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# Hemophagocytic syndrome in the critically ill

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Sir: Our interest was captured by the letter of Rokyta and al. [1] and we would like to report a similar case with an uncommon infectious cause.

A 50-year-old man was admitted to our hospital because of obnubilation. Two months before, he had been treated with chemotherapy in our institution for neoplasia of left amygdala. Neurological clinical examination was normal. Head CT scan and analysis of cerebrospinal fluid were noncontributory. There were no signs of progression of underlying neoplasm.

Three days after admission he developed dyspnea, hypoxemia, and fever. Chest radiography showed bilateral lower pulmonary infiltrates. Third-generation cephalosporin and ofloxacin were introduced. Fifty hours later, the patient was in septic shock and was admitted to the intensive care unit.

An intratracheal tube was rapidly placed, catecholamine was introduced, and erythromycin was added to the previous antibiotherapy. Five days later, thrombopenia occurred (16,000/mm$^3$) with leukopenia and anemia. Analysis of bone marrow aspirate showed a large quantity of hemophagocytosis. Corticotherapy was introduced with a good response in thrombopenia and leukopenia in the two following days. Tracheal aspiration culture, blood culture, and urine culture remained sterile, and two serologies of *Legionella pneumoniae* (LP) were positive at 1/1,280. Despite etiological therapy with erythromycin, multiorgan failure occurred with renal failure, untreatable shock, and ARDS. The patient died 11 days after ICU admission.

Hemophagocytic syndrome (HS) remains a difficult diagnosis in ICU with high mortality. Treatment and research into etiology are other difficult areas to manage in this pathology. Infectious diseases are the leading cause of HS [2]. In our case report, LP was the only etiologic agent found. This bacteria is not known to be commonly associated with HS. To our knowledge, this is the first case reported. Hemophagocytic syndrome is certainly a poor prognosis factor of LP. Treatment of the underlying disease is fundamental but not sufficient. As outlined by Rokyta and al. [1], the management of this cytokinic storm is based not only on high-dose steroids, plasma exchange, and cyclosporin A, but also on VP-16 [3].

## References

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