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Review on Q Fever: Epidemiology, Public Health Importance and Preventive Measures

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Summary

Q fever is worldwide zoonotic disease which is caused by obligate intracellular gram-negative bacteria, called *Coxiella burnetii*. Q fever is air born disease and thus inhalation is considered as primary mode of transmission in both animal and human, sometimes through ingestion they may be infected and ticks play essential role in transmission. The agent is spread a long distance through the wind. The geographical distribution of Q fever is worldwide except New Zealand. The main reservoirs for human infections are cattle, goat, sheep, and pets and ticks are the natural primary reservoir for animal. *Coxiella burnetii* in ruminant cause reproductive problems like miscarriage, infertility and reduced milk production. The organism can be found in the milk, urine, feces placenta and birth fluids of animals. The airborne transmission of *C. burnetii* associated with its highly resistance to environments and the ability to easily produce huge quantities of *C. burnetii* in the after birth of aborted ewes or goats have led to classify *C. burnetii* as a Category-B, biological terrorism agent. The incubation period of Q fever is depending on the size of infectious. The recommended treatment for ruminant administering two injection of Oxytetracycline during the last month of gestation, also Doxycycline is the best drug. *C. burnetii* can be reduced in the farm environment by regular cleaning and disinfection of animal facilities. Q fever is global health problem and it is an OIE notifiable disease. Q fever is one of infectious disease which is considered as being having economic and public health importance in Ethiopia. Therefore, awareness creation and, application of prevention and control method has paramount importance in reduce the hazardous effect of this disease.

Abbreviations: SCV: Small cell variant; LCV: Large cell variant; CFSPH: Central food security public Health; PCR: Polymerase chain reaction; LPS: Lipopolysaccharide; IFA: Indirect fluorescent antibody; OIE: Office international des Epizooties

Introduction

Q fever is a serious zoonotic disease caused by an obligate intracellular bacterium *Coxiella burnetii* and it is presumed to be one of the most widespread zoonosis in the world. The disease has known since the 1930s and has a worldwide distribution, with the exception of the Antarctica and possibly New Zealand [1]. The disease first identified in Queensland, Australia, in 1935, after an outbreak of febrile illness among slaughterhouse workers [2]. Some authors suggested that the Q stood of Queensland, the state in which the disease was first founds [3], but after testing all those who were affected and could not arrive at a diagnosis from the patients' history, physical examination, and a few investigations, Derrick termed the illness "Q" for Query fever, because its etiopathogenesis was not known at the time [4]. It also known by several synonyms such as Abattoir fever, Australian Q fever, Balkan influenza, Coxiellosis, Nine-mile fever, and Pneumorickettsiosis [5].

Domestic animals such as cattle, sheep and goats are considered as the mainreservoir for the pathogen which can infect a large variety of animals, humans, birds, and arthropods [5]. Q fever is a mainly airborne zoonosis; infection is most acquired by breathing infectious aerosols or contaminated dust [6]. Infection can occur also in individuals not having direct contact with animals, such as persons living along a road used by farm vehicles or those handling contaminated clothing. The infection result from

inhalation of endospores and from contact with the milk, urine, feces, vaginal mucus or semen of infected animals [7]. The pathogen is highly resistant to adverse physical conditions and chemical agents, so it can survive for months and even years in the environment which create conducive condition for infection [8].

Q fever is frequently asymptomatic, in sheep and goats it causes abortion, stillbirth, premature delivery, and delivery of weak offspring and in cattle and camel may develop infertility, metritis, and mastitis [9]. The majority of human *coxiella burnetii* infections are asymptomatic, especially among high-risk groups such as veterinary and slaughterhouse workers, other livestock handlers, and laboratory workers [10]. In more recent dates Q fever is classified as a “Category “B” critical biological agent” by the Centre for Diseases Control and Prevention (CDC) and is considered a potential weapon for bioterrorism [11]. The disease so is considered has having public health concern throughout the world alongside with its economic importance. On top this, although Q fever is an OIE notifiable disease, it remains poorly reported and its surveillance is frequently severely neglected [12]. Review of the disease epidemiologic status, public health significance is lacking in different countries, including Ethiopia.

Therefore, the objectives of this seminar paper are:

- i. To overview Q Fevers Epidemiology, Public health importance, and preventive measures
- ii. To highlight current status of Q fever in Ethiopia

History of Q Fever

Q fever was first described in 1935 by Edward Holbrook Derrick [2] in abattoir workers in Brisbane, Queensland, Australia. The “Q” stands for “query” and was applied at a time when the causative agent was unknown; it was chosen over suggestions of “abattoir fever” and “Queensland rickettsial fever,” to avoid directing negative connotations at either the cattle industry or the state of Queensland [13]. Derrick inoculated guinea pigs with blood or urine from the “Q” fever patients. The guinea pigs became febrile. Derrick was unable to isolate the agent responsible for the fever so he sent a saline emulsion of infected guinea pig liver to Macfarlane Burnet in Melbourne. Burnet was able to isolate organisms, which “appeared to be of rickettsia nature” [7].

In 1936, Herald Rea Cox joined Davis at the Rocky Mountain Laboratory to additional characterize the “Nine Mile agent.” Burnet and Freeman, as well as Davis and Cox, demonstrated that the etiological agent was filterable and displayed properties of both viruses and rickettsiae. A

major advance obtained in 1938, when Cox succeeded in propagating the infectious agent in embryonated eggs. Cox termed the Nine Mile Agent *Rickettsia diaporica* (*diaporica* means having the ability to pass through) a reference to the filterable property of the agent. In intervening time in Australia, Derrick suggested the name of *Rickettsia burnetii* for the Q fever agent. In 1948, Cornelius B. Philip proposed that *R. burnetii* considered as the single species of a distinct genus since it was now apparent that this organism was unique among the rickettsiae. He proposed the name *Coxiella*. The Q fever agent is known as *Coxiella burnetii*. *C. burnetii*, a name which honors both Cox and Burnet who had identified the Q fever agent as a new rickettsial species [14].

Etiology and Taxonomy

Coxiella burnetii, the causative agent of Q fever, is a small (3–5mm) polymorphic obligate intracellular gram-negative bacteria belonging to the genus *Coxiella*, family *Coxiellaceae*, order *Legionellales*, class *Gammaproteobacteria*, and phylum *Proteobacteria* [15]. *C. burnetii* display two antigenic phases based on changes that occur in the organism during in vitro culture, such as phase I and phase II [16]. They are liable to the Lipopolysaccharide (LPS) of the membrane. Phase-I *Coxiella burnetii* antigen are more highly infectious and are corresponds to the smooth phase of Gram-negative bacteria and phase-II antigen is corresponding to the granular (Rough) phase which has a lower virulence [17]. *C. burnetii* has a biphasic life cycle, alternating between a large cell variant (LCV), which is the replicating form within a cell, and a small cell variant (SCV), the non-replicating, infectious form. The SCV has an unusual spore-like structure with highly condensed chromatin, and it is highly resistant to environmental conditions [18].

Since, *Coxiella burnetii* thought the only species member of genus *Coxella* but recently several Candidate species have been recognized in reptiles, birds, and humans like *C. cheraxi*, *C. avium* and *C. massiliensis*. *Coxiella*-like bacteria are also common in ticks, and one of these organisms was recently found in horses. The newly-recognized relatives of *C. burnetii* have the potential to alter some aspects of its epidemiology for instance, *C. burnetii* is often said to occur in more than 40 species of ticks; however, the current PCR tests can also amplify *Coxiella*-like bacteria, and whether all of these ticks were truly infected with *C. burnetii* is now in doubt [19].

Genomic group contains reference *Coxiella burnetii* strain that were isolated from infected human end/or animals. Strain in genomic group 1, 2 and 3 have been isolated from tick, human blood (acute Q fever), from milk of persistently infected dairy cattle, and/or aborted

fetal tissue. Strains in genomic group 4 and 5 have been isolated from the heart of humans with chronic Q fever and/ or aborted tissue of animals. Strains in genomic group 6 have been isolated only from rodents; these strains are of unknown virulent for humans and animals [20].

Epidemiology

Reservoirs of Q Fever

The reservoir includes many wild and domestic mammals, birds, and arthropods such as tick [21]. Over 40 species of ticks are naturally infected. The organisms multiply in the cell of the midgut and stomach of the ticks. After multiplying ticks excrete bacteria in the feces and saliva [12]. Throughout the world the most commonly identified reservoirs of human infections are farm animals such as cattle, goats, and sheep, Pets, including cats, rabbits, and dogs, have also been demonstrated to be potential sources of urban outbreaks of disease [22].

Geographical Distribution

Q fever disease outbreak occurs primarily in Queensland, Australia, in 1935 in slaughterhouse workers [2]. Q fever has described worldwide except New Zealand [23]. Two characteristics of the organism are important in the epidemiologic distribution of the disease. These are its ability to withstand harsh environmental conditions, probably as a result of spore formation and its extraordinary virulence for man. *C. burnetii* has been a very successful pathogen because of this a single organism can cause disease in man. In 1955, Q fever had been reported from 51 countries on five continents. From 1999 to 2004, there were 18 reported outbreaks of Q fever from 12 different countries [24].

Transmission and Source of infection

The organism can be found in the milk, urine and feces of the animals as well as the placenta and birth fluids. Humans are most commonly infected through inhalation of contaminated dust or aerosols generated by livestock operations involving these animals [25]. Accordingly, Q fever is an occupational hazard for veterinarians, abattoir workers, dairy farmers and anyone with regular contact with livestock or their products [7]. Urban outbreaks of Q fever are also associated with contact with infected domestic cats [26]. Inhalation is the primary mode of transmission in both human and animal. Under experimental conditions, inhalation of a single *C. burnetii* can produce infection and clinical disease in humans. *Coxiella burnetii* also spreads by wind causing infections at a distance from the initial source of bacteria. In domestic ruminants, milk is the most regular

route of pathogen shedding and thus recently, OIE advises not to drink raw milk originating from the infected livestock farms [27].

Tick may play important role in transmission of *C. burnetii* among the wild vertebrates, especially in rodents, lagomorphs, wild birds. Dog can also be infected by tick bites. Although experimental transmission of *C. burnetii* from infected to uninfected guinea pigs via tick bites has been performed with *Ixodes holocyclus*, *Haemaphysalis bispinosa*, *Rhipicephalus sanguineus*, ticks are not important in natural cycle of *C. burnetii* infection in livestock. Tick expels *C. burnetii* with their feces in the skin of animal host at the time of feeding [28]. Human-to-human transmission does not usually occur [12].

Risk Factors

Agent Factors: The severity of the infection depends on the strains of the infecting bacteria. Phase I type bacteria are more virulent than the phase II type. Acute infection in humans is caused by *Coxiella burnetii* genomic type III, whereas type IV and V are responsible for chronic infection. The virulence of type VI is unknown [29,30].

Host Factors: Age and gender are the two risk factors which are shown to influence the occurrence of Q fever in humans. Old people are the most vulnerable and the clinical disease is mostly prevalent in men [31]. An interaction of *Coxiella burnetii* infection with age and sex was also found in animals, particularly in cattle. The prevalence of *Coxiella burnetii* infection increases with age or with the number of parity in cattle and sheep. Prevalence is higher in dairy cows than in beef cattle. Veterinarians, animal farm workers, abattoir workers, laboratory personnel, and immunosuppressed people are at a higher risk of being infected or seropositive than others; and highly prevalent [32].

Environmental Factors: Seasonal variation is observed in the occurrence of human Q fever. This variation, however, varies according to geographical region. But most cases of Q fever have been reported in the spring or early summer. Human Q fever has been shown to have a relationship with rainfall rather than season [31,33].

A high prevalence of Q fever was observed among people living in close proximity to infected animals or in areas with a high livestock density. Several management factors such as housing systems, isolation of a newly introduced animal may also contribute to the seroprevalence of *Coxiella burnetii* infection in animals [32].

Pathogenesis

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The pathogenesis of *Coxiella burnetii* infection in humans and animals is not clearly understood. But, it is believed that bacterial LPS play an important role in the pathogenesis of Q fever in both humans and animals. The organism probably follows the oropharyngeal route as its port of entry into the lungs and intestine of both humans and animals. The disease is highly infectious, and a very low dose is enough to initiate infection. Primary multiplication takes place in the regional lymph nodes after the initial entry, and a transient bacteremia develops which persists for five to seven days [17].

The SCVs are shed by infected animals. After infection the organism attaches to the cell membrane of phagocytic cells. After phagocytosis, the phagosome containing the SCV fuses with the lysosome. The SCVs can undergo vegetative growth to form LCVs. The LCVs and the activated SVCs can both divide by binary fission and the LCV can also undergo sporogenic differentiation. The spores that are produced can undergo further development to become metabolically inactive SCVs and both spores and SCVs can then be released from the infected host cell by either cell lysis or exocytosis. The entire development cycle of metabolically active *C. burnetii* takes place in acidic phagolysosomes; *C. burnetii* are resistant to microbicidal activities in the host macrophages. The acidic environment also protects *C. burnetii* from the effects of antibiotics. The SCV and spore forms are more difficult to denature than LCVs [34].

Clinical Sign

In animals, during the acute phase, *C. burnetii* can be found in the blood, lungs, spleen, and liver whereas during the chronic phase it is presented as a persistent shedding of *C. burnetii* in feces and urine. Most animals remain totally asymptomatic, including a lack of fever. However, low birth weight animals can occur. Clinical symptom of Q fever in sheep and goats are pneumonia, abortion, stillbirth, premature delivery, and delivery of weak offspring or reproductive failure. In cattle, Q fever is frequently asymptomatic. Clinically infected cows develop infertility, metritis, and mastitis. In addition, *C. burnetii* was found to be significantly associated with placentitis. Placental necrosis and fetal bronchopneumonia were also significantly associated with the presence of *Coxiella burnetii* in the trophoblasts. In most abortive cases, the aborted foetus appears normal but discolored exudates and intracotyledonary fibrous thickening may be observed in an infected placenta. Severe myometrial inflammation and metritis are the frequently observed clinical manifestations in goats and cows, so abortion rate is comparatively higher in ewes and goats than in cows. Abortion is usually observed

in late pregnancy in ewes, goats, and cattle [1]. Infection in most domestic animals remains unrecognized. Coxiellosis is considered a cause of abortion and reproductive disorders in domestic animals.

Temperature is an important factor related to abortion rates in herds, since fewer abortions take place between months of November and December, the occurrence of abortion rate increases gradually from January to February, decreasing again in March [1]. In Humans *Coxiella burnetii* infection can cause either an asymptomatic, acute, or chronic disease. Clinical manifestations of Q fever in human are Prolonged fever, Pneumonia, Hepatitis, Endocarditis, Osteomyelitis, Neurological manifestations, Skin rash and Myocarditis. A prolonged fever, which may reach 39-40°C, usually stays for 2-4 days and then gradually decreases to a normal level through the following 5-14 days. The fever is usually accompanied by severe headaches. However, in untreated patients, fever may last from 5 to 57 days. Pneumonia is mild in most cases being characterized by a dry cough, fever, and minimal respiratory distress.

Almost all patients suffering from acute Q fever pneumonia present with a fever usually associated with fatigue, chills, headaches, myalgia, and sweats. Myocarditis is a rare but life-threatening clinical manifestation of acute Q fever and it is found in 2% of patients with the acute illness and it may be associated with pericarditis, and a pericardial effusion may be observed on chest radiographs. Clinical manifestations of Q fever pericarditis are not specific and most often correspond to a fever with thoracic pain [35]. Q fever hepatitis is usually only revealed by an increase in hepatic enzyme levels. Q fever hepatitis is usually accompanied clinically by fever and less frequently by abdominal pain (especially in the right hypochondrium), anorexia, nausea, vomiting, and diarrhea. Progressive jaundice and palpation of a mass in the right hypochondrium have also been reported. Extensive destruction of liver tissue leading to hepatic coma and death has occasionally been reported. Skin rashes and neurologic disorders such as meningoencephalitis or encephalitis, lymphocytic meningitis and peripheral neuropathy have also been observed in acute Q fever. Skin lesions have been found in 5–21% of Q fever patients in different series. The Q fever rash is nonspecific and may correspond to pink macular lesions or purpuric red papules of the trunk. There are 3 major neurological entities associated with Q fever: (1) meningoencephalitis or encephalitis; (2) lymphocytic meningitis and (3) peripheral neuropathy.

Spontaneous abortion, intrauterine fetal death, premature delivery or retarded intrauterine growth may

occur in women that become infected during pregnancy. When a woman is infected by *C. burnetii* during pregnancy, the bacteria settle in the uterus and in the mammary glands [36]. Q fever endocarditis is the most frequent clinical presentation of chronic Q fever. It occurs almost exclusively in patients with a previous cardiac defect or in immune compromised patients. Unspecific signs like intermittent fever, cardiac failure, weakness, fatigue, weight loss or anorexia may be present. Other manifestations are osteomyelitis, osteoarthritis, chronic hepatitis, hepatomegaly, splenomegaly, digital clubbing, purpuric rash and an arterial embolism. The incubation period is depending on the size of the infectious doses, usually 2 to 3 weeks. Chronic Q fever can develop years after an initial infection [37].

Diagnosis, Differential Diagnosis and Treatment

Diagnosis

The clinical signs of Q fever are nonspecific both in human and animal because of this laboratory evidence of infection is needed for diagnosis. Four categories of diagnostic tests are available: isolation of the organism, which must be conducted in a biosafety-level 3 laboratory using tissue-culture; laboratory animals, or embryonated eggs; serologic tests, including indirect fluorescent antibody (IFA), enzyme immunoassay, and complement fixation test; antigen detection assays, including immunohistochemical staining (IHC); and nucleic acid detection assays, including polymerase chain reaction (PCR) assays [5]. Routine diagnosis of Q fever in animals is usually established by examination of fixed impressions or smears prepared from the placenta stained by the Stamp, Gimenez or Machiavello methods, associated with serological tests [38].

Differential diagnosis

There is some disease that we appreciate the same sign with Q fever such as Salmonellosis, Brucellosis, Leptospirosis, Campylobacteriosis, Listeriosis, Elective Abortion, Influenza, and Rickettsial Infection. At initial stages, i.e., before pulmonary symptoms are present, influenza may be suspected. Listeriosis is called circling disease, affected animal circle in one direction only and show swallowing, fever, blindness and head pressings. There is necrosis of placenta which leads to abortion and the fetus may be macerated or delivered weak and moribund, paralysis and death follow in 2 to 3 weeks later. Listerial abortion occurs in late gestation. Brucella is life longer infection it causes in female animal abortion around seventh month of pregnancy and retention of placenta and metritis are common and in male it causes orchitis, epididymitis, synovitis and sterility.

Salmonellosis has sign like fever, dehydration and foul-smelling diarrhea and cause abortion in the last two months of gestation. Leptospirosis is show sign like excessive salivation, muscular rigidity, conjunctivitis, hemoglobinuria, pallor of mucosa and jaundice. Leptospiral abortion occurs with or without placental degeneration and encephalitis, Abortion usually occurs 3-4 weeks later. Most affected animals are found dead, apparently from septicemia [32].

Treatment

Antibiotics can shorten the course of acute Q fever, and may reduce its severity. The recommended administrations for human acute Q fever are Doxycycline (100 mg daily for 14 days), Fluoroquinolones (200mg three times a day or Pefloxacin (400mg) for 14-21 days), Rifampin (1200mg per day for 21 day). For pregnant patient we use drug like Trimethoprim (320mg) and Sulfamethoxazole (1600mg) for >5 weeks. Doxycycline (100mg per day for 10-14 days) is suitable. Fluoroquinolones are considered to be a reliable alternative and have been supported for patients with Q fever meningoencephalitis, because they penetrate the cerebrospinal fluid. Cotrimoxazole and rifampin can be used in case of allergy to tetracyclines or contraindication). Erythromycin and other new macrolides such as clarithromycin and roxithromycin, could be considered a reasonable treatment for acute *C. burnetii* infection [39].

The recommended administration for human chronic Q fever is Doxycycline (100mg per day) and Hydroxychloroquine (600mg) for >18 months for adult, Trimethoprim and Sulfamethoxazole for >18 months for children. Doxycycline is the first choice of drug treatment for all adults and children in both acute and chronic case those have sever illines. Tetracyclines are recommended most often in non-pregnant patients, but other drugs are sometimes used such as Quinolones, and Trimethoprim/ sulfamethoxazole ("cotrimoxazole"). Cotrimoxazole is much employed in pregnant women to avoid side effects from other drugs. Treatment of chronic Q fever is more difficult because of this single antibiotic are not generally effective. Tetracyclines combined with hydroxychloroquine are traditionally used, typically for 18-24 months, but tetracyclines combined with quinolones have also been used successfully. The recommended treatment for ruminant (animal) is consist in administering two injection of oxytetracycline (20 mg per kg body weight) during the last month of gestation. This treatment does not completely suppress the abortions and shedding of *C. burnetii* at lambing.

Prevention and Control

To prevent and reduce the animal and environmental

contamination, several actions can be proposed. When introducing a new animal into a Q fever free flocks, in order to avoid the spread of infection, specific care taken. An antibody investigation for Q fever should be performed in the flock of the seller and animals from seropositive flocks can only be introduced in seropositive or vaccinated flocks. *C. burnetii* can be reduced in the farm environment by regular cleaning and disinfection of animal facilities, with particular care of parturition areas, using 10% sodium hypochlorite. In the UK, Health Protection Agency guidelines mention the use of 2% formaldehyde, 1% Lysol, 5% hydrogen peroxide, 70% ethanol, or 5% chloroform for decontamination of surfaces. Pregnant animals must be kept in separate pens, placentas and aborted fetuses must be removed quickly and disposed under hygienic condition to avoid being ingested by dogs, cats or wildlife. Since parturition is critical for the transmission of the disease, in infected flocks, birth must take place in a specific location, which must be disinfected as well as every utensil used for delivery.

Appropriate tick control strategies and good hygiene practice can decrease environmental contamination. Infected fetal fluids and membranes, aborted fetuses and contaminated bedding should be incinerated or buried. In addition, manure must be treated with lime or calcium cyanide 0.4% before spreading on fields; this must be done in the absence of wind to avoid spreading of the microorganism faraway. The best methods available for prevention and control of Coxiellosis are antibiotic treatment and vaccination. In-feed addition of tetracycline or injectable oxytetracycline pre-partum has not been shown to prevent *C. burnetii* shedding in feces, milk and vaginal secretions [40].

Human-to-human transmission is extremely rare and Q fever is mainly an airborne disease, measures of prevention are aimed at avoiding the exposure of humans and particularly persons at risk, to animal and environmental contamination. At human level, prevention of exposure to animals or wearing gloves, boots, and masks during manipulation of animals. Pasteurization at 72 °C during 15 s, or sterilization of milk from infected flocks is regularly recommended even if the oral route is not the main one [41].

C. burnetii is able to survive for long periods in the environment and in wild animals. The only way to really prevent the disease in ruminants is to vaccinate uninfected flocks, with an efficient vaccine. Three types of vaccine have been proposed for providing human protection against Q fever: the attenuated live vaccine (produced and trialled in Russia but subsequently abandoned because of concern

about its safety); chloroform–methanol residue extracted vaccine or other extracted vaccines (trialled in animals but not humans); and the whole-cell formalin-inactivated vaccine, which is considered acceptably safe for humans [40]. Vaccines can prevent abortion in animals, and it is evident that a phase I vaccine must be used to control the disease and to reduce environmental contamination and thus, the risk of transmission to humans. The widespread application of such a vaccine in cattle in Slovakia in the 1970s and 1980s significantly reduced the occurrence of Q fever in that country [42].

Public Health Importance of Q Fever

Q fever is a global public health concern, as is reported from more than 59 countries of the world [43]. Human infections occur after inhalation of aerosols generated from contaminated dust resulting from contaminated manure and desiccation of infected placenta.

The existence of *C. burnetii* in the environment allows it to be disseminated by wind far away from its original source. This indicates the appearance of Q fever cases in urban areas, where an important percentage of patients fail to report direct contact with animals [44]. Apparently without animal contact, birds can also be responsible for human cases in urban areas since they are able to transmit Q fever via their feces or their ectoparasites [45].

C. burnetii received as a Category-B, biological terrorism agent. The attention of public health personnel and medical on Q fever, which could be responsible for the apparent increase of Q fever cases and its apparent reemergence, has been focused. Less efficient route of contamination is ingestion of contaminated raw milk or raw milk products. Without clinical signs, seroconversions in human volunteers have been induced by drinking of contaminated milk, but none of them presented aggravating risk factors. Nevertheless, some studies have reported clinical disease linked to the ingestion of cheese; but these outcomes are sometimes contested since it is difficult to guarantee even for prisoners that the patients did not inhale contaminated dust or aerosols [46].

It is highly infectious for risky groups including veterinarians, laboratory workers, farmers and abattoir workers. Surveys have shown that significant numbers of livestock handlers have antibodies indicating exposure to the organism. Less than half of people infected become ill, and most infections are mild. But affected persons can develop a high fever with headache, muscle pains, sore throat nausea and vomiting, chest and stomach pains. The fever can last for one or two weeks, and lead to pneumonia

or affect the liver. People with suppressed immune systems and those with pre-existing heart valve problems are at risk of this complication, which is often fatal. There is also a post Q fever syndrome of chronic fatigue. Q fever is the second most commonly reported laboratory infection with several recorded outbreaks involving 15 or more persons [47].

Status of Q Fever in Ethiopia

In Ethiopia, the existence of antibody against *Coxiella burnetii* was reported in goats and sheep slaughtered at Addis Ababa abattoir, and its peri-urban zone. A seroprevalence of 6.5% was also reported in Addis Ababa abattoir workers according to [48]. A seroprevalence of 31.6%, 90%, and 54.2% of *C. burnetii* was recorded in cattle, camels and goats respectively in South Eastern Ethiopian pastoral zones of the Somali and Oromia regional states as reported by Abebe [49]. Ticks were tested for *C. burnetii* in Ethiopia by quantitative real time polymerase chain reaction targeting two different genes followed by multispacer sequence typing (MST).

An overall prevalence of 6.4% of *C. burnetii* was recorded. *C. burnetii* was detected in 28.6% of *Amblyomma gemma*, 25% of *Rhipicephalus pulchellus*, 7.1% of *Hyalomma marginatum rufipes*, 3.2% of *Amblyomma variegatum*, 3.1% of *Amblyomma cohaerens*, 1.6% of *Rhipicephalus praetextatus*, and 0.6% of *Rhipicephalus (Boophilus) decoloratus*. Significantly higher overall frequencies of *C. burnetii* DNA were observed in *Amblyomma gemma* and *Rhipicephalus pulchellus* than in other tick species as reviewed by Tagesu.

Abortion is one of the most important reproductive health problems of dairy cows in Ethiopia in terms of economic impact. Both infectious and non-infectious agents may cause abortion in cattle. Q fever is one of infectious disease which cause abortion in Ethiopia [50].

Conclusion and Recommendation

Q fever is airborne disease caused by obligate intracellular bacteria of the genus *Coxiella*. It affects both human and animal in the worldwide except New Zealand. It is the second most commonly reported laboratory infection. It is a serious zoonotic disease which affects both human and animal. In Ethiopia it has been reported by causing abortion in dairy cattle and the existence of antibody in sheep and goat. The main source of infection is contaminated dust with birth fluid, faces, urine, and milk and tick. The reservoirs of human infections are farm animals such as cattle, goats, sheep, and Pets, including cats, rabbits, and dogs, have also been demonstrated to be potential sources of urban outbreaks of disease *C. burnetii*

is transmitted through inhalation and sometimes also transmitted through ingestion. *C. burnetii* is highly resistant to physical conditions and chemical agents, so it can survive for months and even years in the environment. Q fever is diagnosed by serological test. Tetracycline is recommended drug in the treatment of Q fever. It can be controlled by controlling tick by using insecticide. The prevention of Q fever is done by burning contaminated bedding and good hygiene practice.

Therefore, based on the above conclusion the following recommendations are forwarded:

- a) Public awareness should be created in consumption of dairy products
- b) Public awareness should be created in proper handling and disposing of aborted birth products.
- c) Government should wide and the availability and accessibility of effective diagnostic techniques.
- d) Government should vaccinate animals were found in endemic area.

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