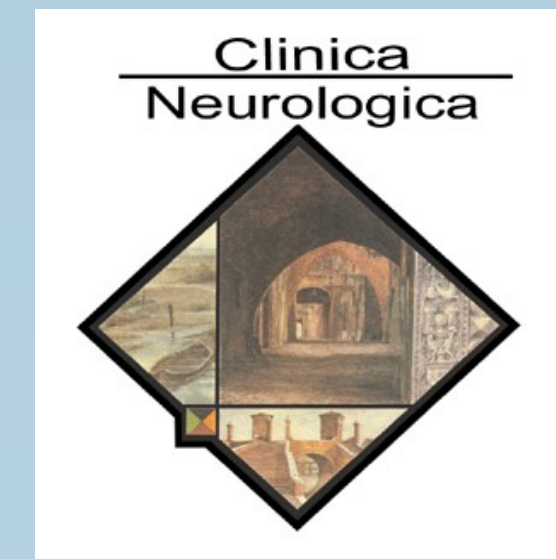


Paraneoplastic cerebellar degeneration and rhino-pharyngeal carcinoma: a case report

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Introduction

Paraneoplastic cerebellar degeneration (PCD) is a disorder caused by immune-mediated effects of cancer, irrespective of metastasis or tumor infiltration of the cerebellum. PCD is found in association with ovary, uterus, fallopian tubes or breast cancer, lung microcitoma and Hodgkin's disease. PCD features cerebellar signs and symptoms (progressive gait ataxia, dysarthria and nystagmus and other eye movement disorders), with no MRI evidence of cerebellar atrophy. There is a correlation with various autoantibodies such as anti-Yo, anti-Tr and anti-mGluR1. There is no standardized treatment of PCD, and plasma exchange (PEX), intravenous immunoglobulins or immunosuppressive agents are used. We describe a case of PCD in a 52 year-old man who undertook chemo- and radio-treatment for a rhino-pharyngeal carcinoma (RPC).

Materials and methods

The patient was diagnosed with RPC in 2014 and underwent 2 chemotherapy and several radiotherapy cycles. In a PET-CT (October 2014) no more metabolic hyperactivity of pharynx was detected. One month later he was hospitalized for fatigue and gait disorders. Thoracic CT scan showed bilateral lung nodular lesions and dorsal spine lesions. In October 2015 he was found positive for anti-Ma2 antibodies, and a cerebral MRI was negative. In December 2015 he was found negative for anti-onconeural antigens antibodies and for oligoclonal bands at isoelectrofocusing analysis in the cerebrospinal fluid (CSF), but positive for serum anti-amphyphysin antibodies. At a PET-MRI no signs of neoplastic activity. The patient was hospitalized in 2016, presenting with gaze-evoked and up-beating nystagmus, oscillopsia, mild dysarthria, left limb hypotonia, hypodiadochokinesia, ataxic gait and right lateropulsion.

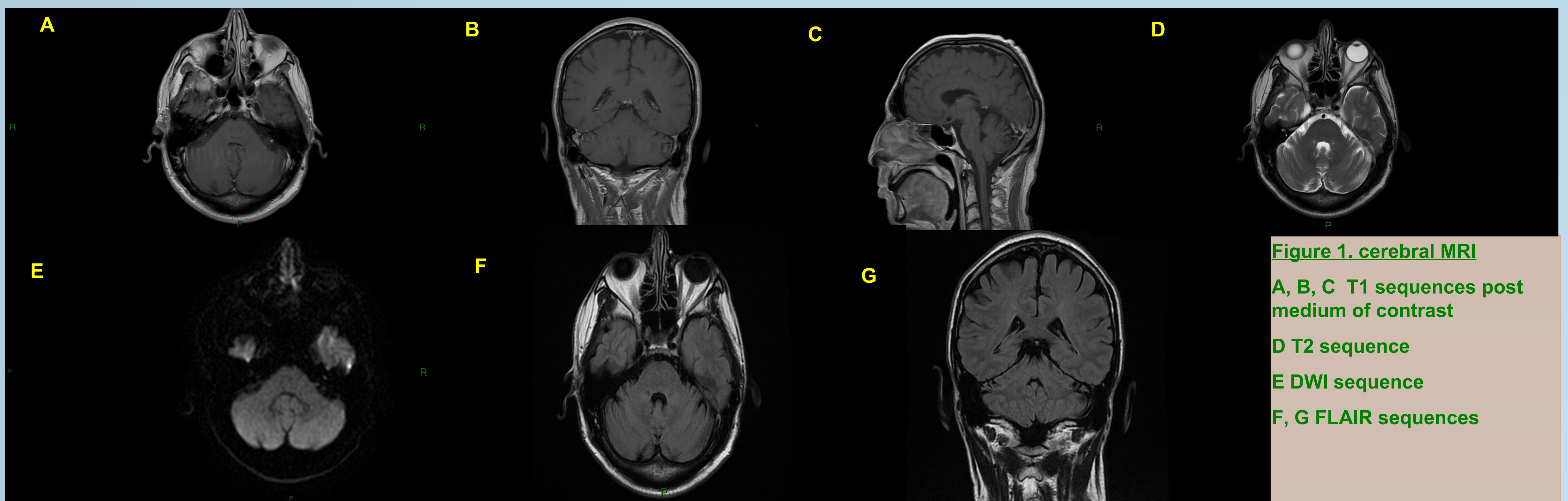


Figure 1. cerebral MRI
A, B, C T1 sequences post
medium of contrast
D T2 sequence
E DWI sequence
F, G FLAIR sequences

Results

He was found negative for onconeural autoantibodies in serum and CSF, the latter showing mild proteinorrachia. Thoraco-abdominal CT scan was negative for cancer replications. The patients showed gradual and progressive improvement after three PEX sessions in a week. At hospital discharge the patient showed improved oscillopsia, no up-beating nystagmus, reduced hypotonia, cautious gait with no ataxic notes, no lateropulsion.

Conclusions

The onconeural antibodies panel of this patient and the relatively rapid clinical remission after few PEX session were atypical for reported PCD. There are no data in literature about association between PCD and RPC. Nevertheless specific tumor-driven immune and therapeutical responses for this patient are hypothesized. RCP could be related to paraneoplastic neurological disorders.

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