neoplastic pericarditis is not easily resolved. The data available in literature are still scarce today and survival of these patients continues to be very limited.

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Pneumocystis Pneumonia in a Patient treated with Fludarabine for Chronic Lymphocytic Leukaemia

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FLUDARABINE (9-B-D-arabinofuranosyl-2-fluoradenine) is currently proposed for the treatment of refractory chronic lymphocytic leukaemia (CLL) [1]. In previously treated patients, response rates reached 50–60% for Rai stages III and IV patients [1]. Complete remissions have been maintained in some patients, but the follow-up is still too short [1, 2]. The major toxicity is myelosuppression and severe infections have been reported, especially pneumonia. Pulmonary toxicity has also been noted but interstitial pneumonitis has generally regressed after discontinuation of the drug [2, 3]. A new case has been recently reported, with negative microbiological studies and recurrence after fludarabine had been resumed, in a patient with prolymphocytic leukaemia [4]. However, clinical and radiological features seen in this particular situation can correspond to non-unequivocal mechanisms. We report here a case of pneumocystis pneumonia survening after a single course of fludarabine. This patient recovered and then relapsed after chemotherapy had been resumed.

A 65-year-old woman with Rai stage IV CLL diagnosed in 1981 and refractory to chlorambucil was referred to our centre for treatment with fludarabine (25 mg/m^2 for 5 days each month). 2 weeks after the first course, she developed fever with bilateral pulmonary infiltrates. Fibroscopy with aspiration revealed *Pneumocystis carinii* pneumonia (Gomori silver stain positive). With high-dose cotrimoxazole therapy (100 mg/kg/day sulphamethoxazole for 3 weeks), the patient recovered. As the patient had responded to this first course with a decrease of 50% of peripheral adenopathies and improvement in peripheral blood count (lymphocytosis decreased from 45×10^9/l to 4×10^9/l), we decided to continue therapy with half-dose fludarabine. The patient received two more courses (6 and 10 weeks after the first cycle), and cotrimoxazole was maintained (20 mg/kg/day sulphamethoxazole). Adenopathies and lymphocytosis, however, progressed; thus a fourth course with full dosage of fluadarabine was realised. On day 8 of this fourth cycle, the patient developed fever with interstitial pneumonia. High-dose cotrimoxazole was recommenced without performing another fibroscopy. Chest films improved and the patient was discharged on day 18. Aerosolised pentamidine was prescribed to prevent recurrence of pneumonia.

To our knowledge, this is the second case of opportunistic pulmonar infection reported after fludarabine treatment. Schilling *et al.* [5] recently reported a case of fatal pneumocystis pneumonia associated with cytomegalovirus infection, survening after three courses of fludarabine in a CLL patient. Fludarabine is known to decrease the number of all lymphocyte subgroups, especially the CD4 cells [1], and that could explain such a toxicity. However, immunological deficiency is well-known in CLL, with a decrease of CD4:CD8 ratio and functional abnormalities of T-cell subpopulations [6]. So, patients with advanced CLL are at risk of pulmonary infection or toxicity with fludarabine treatment. Our patient did not receive concomitant corticotherapy, which can increase this infectious risk. The original point in our observation is the fact that pneumocystis infection was recurrent, suggesting a close dose-dependent risk. Particular attention should be paid in such patients treated with fludarabine.

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