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Annals of Virology and Research

Review Article

Chlamydia psittaci: An Omitted Pathogen at the Human-Animal Interface

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Submitted: 01 June 2018

Accepted: 27 July 2018

Published: 28 July 2018

ISSN: 2573-1122

OPEN ACCESS

Keywords
- *Psittacosis*
- Genotype
- Intracellular
- Immunity

Abstract

Chlamydia psittaci is an intracellular bacterium that causes respiratory disease in birds and other animals. It is important for public health concern because it has zoonotic potential and causes serious respiratory problems. In avian species, C. *Psittaci* infection causes pneumonia, diarrhea, poor growth and nervous symptoms. This bacterium is acquired from poultry and wild birds where it causes *psittacosis*, and after zoonotic transmission in human beings causes atypical pneumonia. In humans, the symptoms start from mild hyperthermia, chill, and headache, later on, these symptoms are changed to coughing and dyspnoea. The infection is curable at the initial stage but if not tackled at the proper time it may lead to myalgia and death in human. The infections due to *Chlamydia psittaci* are treated with tetracycline which is considered as the treatment of choice worldwide but so far there is no vaccine for the prevention of *Chlamydia psittaci*. The main genotype affecting the human is omp an indicating geno type A. There is a need to have a survey in Pakistan for patients suffering from *psittacosis* and respiratory distress. Humans, as well as animals, are at high risk of infection, so it is need of the hour to highlight the importance of C. *Psittaci* at the human-animal interface.

INTRODUCTION

In 1879, an unusual outbreak was reported by Ritter, who used to deal with pet birds and it was named as *Psittacosis*. The infected people showed flu-like symptoms, the organisms responsible for those infections were declared as Chlamydia by Morange in 1895 [1]. In the chlamydia phylum, Chlamydiaceae family is a well-studied group which includes 11 species as *C. trachomatis*, *C. suis*, *C. muridarum*, *C. pneumoniae*, *C. pecorum*, *C. psittaci* , *C. abortus*, *C. caviae*, *C. felis*, *C. avium* y *C. gallinacean,* that causes diseases humans as well as animals.

Chlamydia trachomatis and *C. pneumoniae* are primarily human pathogens while *C. pecorum* causes infection in ovines and bovines. *C. psittaci* has a wide host range; birds are frequently infected as compared to the mammals. While some of these bacterial species are host-specific, others like *C. psittaci* and *C. abortus* represent a potential zoonotic threat [2]. Avian *C. psittaci* has been classified into 15 geno types based on the *omp* a gene which encodes the major immunogenic protein of chlamydiae, more or less closely associated with certain bird species. Seven of these genotypes A-F, E/B are predominant whereas the other eight genotypes (IV, 6N, Mat116, R54, YP84, CPX0308, I, and J) were described as provisional. This systemic disease can take acute, protracted, chronic or subclinical courses and mainly affects psittacine birds and domestic poultry [3,4]. Chlamydia is an obligate intracellular growing pathogen which is Gram-negative in a sense because they have a cell wall which resembles that of Gram-negative bacteria. It divides by binary fission and does not have defined membrane between cytoplasm and genetic material (DNA). It produces extracellular elementary bodies (EB) and intracellular reticulate bodies of 0.2-0.3 µm and 0.6-0.8 µm in diameter respectively. It is not considered as a virus because of their dual nature of having both DNA and RNA and their mode of replication. Once it was considered as Rickettsia, but it is different from Rickettsia due to its inability to synthesize ATP and GTP along with the absence of the electron transport chain [5].

STRAIN CLASSIFICATION OF AND SEQUENCING OF *CHLAMYDIA PSITTACI*

Chlamydia pscittaci has 6 avian (A-F) and 2 mammalian genotypes (WC and M56). Serovar A is associated with psittacine birds, B is linked to pigeons, psittacine and turkeys, C genotype is found in duck and geese while the type D (virulent type) is associated with turkeys, egrets, and gulls. Genotype E has more diverse host range involving turkeys, ducks ratites, and pigeons and sporadically in humans as well. Genotype F has been isolated from turkeys and psittacine birds. The mammalian genotype WC is, isolated from Wolfsen Cattle and M56 from Muskrats. These genotypes are further classified on the basis of outer membrane protein a gene (ompA). This genotyping helps to further resolve the relationship of different genotypes. All of these known genotypes are host specific but can be transferred to human beings [7]. There is evidence of gene reduction, positive selection, and recombination [8]. It appears that speciation and strain emergence can be mediated by horizontal gene transfer or niche-specific genes that circulate among Chlamydiaceae [9,10].

The genome sequencing using an RD1 strain of mixed infection of *Chlamydia psittaci* and *Chlamydia trachomatis* by GS20 machine showed that *Chlamydia psittaci* have a genome of 1,156,417 bases in length and 959 coding sequences. The genome of *Chlamydia psittaci* is highly conserved with thirty-six tRNA and only one rRNA operon. This single rRNA operon shows high conservation of gene content that makes it different from other members of the genus. The outer membrane protein a (ompA) gene encodes the major outer membrane protein. This ompA fragment sequencing supports the classification of *C. psittaci* strains into 7 genotypes, named A-F and E/B and two new genotypes, named I and J as well [11].

In a micro-immunofluorescence assay, a major outer membrane protein mAb has been used to encounter the various isolates of unknown genotypes, which indicates the requirement of other mAbs to identify such isolates. Moreover, there are still various isolates which cannot be classified by the genotyping methods which are being used now [12].

The characterization of *Chlamydia psittaci* suggests that it has omp1 and mAB. The omp1 is the outer membrane gene obtained through PCR and it was characterized by micro immunofluorescence. While Alu1 restriction also been found that is very important for sequencing of this organism. The mapping of Chlamydial isolates reflects that Alu1 restriction has five known restriction pattern with a missing C, while serovar specific mAB showed A, B, C, D and not a single E was found in isolates under study. The restriction pattern by Alu1 was 98% similar to serovar-specific mAB but the serovar A showed the pattern of F. Due to some of these problems it was justified that genotyping is much better to perform rather than serotyping [14]. However, genetic markers that reflect different host or disease specificity have not been found in comparative genomic studies [15], nor have any robust virulence factors been identified [16].

PREVALENCE AND ZOONOTIC POTENTIAL OF *CHLAMYDIA PSITTACI*

The prevalence of *Chlamydia psittaci* and its associated zoonosis was calculated in different countries. In France, the Belgian chicken farms were under study, *Chlamydia psittaci* and atypical Chlamydiaceae were tested and none of the farms were positive for atypical Chlamydiaceae while 90% positive results were recorded for *Chlamydia psittaci*. Culture and PCR were performed for birds and for human in contact; ompA sequence revealed the presence of serotype A and D of *Chlamydia psittaci* in infected individuals. The people working on *Chlamydia psittaci* free farms never reported infection while 86% positive results for *psittacosis* were found in individuals working in diseased farms [17]. The prevalence of *Chlamydia psittaci* was also studied in Switzerland with special reference to *psittacosis* inhuman because of the potential risk of domestic and wild birds. Cormorants, song birds, waterfowl and feral pigeons were considered in the study. *Chlamydia psittaci* was isolated from cloacal swabs of birds and RT-PCR and micro array techniques were applied. The genotyping for *Chlamydia psittaci* was done and outer membrane protein A was found in all three categories except in cormorants while genotype B and E were present only in feral pigeons. The studies showed the data for the presence of *Chlamydia psittaci* in feral pigeons while the rest three were positive for an unclassified chlamydial agent. The feral pigeons were reported to be a continuous threat for the spread of *psittacosis* in human [18].

In Iran, a study was conducted for the prevalence of *psittacosis* in humans and chlamydiosis in birds, and pigeon feces were considered as a source of *Chlamydia psittaci*. For the strains study polymerase chain reaction was used and four hundred and forty-five samples were analyzed and DNA was isolated. Genotyping resulted in genotype B of *Chlamydia psittaci* in the feces of the feral pigeons. The results of the study were clear that *Chlamydia psittaci* has the highest prevalence in feral pigeons; they are the source of contamination and the spread of disease in humans and other animals. Vaccination and the use of antimicrobial drugs were suggested as a treatment regime [19].

The zoonotic potential of *Chlamydia psittaci* is published in a number of research articles and its exact prevalence is still uncertain. *Chlamydia psittaci* is best known zoonotic pathogen, the zoonotic potential of pathogen vary depending upon the region and hygienic practices [40]. *Chlamydia psittaci* is a zoonotic pathogen and causes serious disease in human beings as *psittacosis* pneumonia and a lot of many other infections. The family Chlamydiaceae has a great impact on the transmission of disease from animals to human beings. The animals are also suffering from *Chlamydia aborts* and *Chlamydia psittaci* infections. In chickens, *Chlamydia psittaci* outbreaks are reported and the disease is transmitted to human beings who are involved in any area with these birds [20]. The high zoonotic potential of *Chlamydia psittaci* was associated with genotype A in most of the cases which were ensured after ompA sequencing. The zoonotic impact of *Chlamydia psittaci* was observed by monitoring the interaction of people at turkey and poultry farms. All the genotypes of *Chlamydia psittaci* were considered as dangerous for the human, but genotype A was recorded with high zoonotic impact. A number of parameters including aerosol transmission, isolation, serological analysis and molecular detection showed the zoonotic impact of *Chlamydia psittaci* in people. The test results were confirmed by ompA sequencing for genotype A [21].

In Eurasian siskin, oriental skylarks and black-tailed grosbeaks in China. The antibodies against Chlamydia were detected using an indirect hemagglutination test. Sero positivity was found to be as; Eurasian siskins 6%, oriental skylarks 10% and black-tailed grosbeaks 13%. These results were statistically not significant but when the old birds were compared with younger ones the results were relatively different. The old birds were having a higher prevalence of *Chlamydia psittaci* than the juvenile birds. The study also showed that pet birds are the source of disease spread in the persons who are close to these birds [22].

HOST RANGE AND TRANSMISSION

Chlamydia psittaci is Gram-negative, coccoid, obligate intracellular bacteria that infect humans, wild and pet birds and a vast range of mammalian animals including cattle, sheep, and goat. *Chlamydia psittaci* is transferred between birds through dust particles, feathers, nutritional deficiencies and by contaminated food or water.

The overcrowding and egg laying may be a route of transmission as *Chlamydia psittaci* is present in feces. Once transmitted to birds it causes decreased egg laying, conjunctivitis, rhinitis, blepharitis and cystic oviduct in birds. It causes respiratory infections of zoonotic importance as it can be transferred to the human from pet animals and causes a number of the infections in human. The transfer of the pathogen to the human being may be through the respiratory route by inhalation in a contaminated environment or there may be some other routes as direct contact with feces, carcass, and respiratory fluids also may be through biting [6]. The human beings who are involved with chickens are at higher risk of infections because the infection is endemic in poultry. As, *Chlamydia psittaci* resides in the gut and are excreted through the faces of poultry birds, Oral transmission of this organism is not reported yet, but it can be transmitted through arthropod vectors, while the vertical transmission is limited [23]. In mammalians, the transmission is accompanied by exposure to body fluids and placenta of infected animals, but Human to human transmission is not yet reported [24]. The unique and distinctive features of the mammalian and avian zoonotic pathogen *C. psittaci* include the course of clinical disease, the high proportion of latent infection and wide host range that are not preceding to overt the diseases [25]. At cellular level, the broad spectrum of pathogen, its fast entry and rapid replication, pronounced association of chlamydial inclusions with cell compartments, proficient use of intracellular routes to Golgi apparatus and mitochondria as well as dissident regulation of host survival during persistent and productive states facilitates the characteristic growth and successful transmission from host to host of *C. psittaci* [26]

CLINICAL SYMPTOMS OF C. PSITTACI IN BIRDS AND HUMAN

The clinical symptoms of *C. psittaci* infection vary according to bacterial strain and severity of the infection. The incubation time of *C. psittaci* is from three days to several weeks, which is dependent on the health and nutritional status of the host. *C. psittaci* mostly infects nutrient deficient and immune suppressant animals. The clinical signs in case of birds begin with loss of appetite leading to a lethargic condition furthermore ruffled feathers, ocular and nasal discharge are associated with disease progression. The bird shows diarrhea with yellowish to greenish feces, which turn dark green with the severity of the infection. This diarrhea leads to the dehydrated and emaciated body which may lead to the death of the birds. The infected birds show high mortality rates of up to 30%. Clinical diseases are induced by stress, such as overcrowding, poor nutrition, or induced by another disease. Generally, Young birds are more susceptible than the mature birds. The susceptibility also depends on the bird species. In some cases, *C. psittaci* is excreted through feces for several months. Infective chlamydiae also excreted through lacrimal and nasal secretions [27].

In the case of human being, incubation time is up to fifteen days, the clinical signs appear with sudden hyperthermia, the patient shows a headache, and feels chills. Due to respiratory infection non- productive cough along with difficulty in breathing is initiated. The patients with infection of *C. psittaci* also exhibits malaise and myalgia, these all conditions were fatal in almost 20% of the patients until the use of antimicrobials against *C. psittaci* infections [28]. Furthermore, treatment with antibiotics might lead to subclinical persistent infections, possibly evolving to chronic disease and relapse when antibiotics are no longer administered [29]

DEVELOPMENTAL CYCLE

Once in the host cell, *Chlamydia psittaci* loses most of its energy producing substances but retains some of the metabolic mechanisms to produce energy, but its development inside the host depends upon ATP which is derived from various host components. *Chlamydia psittaci* uses host cell components for its DNA and RNA synthesis. In its development, the elementary body (EB) which is infectious and did not replicate is entered in the cell through endocytosis where it is converted into the reticulate body (RB) after 6-8hr. These reticulate bodies are non-infectious but can replicate and increase in number by binary fission after 10 to 18hr. This EB shows more adapted to the extracellular environment and can switch from one cell to another by evading immune system through an unknown mechanism. The proportion of DNA is four times more as compared to RNA in EB while it is the same in RB. *C. Psittaci* achieves a specified number of RB during the period of 18 to 24hr and then takes next 24hr to reorganize these RB into EB. The period of 48 to 72hr is a crucial period during which the cell bursts and it releases EB in the host body which causes severe infection and new cycle [30]. During this whole process, *C. psittaci* exhibits different functional and morphological changes [25].

HOST IMMUNE RESPONSE TO *C. PSITTACI*

The epithelial cells and macrophages are the site of replication of *Chlamydia psittaci*. The macrophages are part of the innate immune response that recruits myeloid cells to the site of infection by releasing cytokines. Since macrophages also serve as a bridge between innate and adaptive immune system they recruit CD4+ cells to the infection. However, *C. psittaci* manage to survive and replicate in these cells [41].The pathogen use mechanisms of protein (FtsW) synthesis that confer viability and protective immunity to the bacteria. It may block the fusion of phagosome and lysosome or block the production of host cell mediators [42]. The interferon (IFN-γ) provides a protective role through non-oxidative mechanism by altering certain metabolic mechanisms. The availability of machinery for multiplication of bacteria is restricted. However, the exact mechanism of action adopted by interferon (IFN-γ) is still unknown [43]. The growth of *C. Psittaci* can be retarded by lymphokines in murine macrophages under *in-vitro* conditions and reversed by addition of anti-interferon in culture media. IFN-γ initiated the microbicidal action of macrophages and suicidal activity in infected cells [31]. The indoleamine-2,3-dioxygenase (IDO) mediate the effect of IFN-γ on *C. psittaci* . IDO catabolizes tryptophan (Trp) and makes it unavailable for the persistence of the bacteria in the cells. The natural development can be halted at the intracellular stage through the action of adverse factors, such as interferon (IFN)-γ exposure, iron or amino acid depletion, and antibiotic treatment [32,33]

CO-INFECTIONS OF *CHLAMYDIA PSITTACI*

Chlamydia psittaci can cause co-infection with other

bacterial pathogens as it can evade the immune system; it involves up-regulation of certain genes which control membrane permeability. The expression of these genes is increased due to the increased expression rate of mRNA of Inca, ftsW, groW, groEL and cpaf and this help to establish the *Chlamydia psittaci* infections. The complement system is a central portion of the innate immune response. This system is activated by forty kinds of surface antigens of bacterial infection and produces an inflammatory response, but *Chlamydia psittaci* causes long-lasting inflammatory responses if the bird is deficient in C3aR because these C3a peptides are protective against *Chlamydia psittaci*. This organism is proficient in evading pro-inflammatory mediators [39]. In human, *C. psittaci* leads to atypical pneumonia. There are difficulties in the laboratory diagnosis and lack of relative knowledge about this disease by health professionals, so its prevalence is still unknown [34]. A case report also demonstrated co-infection with *C. pneumoniae and C. Psittaci* to be a hitherto unknown cause of fulminant myocarditis [35]. ORT and *C. psittaci* has been identified in Belgian turkeys [36]. During co-infection, both *C. psittaci* and *Aspergillus fumigates* induce suppression by the impairment of immune organs and Th1/Th2 imbalance, while the combination of H9N2 with ORT might play an exacerbating role in the respiratory disease by causing lung damage [37]. The co-infection of *C. psittaci* and H9N2 AIV induces a 35% mortality rate with severe pneumonia in SPF chickens [38]. *C. psittaci* suppress the immune system, more importantly, the adaptive immune system is compromised that may lead to increased co-infections. The increased mortality rate was reported in birds co-infected with *C. Psittaci* infection [44]. Previous studies reported the super infection of *E. Coli* and *C. psittaci* that the impact of *E.coli* infections [45].

PREVENTION AND CONTROL STRATEGIES

Screening on regular basis is very important for prevention of infection and birds which are newly purchased should be examined and kept in isolation for at least thirty days. The environmental disinfection can be made since *C. psittaci* is not resistant to quaternary ammonium compounds. Humans and birds can be prevented from the exposure of this serious zoonotic disease by maintaining the disease-free environment. Oral doxycycline is often recommended for the treatment of people who develop *psittacosis* [39]. The hygienic measures may restrict the spread of the infections. However, an effective vaccine or interferon-based immune therapy may be helpful in disease control.

CONCLUSIONS

Several studies disclose the fact that *C. psittaci* is a zoonotic pathogen that can infect birds, mammals, and human beings as well. There is a need to adopt effective control measures to prevent the spread of disease from animals to human *C. Psittaci* is also found to be associated in co-infection with most other pathogens. The host-pathogen interactions might be a key component in co-infection with other pathogens. The studies must be performed to evaluate the role of C. *psittaci* in the immune system that makes the birds and animals vulnerable to the pathogens allows the other pathogens to colonize and cause infections in. More importantly, the development of an effective vaccine as a preventive and the immune prophylactic measure for *C. Psittaci* infections are needed to control these infections.

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Cite this article

Naveed A, Abdullah S, Naveed R, Naveed MA (2018) Chlamydia psittaci: An Omitted Pathogen at the Human-Animal Interface. Ann Virol Res 4(1): 1033.