamushi can be isolated and cultured by inoculating it intraperitoneally in to mice and it can be demonstrated in the tissues of the mice, by Giemsa staining.
- CT Scan Chest, Abdomen and Brain
- MRI brain
- Ultrasound Abdomen,
- UGI endoscopy – superficial mucosal hemorrhages

Treatment [13]

Choice of agent: Following antimicrobial agents are commonly used to treat scrub typhus.

Name of the drug	Dose and administration in adults
Tab/Inj.Doxyecycline Drug of choice	• 100 mg twice daily for 7 days
Azithromycin It is safest drug for pregnancy and children	• Mild infections: 500 mg single dose • Severe infections: 500 mg once daily for 3 to 5 days • 1g loading dose may be given
Chloramphenicol It is a 2nd line treatment	• 500 mg every 6 hourly for 7 days
Tetracycline	• 500 mg four times daily

Supportive treatment

- Fever –Antipyretics
- Respiratory failure – Oxygen therapy, Non invasive ventilation (BiPAP), Mechanical ventilation
- Circulatory shock – Fluid resuscitation and inotrops
- Coagulation disorder (DIC) – Blood products
- Renal failure – Renal replacement therapy

Complications includes [2]

Pulmonary

- Acute respiratory distress syndrome (ARDS)
- Diffuse Interstitial pneumonia
- Pleural effusion

Neurological

- Meningitis
- Meningo encephalitis
- Altered sensorium
- Seizures

Cardiac

- Myocarditis
- Reversible Cardiomegaly
- Atrial flutter
- Atrial fibrillation
- Atrial standstill
- Heart block

Renal

- Acute kidney injury
- Vasculitis of the renal vessels

Hepatic

- Acute hepatic failure

Other

- Hemophagocytic syndrome
- Systemic vasculitis
- Transient adrenal insufficiency
- Septic shock
- Gastrointestinal bleeding
- Disseminated intravascular coagulation
- Multi organ failure

Prognosis

- Scrub typhus is curable if it is identified early and treated with appropriate antibiotics. Untreated and undiagnosed Scrub typhus leads to 70% mortality rate due to fast and unpredictable progression and multi organ failure.

Prevention [10]

- No vaccine is available
- Wear protective clothing
- Avoid travelling to areas where scrub typhus is common
- Avoid areas with lots of vegetation and bush where chiggers present
- Avoid drying clothes on bushes
- Change clothes and have a bath, after coming back home from working in the fields
- Chemoprophylaxis
- Long acting tetracycline
- Doxycycline

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Case Report-1

A 40 years old female presented to Emergency Department (ED) with the chief complaints of high grade fever and breathing difficulty for 4 days, worsened over the last 2 days. She is a known case of mitral valve regurgitation secondary to Rheumatic heart disease. On arrival to ED, she was conscious and oriented with a GCS of 15/15. Her vital signs were; Pulse rate 118/minute, Respiratory rate 44/minute, Blood pressure 90/60mmHg, Temperature 100^0F and SpO2 of 98%. Inj. Nor adrenaline was started at the rate of 5mcgs/min. Physical examination revealed the presence of an eschar in right axilla. On chest auscultation, diffuse crepts were heard bilaterally. The IgM ELISA for Scrub typhus was positive. She was diagnosed to have Scrub typhus with acute respiratory distress syndrome and shock. She was admitted to the general medical ward. Initially, she was on Non- invasive ventilation (NIV) BiPAP (IPAP; 12cmH2O, EPAP; 6cm H2O) support for tachypnea and increased work of breathing. She continued to be on Inj. Nor-adrenaline for her low BP management (80/56mmHg) however, her BP continued to remain the same; hence the infusion rate was increased to 10mcgs/min. She de-saturated (SpO2 86%) and her ABG value were PH-7.50; PCO2- 27mmHg; HCO3- 21mEq/L; and PF ratio of 277 mmHg (Partially Compensated Respiratory Alkalosis). Therefore, she was intubated and transferred to Medical Intensive Care Unit for mechanical ventilation support. She was treated effectively with mechanical ventilation, SIMV mode with volume control, settings are FiO2-50; Pressure support (PS) - 15cmH2O; PEEP-12cmH2O; and Tidal volume -340ml. She was on Inj. Vasopressin infusion at 1.2u/hour; Inj. Fentanyl infusion at 50mcg/hour; and Inj. Lasix infusion at 2mg/hour. Proning was done to improve gas exchange. Antibiotic therapy, IV fluids and Inotropic support was given then, slowly she was weaned off the ventilator and inotropic support. Following which she was started on oxygen therapy via face mask and was shifted to general medical ward for step down care. With continuous monitoring and effective nursing care, she was discharged and advised to come for regular follow up.

1. Nursing Diagnosis: Ineffective airway clearance related to pooling of tracheo-bronchial secretions

Expected outcome: She maintains patent airway as evidenced by equal air entry bilaterally and absence of secretion

Intervention:

- Bilateral air entry was auscultated. Air entry was equal, bilateral crepts were heard
- Performed chest physiotherapy and tracheal suctioning done every Q2hourly and whenever necessary
- Assessed the color, amount and consistency of the secretion. It was thick and white in color
- Q6h normal saline nebulization was administered
- Q2hourly position was changed
- IV fluids was administered to maintain hydration

Evaluation: Her airway was maintained patent as evidenced by bilateral air entry is equal and absence of copious secretion.

2. Diagnosis: Impaired breathing pattern related to respiratory muscle fatigue and decreased energy

Expected outcome: She maintains an effective breathing pattern as evidenced by relaxed breathing at normal rate and depth and blood gas level within normal limits

Intervention:
- Assessed the respiratory rate, rhythm and depth. She was tachypneic RR- 44/ mt and increased work of breathing
- On auscultation bilateral crepts were heard
- Vital signs are monitored PR- 110/mt, BP-80/60mmHg, and SpO2 86%
- ABG results revealed respiratory alkalosis
- Endo tracheal intubation was done and mechanical ventilation provided
- Gradually she was weaned from ventilator and maintained effective breathing pattern
- Oxygen provided via face mask, it was discontinued within few days
- Iv fluids was administered to meet her energy
- Naso Gastric feeds was started 200 every q2hourly

Evaluation: She maintained effective breathing pattern as evidenced by normal breath sounds, SpO2 100% without mechanical ventilation

3. Nursing Diagnosis: Impaired gas exchange related to ventilation perfusion mismatch

Expected outcome: She maintains effective gas exchange as evidenced by P/F ratio more than 300 and SpO2 more than 90%

Intervention:
- Assessed her ABG values. PH -7.50; PCO2- 27mmHg; HCO3- 21mEq/L
- Assessed her spO2 level it was 86%
- Initially she was on NIV support later intubated endotracheally and connected to mechanical ventilator
- Maintained cuff pressure of 25mmHg to prevent air leak
- PEEP was maintained 12cmH2o to open fluid filled alveoli and improve the gas exchange
- She was positioned in prone position to improve gas exchange
- Her PF ratio improved progressively day by day likewise 320, 338, 370, 390, 395 mmHg

Evaluation: She maintained effective gas exchange as evidenced by a P/F ratio of 395mmHg and 100% of SpO2

4. Nursing Diagnosis: Fluid volume deficit related to increased capillary permeability

Expected outcome: She maintains adequate fluid volume as evidenced by urine output greater than 30ml/hr, normal blood pressure, PR less than 100 beats/mt and normal skin turgor

Intervention:
- Assessed her HR, BP, Peripheral pulses, temperature and color of the extremities, capillary refilling time (CRT), mean arterial pressure (MAP) and urine output. Initially Her PR rate was 110/mt, BP was 90/60mmHg, MAP was 70mmHg, and peripheral pulses were weak and thready. Extremities were pale and cold to touch, CRT was 3seconds and urine output was normal.
- Fluid resuscitation was done with crystalloids
- Inj. Nor adrenaline infusion was started @10mcg/mt to improve cardiac contractility
- IV antibiotics administered as per the order to treat gram negative bacteria which affected the permeability of the capillary wall.
- Strict intake output chart was maintained
- Skin looks warm and has normal skin turgor

Evaluation: She maintained normal fluid volume as evidenced by normal BP 120/80mmHg, PR 72/mt, normal urine output and normal skin turgor

5. Nursing Diagnosis: Hyperthermia related to increased basal metabolic rate secondary to gram negative bacterial infection

Expected outcome: She maintains normal body temperature as evidenced by Temperature 98 degree F

Intervention:
- Temperature was monitored it was 100degree F
- Adequate ventilation provided
- Removed tight cloths
- Administered Inj. Paracetmaol IV as per order
- IV antibiotics administered as per the order to treat gram negative bacteria

Evaluation: She maintained normal body temperature as evidenced by Temperature reduced to 98degree F
6. Nursing Diagnosis: Anxiety related to lack of clients knowledge regarding disease condition and prognosis
Expected outcome: Anxiety will be reduced as evidenced by patient relatives will be looking calm and cooperative

Intervention:
- Assessed their level of anxiety, patient relatives were looking anxious
- Reassured them
- Explained about the disease condition and prognosis of the patient
- Explained about treatment and procedures
- Allowed them to express their feelings and thoughts
- Clarified their doubts
- Psychological and spiritual support given to them

Evaluation: Anxiety was reduced as evidenced by relatives were looking calm and cooperative

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