

Guillain-Barré Syndrome as A Rare Complication of Mycoplasma Infection in A Child

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Abstract

Mycoplasma pneumoniae is a common cause of community-acquired pneumonia (CAP) however it can rarely cause Guillain-Barre syndrome (GBS) which is a serious acute inflammatory demyelinating polyneuropathy. We present an interesting case of a 2 years and 8 months' old boy who was seen for limping and was diagnosed with transient synovitis of the hip. He also had mild cough, transient erythematous rash with centripetal spread and decreased appetite. Initially he was prescribed a course of Clarithromycin to cover possible mycoplasma infection but only managed a few doses as he did not tolerate it. He developed progressive hypotonia, reduced muscle power and hyporeflexia in both legs. His laboratory investigations revealed him to be positive for mycoplasma IgM which was an evidence of current or recent mycoplasma infection. He was treated with repeat course of IV Clarithromycin and intravenous immunoglobulins for Guillain-Barré syndrome and had a slow but successful recovery.

Although Guillain-Barré is a rare complication of Mycoplasma infection in children, it should be considered in the differential diagnosis of any prolonged atraumatic limp in children. Increased awareness of this presentation among paediatricians, primary care clinicians and orthopaedic surgeons is required.

Keywords: Guillain-Barré Syndrome; Mycoplasma Infection; Transient Synovitis of the Hip

Introduction

Mycoplasma pneumoniae is a common cause of community-acquired pneumonia (CAP), and the illness usually has a prolonged, gradual onset. Mycoplasma organisms are usually resistant to penicillins and cephalosporins. Initially noticed as acute areflexic paralysis in soldiers with prompt recovery; Guillain-Barre syndrome is a rare and serious condition of the peripheral nervous system. One theory is that infection may elicit an immune response that cross-reacts with Schwann cell antigens resulting in damaging the peripheral nerves. The diagnosis is by finding raised concentration of cerebrospinal fluid (CSF) protein with a normal cell count along with acute areflexic paralysis.

Case Presentation

We present an interesting case of a 2 years and 8 months' old male child When he was referred to the paediatric ward at a District General Hospital. Although he was previously independently mobile but for nearly a week before presentation he was non-weight bearing on legs. There was no history of any prior systemic symptoms or trauma. He was diagnosed with atraumatic limp due to irritable hip (transient synovitis of the hip).

His initial haematological investigations showed normal CRP and ESR (inflammatory markers; C - reactive protein and Erythrocyte Sedimentation Rate) but slightly raised white cell. He was managed with simple analgesia in the form of paracetamol and Ibuprofen.

He also had mild cough, transient erythematous rash with centripetal spread and decreased appetite. Parents did not report any history suggestive of morning headaches. His past medical and family history was unremarkable. His development was age appropriate. He was prescribed a course of Clarithromycin to cover possible mycoplasma infection but only managed a few doses as he did not tolerate it.

His clinical examination including cardiovascular, ENT (Ear, Nose and Throat), respiratory and abdominal systems were unremarkable along with normal GCS (Glasgow Coma Scale). His neurological examination revealed progressive hypotonia, reduced muscle power and hyporeflexia in both legs. Neurological examination of upper limbs, cerebellar function tests and cranial nerve examination were unremarkable. There was no obvious nystagmus, ataxia or squint. His laboratory investigations revealed him to be positive for mycoplasma IgM which was an evidence of current or recent mycoplasma infection (Table 1).

Investigation:	Results/report
FBC, ESR, CRP, U&Es and LFTs, C3, C4, Gamma globulins, Creatine Kinase	Within normal limits
X-ray Pelvis report:	Normal bone and joint appearances.
Blood film	No diagnostic features on the blood film
Urinary catecholamine	Within normal limits
EBV, CMV, Enterovirus and Parvovirus serology	Negative
Viral titres, Autoimmune profile, Rheumatoid factor and ASOT	No abnormality detected
Mycoplasma IgM	Positive as an evidence of current or recent mycoplasma infection
USS abdomen and hips	Unremarkable with no evidence of joint effusion.
MRI scan head and Spinal cord (Pre and post gadolinium)	Imaging of the brain and cord showed no abnormality. The cauda equina nerve roots were thickened and showed avid enhancement on MRI.
CSF protein	Raised at 2.23 gram/Litre (0- 0.4)
CSF glucose	2.4 mmol/litre (2.2-4.4)
CSF cell count	WBC 10/mm ³ RBC 1430/mm ³
CSF cultures	No growth
CSF oligoclonal bands	No abnormality of the CSF IgG pattern detected.

Table 1: Investigation Table.

Our working diagnosis was post viral/mycoplasma synovitis but the course of illness was more prolonged than one would expect. He was referred to paediatric oncology for further investigations about oncological aetiology e.g. neuroblastoma or leukaemia. A neurological cause was also considered as a reason for his presentation.

He was diagnosed with Guillain-Barré syndrome after he had an MRI (Magnetic Resonance Imaging) scan of head/spine showing thickening and enhancement of cauda equina nerve roots (Figure 1). His lumbar puncture confirmed cyto-albuminologic dissociation which has high diagnostic value for Guillain-Barré syndrome. The CSF protein was raised at 2.23 gram/Litre with normal CSF cell count.

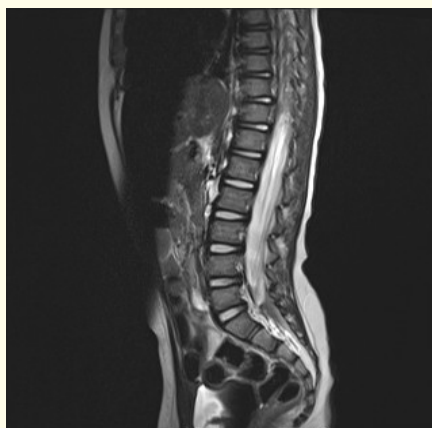


Figure 1: This MRI is showing avid enhancement of cauda equina nerve roots due to thickening.

He was treated with intravenous immunoglobulin and received a repeat course of IV Clarithromycin along with intensive inpatient physiotherapy. He was discharged home after making progress. Our patient made very good and steady progress and was walking again after about 5 months of initial diagnosis. He continues to have hyporeflexia in both legs.

Discussion

Mycoplasmas are the smallest free living micro-organisms. *Mycoplasma pneumoniae* is a common cause of community-acquired pneumonia (CAP), and the disease usually has a prolonged, gradual onset. Mycoplasma organisms are usually resistant to penicillins and cephalosporins [1]. *Mycoplasma pneumoniae* has been reported as the most common cause of lower respiratory tract infection in young adults and children [2]. *Mycoplasma pneumoniae* account for 5% of GBS cases [3].

Initially noticed as acute areflexic paralyses in soldiers with prompt recovery; Guillain-Barré syndrome is a rare and serious condition of the peripheral nervous system. The exact cause of Guillain-Barré syndrome is unknown. In most patients, it develops two to four weeks after a minor viral or bacterial infection. One theory is that infection may elicit an immune response that cross-reacts with Schwann cell antigens because of molecular mimicry [4] resulting in damaging the peripheral nerves [5]. Mycoplasma infection along with EBV have been reported with GBS in past [3,6].

The diagnosis is by finding raised concentration of cerebrospinal fluid (CSF) protein with a normal cell count along with acute areflexic paralysis [4]. Male are 1.5 times more likely to be affected and the incidence is relatively uniform between 0.6 and four cases per 100 000 per year throughout the world but less than one per 100 000 in patients younger than 30 years [5]. Mycoplasma pneumoniae infections can result in neurological complications in one in fifteen children and may present in unusual ways [7].

Conclusion

Although Guillain-Barré is a rare complication of Mycoplasma infection in children, it should be considered in the differential diagnosis of any prolonged atraumatic limp in children [3]. Increased awareness of this presentation among paediatricians, primary care clinicians and orthopaedic surgeons is required.

Consent

Parental consent was obtained for this publication.

Declaration of Conflicting Interests

None declared.

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