Concomitant *Chlamydia trachomatis* Pneumonia in a 40-day-old Infant with Incomplete Kawasaki Disease

YY ZHANG, YF HUANG, WJ XIANG, ZM CHEN

Abstract  
Kawasaki disease is an acute febrile illness of unknown aetiology that primarily affects children younger than 5 years of age. We present a 40-day-old male infant with incomplete Kawasaki disease complicated with coronary artery dilatations during *Chlamydia trachomatis* pneumoniae.

Key words  *Chlamydia trachomatis* pneumonia; coronary artery dilatation; Kawasaki disease

Introduction  
Kawasaki disease is an acute, febrile, multi-systemic vasculitis of unknown aetiology, first reported by Dr. Tomissaku Kawasaki. The diagnosis of Kawasaki disease requires the presence of fever for 5 days, 4 of 5 characteristic clinical features. It predominantly occurs in children aged 6 months to 5 years. The aetiology of it remains unknown despite extensive studies. Some researchers suggest that it is caused by an infectious agent. Here we described a 40-day-old male infant with incomplete Kawasaki disease complicated with coronary artery dilatations during *Chlamydia trachomatis* pneumonia.

Case Report  
A 40-day-old male infant was referred to our hospital in July 2012. There was history of irregular fever and cough for 8 days, rash for 4 days. He had already received wide spectrum antibiotics in another hospital prior to admission, but fever, rash and cough persisted. On admission the patient's body temperature was 38.4°C, pulse rate was 158 per minute, respiratory rate was 48 per minute. Physical examination revealed rough in breath sound, irritability and erythematous rash in the bilateral thigh and diaper area. The weight was 5000 grams. Blood pressure and growth parameters were normal.

Initial laboratory findings revealed white blood cell count of 11.8×10^9/L with a differential of 40.1% neutrophils, 44.8% lymphocytes and 8.3% monocytes. The platelet count was 335×10^9/L. Erythrocyte sedimentation rate was 89 mm/hr. Haemoglobin level was 103 g/L and C-reactive protein was 149 mg/L. On repeated blood tests white blood cell rose to 19.57×10^9/L with a differential of 60.1% neutrophils, 29.4% lymphocytes and 7.2% monocytes. Haemoglobin level was 92 g/L. The platelet count was 494×10^9/L. Erythrocyte sedimentation rate was 104 mm/hr. Haemoglobin level was 92 g/L and C-reactive protein was 124 mg/L. Serology for *Chlamydia trachomatis* was positive (IgM titer ≥1.1), and chest X-ray showed interstitial pneumonia. Cultures of blood, urine, sputum, cerebrospinal fluid for bacteria were negative. Sputum studies for respiratory viruses, polymerase chain reaction for enterovirus were negative. Serology for herpes simplex, cytomegalovirus, rubella virus and toxoplasm were all negative.

On day 2 (day of admission = day 0), periungual desquamation appeared. On the same day, the patient was assessed by echocardiography just on a clinical suspicion that the illness might have been Kawasaki disease.
Echocardiography revealed dilatations in the proximal segment of both left and right coronary arteries (left coronary artery 3.2 millimeters, right coronary artery 2.7 millimeters) (as shown in Figure 1).

A diagnosis of Kawasaki disease was made and the child was administered with intravenous γ-globulin infusion (1 g/kg/day) for two days. Aspirin was then given as 50 mg/kg/day for three days and was subsequently reduced to 5 mg/kg/d. Within 24 hours the patient became less irritable, the rash faded, and the temperature normalised. The response to the above treatment lent support to the diagnosis of Kawasaki disease. Meanwhile, the patient was administered with erythromycin (20 mg/kg/day) for treating Chlamydia trachomatis pneumonia. Eight days later white blood cell and C-reactive protein were normalised. There was marked symptomatic and clinical improvement. On day 14 the patient was discharged in excellent general condition on aspirin (5 mg/kg/d). After 1 month of treatment, Erythrocyte sedimentation rate was normalised, and the echocardiogram indicated that the left coronary dilatation had decreased to 2.6 millimeters and the right coronary dilatation had decreased to 1.8 millimeters. Aspirin was continued until resolution of coronary dilatation and normalisation of platelet count.

Five months after the diagnosis the patient were in stable condition and free of symptoms while receiving aspirin. The last echocardiography was normal – left coronary artery 1.6 millimeters and right coronary artery 1.5 millimeters. We summarised the clinical parameters of this patient during the illness in Table 1.

![Figure 1](image)

**Figure 1**  Echocardiographic pictures of this patient on day 2 after admission. (a) Left coronary artery; (b) Right coronary artery.

**Table 1**  The clinical parameters of this patient during the illness

<table>
<thead>
<tr>
<th>Date</th>
<th>Day 0</th>
<th>Day 1</th>
<th>Day 2</th>
<th>Day 3</th>
<th>Day 7</th>
<th>1 month after discharge</th>
<th>5 months after discharge</th>
</tr>
</thead>
<tbody>
<tr>
<td>Max temperature</td>
<td>38.4°C</td>
<td>38.2°C</td>
<td>38.1°C</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
</tr>
<tr>
<td>Skin rash</td>
<td>Positive</td>
<td>Positive</td>
<td>Positive</td>
<td>Fade</td>
<td>Negative</td>
<td>Negative</td>
<td>Negative</td>
</tr>
<tr>
<td>Desquamation</td>
<td>Negative</td>
<td>Negative</td>
<td>Appear</td>
<td>Positive</td>
<td>Positive</td>
<td>Negative</td>
<td>Negative</td>
</tr>
<tr>
<td>WBC (x10⁹/L)</td>
<td>11.8</td>
<td>19.5</td>
<td>–</td>
<td>9.13</td>
<td>8.62</td>
<td>7.24</td>
<td>6.88</td>
</tr>
<tr>
<td>Hb (g/L)</td>
<td>103</td>
<td>92</td>
<td>–</td>
<td>89</td>
<td>91</td>
<td>95</td>
<td>101</td>
</tr>
<tr>
<td>Plt (x10⁹/L)</td>
<td>335</td>
<td>444</td>
<td>–</td>
<td>872</td>
<td>859</td>
<td>621</td>
<td>388</td>
</tr>
<tr>
<td>CRP (mg/L)</td>
<td>149</td>
<td>124</td>
<td>–</td>
<td>53</td>
<td>&lt;1</td>
<td>&lt;1</td>
<td>&lt;1</td>
</tr>
<tr>
<td>ESR (mm/hr)</td>
<td>89</td>
<td>14</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>14</td>
<td>5</td>
</tr>
<tr>
<td>Echocardiography</td>
<td>–</td>
<td>LCA:3.2 mm</td>
<td>–</td>
<td>LCA:2.8 mm</td>
<td>LCA:2.8 mm</td>
<td>LCA:1.6 mm</td>
<td>LCA:1.6 mm</td>
</tr>
<tr>
<td>RCA:2.8 mm</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>14</td>
<td>5</td>
</tr>
</tbody>
</table>

Day 0: admission day; WBC: white blood cell; Hb: haemoglobin; Plt: platelet; CRP: C-reactive protein; ESR: erythrocyte sedimentation rate; LCA: left coronary artery; RCA: right coronary artery; "–" means not detected; IVIG: intravenous immunoglobulin
Discussion

Kawasaki disease is an acute necrotising vasculitis of the medium and small-sized vessels, predominantly occurring in children aged 6 months-5 years and rare among infants younger than 3 months. Complete Kawasaki disease was diagnosed with fever ≥5 days and at least four of the five principal clinical features: polymorphous rash, non-purulent conjunctivitis, cervical lymph node enlargement, changes of the extremities and changes in the oral mucosa. Patients with coronary artery involvement but less than four criteria were labeled as incomplete Kawasaki disease when other diseases were excluded. Such patients are usually at extreme of ages and are at more risk for developing coronary artery disease. Though this patient was only a 40-day-old infant, because of his prolonged history of unexplained fever, rash and periungual desquamation, incomplete Kawasaki disease was considered. The patient only presented with two of the five principal diagnostic criteria along with prolonged fever characteristic of complete Kawasaki disease. The diagnosis was confirmed by the echocardiogram findings of coronary artery dilatation. However, Muniz et al found that children with non-Kawasaki disease febrile illnesses could have coronary artery dilatation, but have lower white blood cell count, erythrocyte sedimentation rate, and platelet count. So when clinicians encounter a patient with unexplained fever, Kawasaki disease should be suspected regardless of the age, and white blood cell, Erythrocyte sedimentation rate, C-reactive protein, echocardiogram should be done at the same time.

Despite more than 40 years of active research, the etiologic agent of Kawasaki disease remains unknown. Recently, multiples studies have evaluated the role of various infectious pathogens as potential agents for it. Recently specific infections are being reported in Kawasaki disease patients such as Yersinia pseudotuberculosis, streptococcus, Adenovirus, Mycoplasma pneumonia, and etc. To the best knowledge of the writers, this is the first case report of Chlamydia trachomatis infection, concomitant with incomplete Kawasaki disease. In the present case, the association of Chlamydia trachomatis detection with high specific IgM titer was consistent with the diagnosis of Chlamydia trachomatis infection. Moreover, the patient was aged 40 days and X-ray showed interstitial pneumonia. The age, presenting clinical manifestation and features of X-ray were consistent with Chlamydia trachomatis infection. We hypothesised that the Chlamydia trachomatis infection perhaps was involved in the pathogenesis of Kawasaki disease and coronary dilatation. However, since Chlamydia trachomatis infection is relative common in infants below 3 months and hence a causal link of both illnesses is not possible. And Strigl et al found that there were no significant differences in the prevalence of anti-chlamydial IgG, IgM, and IgA between children with Kawasaki disease and that of asymptomatic controls. In this patient, we only found the positive results of anti-chlamydial antibodies, and have not detected the Chlamydia trachomatis DNA in the blood, which could not verify an association between Chlamydia trachomatis and Kawasaki disease. To elucidate a true association between pathogen and Kawasaki disease, direct detection of the organism in tissue from patients with Kawasaki disease should be done in the future.

In conclusion, this case illustrated the fact that incomplete Kawasaki disease is often a late consideration, especially when the symptoms of the classical form are absent. Because young infants with Kawasaki disease are an extremely high risk of developing coronary arterial abnormalities, early diagnosis and appropriate therapy are particularly important. Kawasaki disease patients may have concurrent infections especially with pulmonary involvement. It is important to warrant future prospective studies to looking for additional pathogens for the Kawasaki disease.

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Conflicts of Interest

None
References


