Pneumonia and Sepsis Caused by Gemella Morbillorum: an Unusual Association

Giuseppe Famularo¹, Claudio De Simone², Giovanni Minisola¹ and Giulio Cesare Nicotra¹

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An 83-year-old man presented with the sudden onset of fever (40.3°C), confusion and lethargy. His past medical history included a monoclonal gammopathy of uncertain significance (MGUS), hypertension, and diabetes that were well controlled with glibenclamide and ramipril; there was no history of alcohol use, toxic habits or taking any other medications, including over-the-counter medications, or herbal remedies. On admission, the patient appeared unwell and lethargic; we found a poor dental state, dehydration, tachycardia (140 bpm), tachypnea (28 breaths/min), and blood pressure 110/80 mmHg. The remaining physical examination revealed a systolic bruit along the left sternal border with no bruits heard around the neck, dullness to percussion, and decreased breath sounds with rales and wheezing over the right lower lung; no other abnormalities were noted. On neurological examination, the patient was lethargic but no focal weakness or hemisensory change was recognized; the motor function was graded at 3/5 throughout and deep-tendon reflexes were ++ bilaterally with flexor plantar responses.

A laboratory work-up disclosed glucose 402 mg/dl (normal range 60-110), sodium 150 mg/dL (normal range 135-145), chloride 113 mg/dL (normal range 100-112), leukocytes 11.4×10⁹ cells/L; amylase, lipase, coagulation, and liver and renal function tests were normal. Arterial blood gas analysis on room air revealed moderate hypoxemia with a PaO₂ of 70 mmHg and an oxygen saturation of 86.6% SaO₂, pH 7.43 and PaCO₂ 55.1 mmHg. An electrocardiogram was normal and a chest X-ray showed a right-sided consolidation with a moderate pleural effusion.

After blood was taken for culture, the patient was started on intravenous fluids, oxygen, regular insulin, theophylline, inhaled budesonide, levofloxacin (500 mg i.v. daily), cefotaxime (2 g i.v. thrice daily), enoxaparin (50 IU/kg daily), and on the 3rd day of hospital stay he was stable, alert and oriented but still pyrexial; at this time glucose, electrolytes, liver and renal function tests, and hematologic variables were all within the normal range. On the 6th day, pyrexia settled and Gemella morbillorum, which was fully sensitive to cefotaxime, levofloxacin, cloramphenicol, penicillin, teicoplanin, and vancomycin and resistant to erythromycin, was grown from blood cultures. G. morbillorum was identified based upon its phenotypic characteristics by conventional morphologic and physiologic tests. We observed Gram-positive coccal bacteria in pairs and short strains of variable sizes on Gram stain, that showed no easy decolorization on the Gram staining procedure and a positive pyrrolidonyl arylamidase reaction; the isolated strain was catalase-negative and leucine aminopeptidase-positive. Additional biochemical investigation was performed by the API rapid ID 20 strep (BioMerieux, Italy), which was read automatically after 24 hours by using an ATB Expression instrument and ATB Plus software (BioMerieux). Both the assay strips and the reading equipment were used according to the manufacturer’s instructions and G. morbillorum was identified with a 84% confidence. Microbiologic studies of respiratory samples were not done because the patient was unable to produce sputum and we decided to not take samples of tracheal aspirates because the patient rapidly achieved clinical stability and at no time during his hospital stay there was indication to tracheal intubation and mechanical respiration. We also decided to not perform a thoracentesis for microbiologic examination because of the small amount of the pleural effusion and also because of the prompt clinical response to antibiotics. We performed a trans-thoracic echocardiography, which disclosed no vegetations, and continued antibiotics; the patient had a full recovery and was discharged free of symptoms on the 16th day. At a follow-up visit one month later, he was doing well with no clinical or laboratory evidence of sepsis or pneumonia and a repeated chest X-ray showed a complete clearance of the right-sided consolidation and pleural effusion.

Gemella morbillorum, formerly Streptococcus morbillorum, is a catalase-negative, facultatively anaerobic Gram-positive coccus that was first described in 1917 by Tunnicliff (1). The bacterium was transferred into the genus Gemella in 1988 based on DNA homology, physiological properties, and 16S RNA cataloguing (2). It forms part of the commensal endogenous flora of the oropharynx, gastrointestinal tract, and genitourinary tract and is a relatively infrequent cause of infection in humans. G. morbillorum has been most commonly associated with infectious endocarditis.
but also a few cases of meningitis, pericarditis, arthritis, osteomyelitis, abscesses, mediastinitis, peritonitis, spondylodiscitis, and sepsis have been reported (3). Our Medline search yielded no more than eight patients with respiratory infections caused by the organism which were complicated in most of them by a lung abscess or a pleural empyema (reviewed in 4). Aspiration is the most important mechanism claimed to explain the spreading of \textit{G. morbillorum} to the respiratory tract particularly among patients with esophageal stricture and dysphagia or those who suffered a laryngectomy. A poor dental state along with poor oral hygiene could favor access to the bloodstream and the occurrence of bacteremia with \textit{G. morbillorum} and this risk is greatly increased after dental procedures. Even endoscopic procedures or disruption of the epithelial barrier of the digestive tract due to inflammation, ulcer, and cancer may favor the translocation of the organism to the bloodstream (5). Our patient had pneumonia and the sepsis syndrome with positive blood cultures, however it remained unclear whether the active \textit{G. morbillorum} infection was the result of aspiration rather than of hematogenous spread. As a matter of fact, our patient had none of the above mentioned risk factors for the spreading of \textit{G. morbillorum} or any other bacteria of the oro-pharyngeal flora to the respiratory tract through aspiration, which could indirectly point out to dissemination through the bloodstream as the main mechanism of lung involvement in this case.

The patient presented with severe clinical features nonetheless outcome was ultimately favorable and there was a rapid and full recovery with appropriate antibiotic treatment. This is in agreement with current data from the literature which indicate that patients with pneumonia caused by \textit{G. morbillorum} usually have a good course, even in the presence of pleural effusion or bacteremia, if appropriate and aggressive antibiotic and support therapy is initiated as early as possible (4). There is some evidence that resistance of \textit{G. morbillorum} to penicillins may be relatively prevalent, which suggests that the initial empirical therapy of the suspected case should include the combination of a $\beta$-lactam agent and an aminoglycoside or, alternatively, vancomycin (4). In the present case we achieved a full clinical and microbiological response with combination therapy with a $\beta$-lactam agent and a quinolone. We cannot rule out that the pleural effusion in our patient had the features of empyema but a thoracentesis was not performed mainly because of the prompt clinical response to antibiotic treatment. However, in our opinion this observation represents an indirect evidence against the hypothesis of an empyema, as in this latter setting antibiotics alone, even if given for a prolonged course, are usually not adequate to achieve the complete disappearance of the effusion and pleural drainage is ultimately required in most instances.

The true prevalence of pneumonia or sepsis caused by \textit{G. morbillorum} is unknown, however it is probably underestimated as blood or sputum cultures are not routinely performed in all patients admitted with pneumonia. Infection with this organism should be suspected in selected subgroups of patients, particularly those at greater risk of aspiration, those with a poor dental state, and those presenting with pneumonia or fever after dental or endoscopic procedures.

### References


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