Facial paresis after fludarabine treatment for advanced chronic lymphocytic leukaemia

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Abstract—This case report discusses a case with advanced-stage chronic lymphocytic leukaemia (CLL) that presented with facial paresis after fludarabine treatment. A 68-year old patient with CLL (Rai classification, stage IV) was admitted to Gülhane Military Medical Academy for treatment. Fludarabine, 30 mg/m² daily for 5 days, was given. Right facial paresis was observed at day 8 after administration of fludarabine. The general and psychiatric condition of the patient in myelosuppression did not permit aetiological investigation for paresis. Thereafter, the patient died due to septic shock. Possible aetiological reasons why the patient being treated for advanced-stage CLL had facial paresis after the administration of fludarabine ended are discussed.

Key words: Bell’s palsy; chronic lymphocytic leukaemia; facial paralysis; fludarabine; neurotoxicity.

INTRODUCTION

Peripheral facial paralysis is one of the most common mononeuropathies, with an incidence of 14–25 in 100 000 per year. In 62–93% cases, the aetiological factor is unknown [1].

In this case report we discuss a case with advanced-stage chronic lymphocytic leukaemia (CLL) that presented with facial paresis after fludarabine treatment.

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CASE REPORT

A 68-year-old male patient was hospitalized suffering from weakness, anorexia, and weight loss. He was taking lithium for depression; leukocytosis was detected 2 years ago. For evaluation of leukocytosis, CLL was diagnosed. As his disease was in stage 0 according to the Rai classification, he was monitored without any treatment. At his last visit, he was complaining of progressive weakness and had lost approximately 10 kg during the last month. Physical examination revealed numerous bilateral cervical and axillary lymphadenopathies, and splenomegaly palpable at 3 cm from the left costal margin. Blood count results were as follows: WBC 81 000 per mm$^3$, haemoglobin 10.8 g/dl, haematocrit 30%, and platelets 10 000 per mm$^3$. Blood smear results were mature lymphocytes 96%, neutrophils 4%, erythrocytes normochromic and normocytic, frequent basket cells, very few platelets without clumps, and reticulocytes 0.6%. In the bone marrow smear, mature lymphocyte infiltration was 94% and there were a few megakaryocytes. Direct and indirect Coombs tests were negative. The stage of the disease was IV according to the Rai classification. He was treated with a daily dose of fludarabine at 30 mg/m$^2$ for 5 days. On the eight day after chemotherapy, right facial asymmetry emerged.

Otoscopic examination was normal. In the facial nerve examination, he was closing his eyes with difficulty and elevating his eyebrows easily; movement of the right angle of the mouth was minimal. Temporal bone CT, posterior fossa MRI, pure voice odiometry, and a stapes reflex test were planned for aetiological assessment but not performed, since he was in myelosuppression and his physical and psychological health condition was poor. Methyl prednisolone was given at a dose of 1 mg/kg daily, but haematemesis occurred and so the treatment was discontinued. At day 21 following chemotherapy, he had fever, hypotension, and tachycardia. The condition was evaluated as septicaemia/septic shock and meropenem $3 \times 1$ g daily + amikacin $1 \times 1$ g daily + vancomycin $4 \times 0.5$ g daily and dopamine $5 \mu$g/kg daily as an intravenous infusion for hypotension was started. The same day he deteriorated and died.

DISCUSSION

Peripheral facial paralysis has been reported in patients with CLL. The mechanism can be leukaemic infiltration [2, 3]. Spontaneous aggravation and relapse and a change of the site of the lesion can be seen during the course of the disease [4]. Peripheral facial paralysis has also been reported in some other lymphoproliferative disorders accompanied by leukaemic infiltration [5–8]. Although the same mechanism could also be in action in our case, the emergence of paresis following chemotherapy reduces this possibility.

In the majority of patients with peripheral facial paralysis, the aetiology remains unknown. Some aspects of the disease have similarities to viral infections. In one study, possible infectious origin was reported in 11 of 38 patients (29%); of