were identified as risk factors for this phenomenon.[5] Both our patients had a high percentage of BM blasts without karyotypic aberrations, but received higher G-CSF doses and demonstrated earlier progression. Although we cannot provide objective proof of a causal relationship between G-CSF and MDS acceleration, the clinical course of our cases is highly suggestive of *in vivo* priming of leukemic cell proliferation. We believe stricter G-CSF administration criteria are warranted in the heterogeneous group of high-risk MDS and advocate that this agent be used only in life-threatening infections under close monitoring.

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Chlamydia pneumoniae pneumonia with acute hemorrhagic pericarditis in patient with acute leukemia

Sir,

Chlamydia pneumoniae, a recently discovered pathogen, is mainly responsible for respiratory tract infections. It has also been associated with endocarditis and myocarditis,[1,2] but there are no reports implicating *C. pneumoniae* in pericarditis. We present a patient with pneumonia and acute hemorrhagic pericarditis caused by *C. pneumoniae*.

A 27-year-old man was diagnosed with acute myeloblastic leukemia. The first cycle of chemotherapy according to the MRC 10 protocol resulted in complete remission. On day 2 following completion of the third cycle of chemotherapy (amsacrine, cytosine arabinoside, etoposide) he developed pancytopenia and his temperature rose above 39°C. Empirical therapy with amikacin and cefoperazone was initially successful. On day 20, a temperature of 39°C reappeared together with a dry, irritating cough. Bacterial cultures revealed methicillin-resistant *Staphylococcus aureus* and *Candida krusei*. Vancomycin and amphotericin B were started. Three days later X-rays chest, which had previously been normal, revealed bronchopneumonia of the right middle lobe. On day 27, the patient developed pericardial friction rub and pleural pain. Temperature had decreased to 37-38°C. Echocardiographic examination, which had been normal at admission, revealed pericardial effusion measuring up to 27 mm in depth. Repeated echocardiography showed fibrous strands attached to the pericardium. Because the patient's condition was progressively deteriorating, erythromycin in doses of 0.5 g q6h IV was instituted on day 34. Blood tests indicated bone marrow regeneration. On day 40, the patient developed cardiac tamponade. Pericardiocentesis yielded 320 mL of sanguinous exudate, bacterial and fungal cultures of which were negative. Serologic tests for cardiotropic viruses, Epstein-Barr virus, cytomegalovirus, *Mycoplasma pneumoniae*, *Legionella pneumoniae*, fungal antigens and polymerase chain reaction to *Mycobacterium tuberculosis* were all negative. The pericardial fluid was also analyzed with direct staining for *C. pneumoniae* and cultured.[3] A direct immunofluorescence test using *C. pneumoniae*-specific monoclonal antibodies (Cellabs, Australia) was positive for chlamydial elementary bodies. Subsequent cultures of the pericardial effusion also contained *C. pneumoniae* elementary bodies. The IgG titre to *C. pneumoniae* determined with a microimmunofluorescence assay was 1:64, while no IgM were apparent. Two weeks after pericardiocentesis and initiation of erythromycin, echocardiogram and chest X-rays normalized. The patient was discharged after remaining good physical condition for two weeks, until his next cycle of chemotherapy. Unfortunately, he died several months later during first relapse of acute leukemia of an intracerebral hemorrhage.

In conclusion, *Chlamydia pneumoniae* should be suspected in patients with pneumonia and concurrent pericarditis, especially in those who are immunocompromized. Empirical therapy with erythromycin may be beneficial.

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