



Short communication

Sequential FDG PET and MRI findings in a case of SMART syndrome



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1. Introduction

SMART syndrome (stroke-like migraine attacks after radiotherapy) is a rare and late complication of cerebral radiotherapy, occurring at least 10 years after irradiation; it is characterized by headaches and reversible unilateral episodic neurological dysfunction, less often by epileptic seizures. SMART syndrome was first described by Shuper et al. [1]. Diagnosis of SMART syndrome requires integration of clinical features, analyses of CSF, EEG, MRI and functional imaging findings. Indeed, F-18-FDG PET/CT, an imaging technique assessing glucose metabolism, is a recognized tool to localize cortical substrate in refractory epilepsy. This technique helps to better understand SMART physiopathology. Here, we report the case of a 53-year-old man with previous lung cancer, who presented epileptic seizures despite antiepileptic treatment. Such a clinical presentation evoked a SMART syndrome and interesting functional imaging findings helped in consolidating diagnosis.

2. Case report

A 53-year-old man was admitted to the emergency department because of headaches, fluctuant confusion, agitation, aphasia, right hemiplegia, right homonymous hemianopia, hyperthermia and focal epileptic seizures (right upper limbs abnormal movements) despite his antiepileptic treatment (p.o. 2 × 300 mg/day lamotrigine). This treatment was given because of a previous similar episode with continuous partial seizures one year before. At that time, no etiology was found. The patient had a history of two metachronous frontal cerebral metastases of lung adenocarcinoma treated with radiotherapy (45 Gy) 12 years before. Cerebrospinal fluid analysis revealed hyperproteinorachia with no malignant cells. EEG assessed signs of left posterior slowing and frontal slowing focus without epileptiform activity.

MRI showed increased signal on FLAIR and postcontrast enhancement on T₁-weighted images in the left temporo-parietal region (Fig. 1A and B). It is important to emphasize that there was no evidence of residual or recurrent tumor, in the brain as much as in the cerebellum. The patient underwent F-18-FDG PET/CT for oncologic restaging, which showed intense and large cerebral cortical hypermetabolism in the left temporal, centro-parietal and occipital regions (Fig. 1C and D), based on which we suspected a SMART syndrome. Seizures progressively regressed under i.v. 1 g methylprednisolone and modification of antiepileptic treatment (p.o. 2 × 500 mg levetiracetam and 2 × 200 mg lacosamide). MRI and F-18-FDG PET/CT performed 10 days later, while the patient

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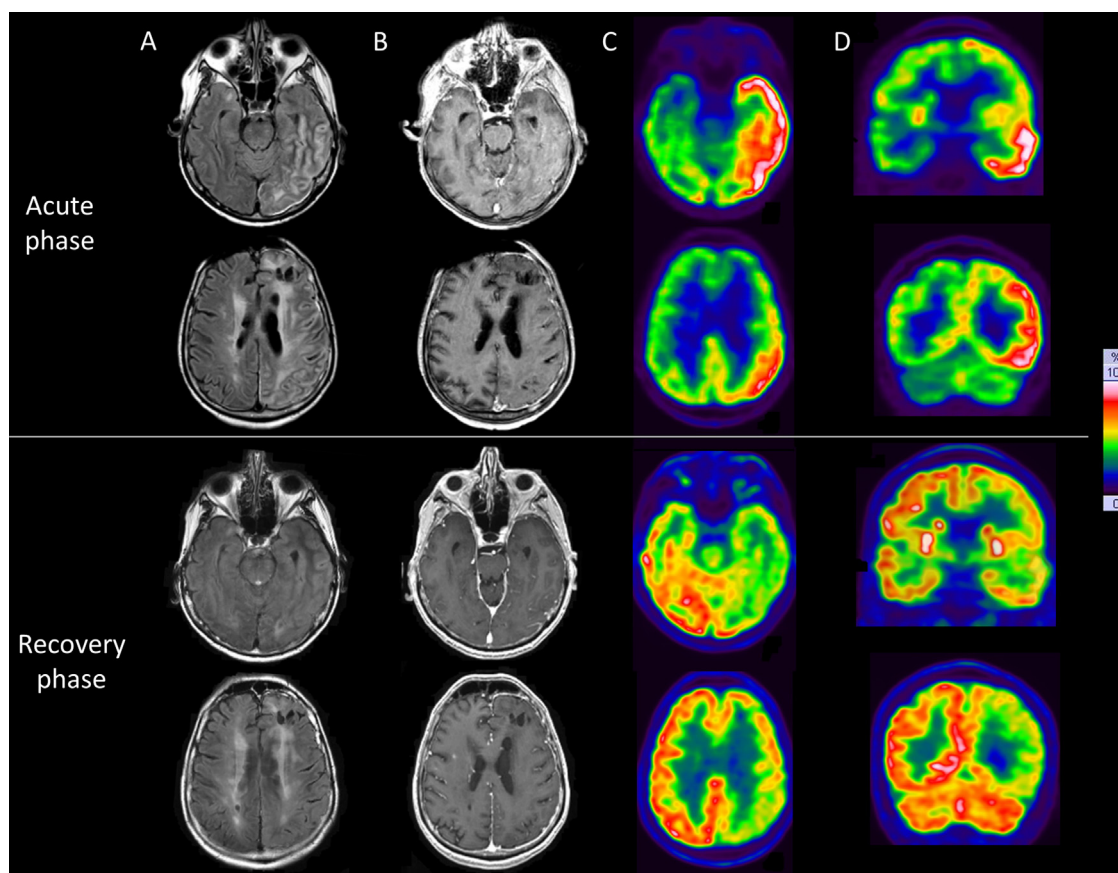


Fig. 1. Matched temporal and parietal level transverse-slice fluid-attenuated inversion recovery (FLAIR) MRI (A), postcontrast T₁-weighted MRI (B), F-18-FDG PET (C) and coronal F-18-FDG PET (D) during SMART episode and 10 days later during recovery period.

only presented discrete right hemiparesis, showed cerebral cortical hypometabolism in the left temporal, centroparietal and occipital regions (Fig. 1C and D) and regression of signal abnormalities on MRI (Fig. 1A and B). These patterns thus achieve to characterize cerebral dysfunction.

3. Discussion

While pathophysiology of SMART syndrome is unknown, neuronal dysfunction seems to be an explanation. Indeed, there are mostly case reports in the literature with characteristic intense cortical ribbon enhancement on MRI [2]. However, only a few case reports have been published pointing out FDG brain uptake [3]. Based on MRI findings, several diagnoses were discussed here: a potential infection, radionecrosis, seizure-induced transient MRI abnormalities or SMART syndrome. F-18-FDG PET/CT enabled to decide. Functional neuroimaging findings, such as F-18-FDG PET/CT, show hypermetabolism and argue in favor of an ictal epileptic origin of the symptoms, as in our patient. Moreover, we report here both ictal and recovery glucose metabolic studies, revealing respectively hyper- then hypo-metabolism in large cerebral left regions. It has to be noticed that the hypothesis of radiation-induced cerebral vascular reserve dysfunction has also been studied with brain perfusion SPECT but a vascular mechanism was finally not retained [4]. To our knowledge there are few studies showing ictal and recovery glucose metabolism, enabling hypothesis about pathophysiological mechanism. F-18-FDG PET/CT assessed hypermetabolism 4 days later after acute phase. However, classical hypometabolism linked to tissue depletion should have been awaited. Finally, F-18-FDG PET/CT in recovery was linked to the good clinical evolution and enabled to monitor therapeutic. It is

also important to underline that F-18-FDG PET/CT showed no neoplastic recurrence. In fact, this technique allows distinguishing neoplastic recurrence *versus* radionecrosis.

4. Conclusion

Diagnosis of SMART syndrome requires integration of clinical features, analyses of CSF, EEG and imaging findings, especially with MRI and F-18-FDG PET/CT. Assessment of this syndrome is of value because of its reversibility which is important to explain to patients and their families and to avoid invasive investigations. Hence, F-18-FDG PET/CT is also a complete tool allowing assessing a potential neoplastic recurrence in the brain and providing a whole-body oncologic exploration for these patients with a history of tumor.

Conflicts of interest

All authors declare no conflict of interest.

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