Mycoplasma pneumoniae induced popliteal artery thrombosis treated with urokinase

C U Joo, J S Kim, Y M Han

Abstract
A 5 year old boy with serological and clinical evidence of Mycoplasma pneumoniae infection, which was complicated by popliteal artery thrombosis, is described. Intra-arterial urokinase, in conjunction with medical treatment, resulted in clinical recovery and angiographic resolution of the thrombus. The variety of extrapulmonary complications associated with the M pneumoniae infections continues to broaden. Thrombolytic therapies should be considered when similar clinical circumstances arise.

Keywords: Mycoplasma pneumoniae, arterial thrombosis; urokinase

Mycoplasma pneumoniae is a common cause of respiratory disease in children and adults. The incidence of infection peaks among individuals 10 years of age, with the disease being less common in those under 5 years and over 40 years of age. In general, mycoplasma disease is mild and self limited, although infections may occasionally be associated with a variety of extrapulmonary conditions, although severe complications are rare. Popliteal artery thrombosis is an extremely rare complication of the infection. We report a case of popliteal artery thrombosis in a child with clinical and serological evidence of M pneumoniae infection and the clinical course of recovery after intra-arterial infusion of urokinase.

Case report
A previously well 5 year old Korean boy was admitted to a local hospital with a 10 day history of intermittent fever, anorexia, and non-productive cough with a presumptive diagnosis of M pneumoniae pneumonia. He was transferred to the Chonbuk National University Hospital because of acute onset of right leg pain accompanied by pallor and coldness. He had been on azithromycin (10 mg/kg/day for three days). An examination at the time of the transfer revealed a febrile child with a diminished breath sound over the right upper chest and a cool and pale right leg. The popliteal and pedal pulses in the right leg were not palpable. Neurological examination was normal. On admission, a chest x ray film showed an ill defined consolidation in the right upper lobe, compatible with M pneumoniae pneumonia. The haemoglobin concentration was 113 g/l and white blood cell count was 14.8 × 10^9/l. The differential count was neutrophils 72% and lymphocytes 17%. The platelet count was 480 × 10^9/l.

Because of the perceived urgency of the situation, an emergency angiogram was obtained. The catheter was introduced through the left femoral artery and advanced into the aortic bifurcation where an injection of the contrast media (Visipaque 15 ml; Nycomed Ireland Ltd, Cork, Ireland) was made. The angiogram showed non-opacification of the right popliteal artery and its distal branches (fig 1). The catheter was placed in the aortic bifurcation and a 100 000 units of urokinase infusion was done for one hour. A repeat angiogram at this time showed a marked improvement in the filling of the distal arteries. Thus, the catheter and sheath were removed and haemostasis accomplished. Urokinase was administered systemically at a dose of 70 000 units/hour for the next 12 hours. After the infusion of urokinase, the coldness of the leg gradually improved, and the dorsalis pedis pulse became palpatble.

At the time of discharge on the 12th hospital day, all the signs of arterial occlusion were resolved. Follow up magnetic resonance angiogram showed normal visualisation of the both popliteal arteries, and the anterior tibial, posterior tibial, and peroneal arteries (fig 2). We checked protein C, protein S, and other coagulation factors to study the cause of thrombosis. The coagulation profile revealed the following:

Figure 1 Lower extremity angiogram showing abrupt cutting of the right popliteal artery and non-visualisation of the anterior tibial, posterior tibial and peroneal arteries, which are due to thrombi. There are no-filling contrasts in left posterior tibial and peroneal arteries.
free protein S 63% (normal range 60%–90%),
total protein S 79% (70%–140%), protein C
81% (73%–142%), antithrombin III 22.6
mg/dl (20–28), fibrinogen 2.9 g/l (2.0–4.0),
and prothrombin/activated partial thrombo-
plastin time normal. Acute
M pneumoniae
infection was confirmed with a positive IgM
and a subsequent rise in the antibody titre from
1:32 to 1:256.

Discussion
This is a case of popliteal artery thrombosis
associated with M pneumoniae infection, which
was successfully treated with intra-arterial
urokinase infusion. To the best of our knowl-
dge, this is the first report in English
literature: there has been one similar report in
the French literature.1

M pneumoniae is known to cause a mild dis-
ease of the upper respiratory tract and
pneumonia, with a peak incidence between 5
and 25 years of age. Although serious pulmo-
mary and extrapulmonary complications are
known to occur rarely, there have been an
increasing number of reports of such complica-
tions. The extrapulmonary manifestations of
the M pneumoniae infection are diverse and
involve nearly every organ system. Complica-
tions reported to date include neurological,
haematological, renal, dermatological, per-
imyocardial, gastrointestinal, and musculo-
skeletal dysfunction. The complications de-
scribed have occurred one to 23 days after the
onset of respiratory tract symptoms.2

Figure 2 Follow up magnetic resonance angiogram
showing normal visualisation of the both lower extremity
artery.

Despite the numerous reports detailing the
extrapulmonary manifestations associated with
M pneumoniae infection, the exact mechanism
by which the extrapulmonary injury occurs has
not yet been established. Possible mechanisms
include direct invasion by the organism, toxin
production, autoantibody and immune com-
plex formation, microthrombosis, and im-
paired immunity.2

Although intravascular coagulation disor-
ders have been reported during the myco-
plasma infection in a few cases, the pathogenic
mechanisms are obscure. In in vitro experi-
mental studies, Fumarola suggested that
lipoglycans from some mycoplasma species,
including M pneumoniae, could induce proco-
agulant activity (tissue factor-like ability) by
human mononuclear cells.3 The potent trigger-
ing coagulation activity of the lipoglycans may
explain the mechanism of the intravascular
coagulation associated the mycoplasma infec-
tion. Other factors including antithrombin III
deficiency (congenital or acquired), deficiency
of protein C or S, or both, should be
investigated to identify possible contributors to
the pathogenic mechanisms of the thrombosis
associated with the disease.4,5

Summary/learning points

- M pneumoniae is known to cause respira-
tory infections including pneumonia and
extrapulmonary complications.
- The exact mechanisms of extrapulmon-
ary manifestations of the infection are
obscure. The development of autoanti-
bodies and immune complex during the
course of the infection has been sug-
gested. Lipoglycans formed by the organ-
isms may be responsible for intravascular
thrombosis.
- Popliteal artery thrombosis is an ex-
remely rare extrapulmonary manifesta-
tion of the M pneumoniae infection.
- Early intra-arterial urokinase infusion is
effective in the treatment of the thrombo-
sis.

We thank Dr Myung K Park, Department of Pediatrics, the
University of Texas Health Science Center at San Antonio,
Texas, for his assistance with this manuscript.

1 Ducloux G, Defaux D, Folliot JP, et al. [Femoral artery
thrombosis associated with Mycoplasma pneumoniae infec-
2 Moskal MJ. Mycoplasmal infections. Pulmonary and extra-
3 Fumarola D. Intravascular coagulation and Mycoplasma
4 Creagh MD, Roberts IF, Clark DJ, et al. Familial
antithrombin III deficiency (congenital or acquired), deficiency
of protein C or S, or both, should be
investigated to identify possible contributors to
the pathogenic mechanisms of the thrombosis
associated with the disease.4,5

5 Peyton BD, Cutler BS, Stewart FM. Spontaneous tibial
artery thrombosis associated with varicella pneumonia and

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Postgrad Med J 2001 77: 723-724
doi: 10.1136/pmj.77.913.723

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