Focal status epilepticus originating from the central and parietal regions: the clinical and etiological spectrum

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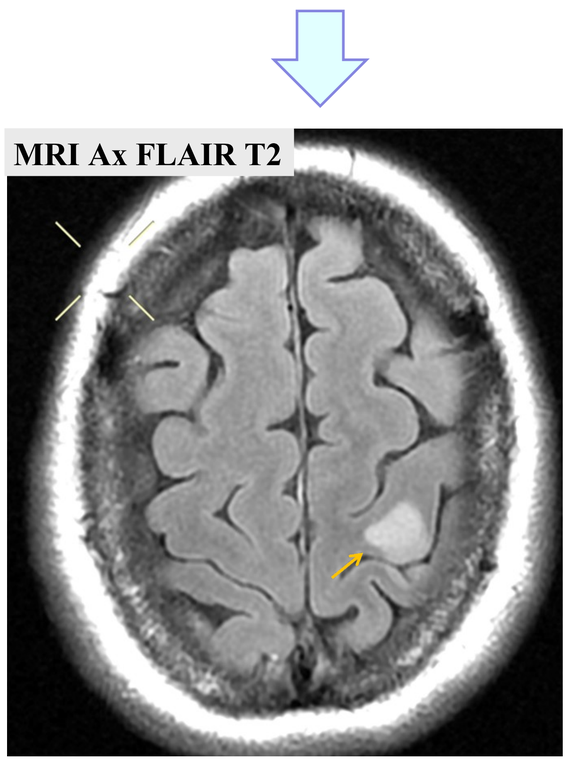
Objectives: focal status epilepticus (FSE) has been extensively studied in order to identify the proper etiology and best treatment to adopt. Whether localization of FSE may influence the response to treatment is largely unknown. Moreover, even within the same localization, the clinical manifestations and etiologies of FSE may vary. Here we report four cases of successfully treated central and parietal FSE characterized by a variety of etiologies and clinical features.

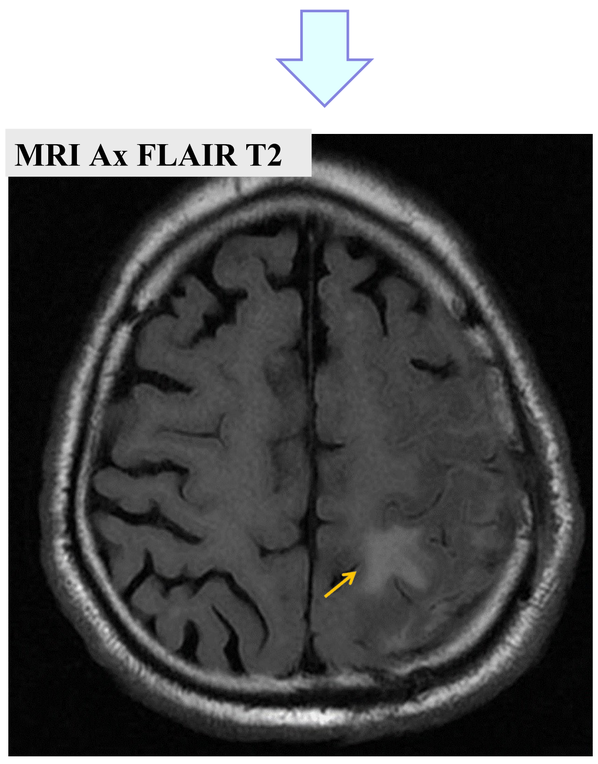
Methods: four adult patients with FSE originating from parietal lobe structures have been extensively studied by means of detailed history, repeated video-EEG monitoring, neuroradiological exams and extensive laboratory tests. These patients underwent also multiple therapeutic trials.

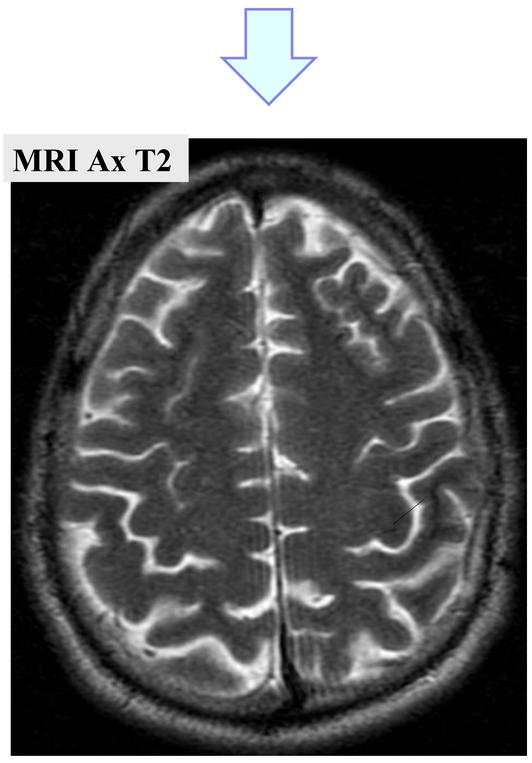
	CASE 1	CASE 2	CASE 3	CASE 4
SEMIOLOGY *	Focal motor (Epilepsia partialis continua)	Focal onset (motor) evolving into bilateral convulsive SE	Without impairment of consciousness (somato-sensory "pain") → Repeated focal motor seizures	Repeated focal motor seizures (Jacksonian) → Without impairment of consciousness (somato-sensory- "alien limb")
ETIOLOGY	Unknown (autoimmune suspected)	Gliosis following parietal meningioma ablation	Non-ketotic hyper-glycaemia	Post-haemorrhagic lesion
EEG CORRELATES **	No ictal change	Fronto-central spike-and-wave discharges → generalised spike-and-wave dicharges → lateralised periodic discharges + fast activity → burst suppression	Centro-parietal spike-and-wave discharges →centro-parietal sharp-and-wave activities → lateralized quasi-periodic discharges	Parieto-temporal spike-and-wave discharges → lateralized quasi-periodic discharges
AGE	42	81	60	71
THERAPY	1°→ DZP 2°→ PHT ev, 3°→ VPA ev, 4°→ LEV ev, 5°→ LEV per os + CLB 6°→ LEV + CLB + CBZ 7°→ LEV + CLB + LCM 7°→ LEV + CLB + CBZ + TPM	1° → LEV 2° → LEV + PHT 3° → LEV + PHT + MDZ (until 0,3 mg/Kg/h)	1° → PHT ev 2° → PHT per os + CLB 3° → PHT + CLB + LEV	1° → PHT
LENGHT	≈ 40 days	9 days	15 days	6 days

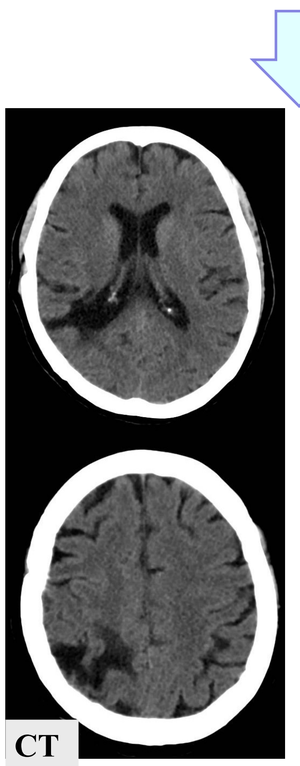
* Trinka E, Cock H, Hesdorffer D, et al. A definition and classification of status epilepticus – Report of the ILAE Task Force on Classification of Status Epilepticus. Epilepsia 2015: Sept:1–9.

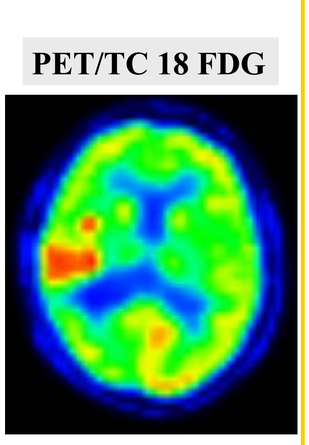
**Hirsch LJ et al., LaRoche SM, Gaspard N, et al. American Clinical Neurophysiologyndardized Critical Care EEG Terminology: 2012 version. J Clin Neurophysiol 2013;30:1–27.











We have reported 4 cases of SFE originating from central and parietal structures showing a variety of etiologies, clinical features and response to treatment. In the literature central and parietal status is considered exceedingly rare and has not been described in detail so far. Moreover there is no study addressing the usefulness of a localization-related classification of FSE.
THE SEMIOLOGY AND ETIOLOGICAL BASES OF PERI-ROLANDIC FSE ARE POLYMORPHOUS. THE RESPONSE TO TREATMENT MAY BE ALSO VARIABLE, DEPENDING ON ETIOLOGY. IN EACH PATIENT THE CLINICAL MANIFESTATIONS AND THE UNDERLYING BRAIN PATHOLOGY ARE CRUCIAL TO CORRECT DIAGNOSIS AND TAILORED MEDICAL THERAPY.