

A Frontal Lobe Syndrome Case Unresponsive to Antipsychotic Medication

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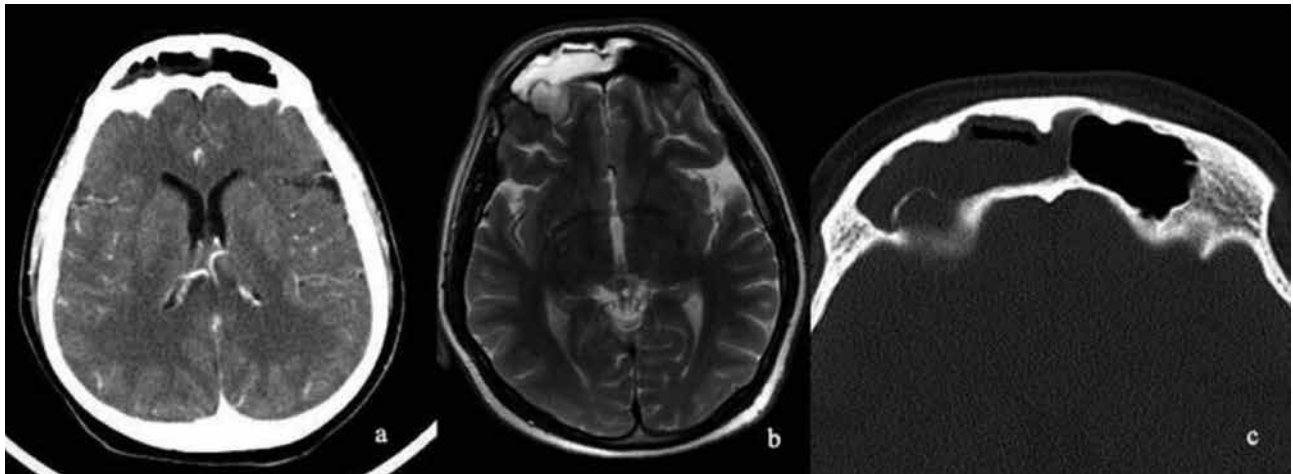
Dear Editor;

Frontal lobe syndrome develops due to damage of prefrontal region by various reasons. Majority of patients have common clinical signs as executive function disorders, apathy, social dissonance, problems with impulse control, obsessive behaviors, and aggressive behaviors. Behavioral changes may be confused with symptoms in personality disorders and schizophrenia (1). In this case, frontal lobe syndrome in a schizophrenia patient who was under control for a long time, and deteriorated suddenly, but did not respond to electroconvulsive treatment (ECT) and combined drug treatment, would be discussed.

A 39-year old male patient treated and followed up with schizophrenia approximately for 10 years. He had no accompanying disease, and was under control with risperidone 2mg/day and quetiapine 50mg/day. In the last 2 months, he started to yell at his surroundings, and behave aggressively, such as using abusive language and spitting, that he had never done before. He was hospitalized. Despite increments of drug doses (long-acting intramuscular risperidone, quetiapine 300mg/day), he showed no sign of recovery within one

week, so treatment protocol was switched (amisulpride 800mg/day, valproic acid 1000mg/day, clozapine 400mg/day, biperiden 2mg/day, zuclopenthixol decanoate 200mg weekly).

As no marked clinical improvement was observed during two weeks' of treatment, ECT treatment was added on for seven days. In majority of this patient group, combination of antipsychotics with ECT is a treatment option (2,3). After ECT, he improved clinically for a very short time (1-2 hours). Therefore, neurology consultation was requested to rule out an underlying organic cause. His verbal communication was unclear; his judgement was poor; and he had no insight. Neurological examination was roughly within normal limits. There was no stiff-neck. Routine blood tests were within normal limits. He had a contrasted cranial computerized tomography (CT) which was performed months ago during outpatient clinic follow-ups, and it was reported within normal limits (Picture 1a). Cranial magnetic resonance imaging (MRI) imaging examination and electroencephalography (EEG) were requested. In EEG, mild background activity was observed, whereas an infiltrative lesion which was supposed to be originated from the right frontal sinus,



Picture 1: Contrasted cranial CT within normal limits performed before the clinical picture (a). In cranial MRI T2 sequence, increased soft tissue intensity in the right compartment of the frontal sinus, thinning of calvarial bone in posterior cross-section, and infiltration into the right frontal lobe (b). In paranasal CT, frontal sinusitis on the right, and mucocele formation with decreased bone density at the posterior (c).

and invading the right frontal lobe was observed in MRI (Picture 1b). Ear-nose & throat consultation was requested. In his paranasal CT, sinusitis and thinning of bone were observed at this region (Picture 1c). According to culture results, it was defined as aspergillum, so amphotericin B and itraconazole treatment was started. He is still having the treatment.

Frontal lobe syndrome may be encountered due to frontal area damage caused by various reasons. Krudop et al. (4) diagnosed a pathology in 89% of patients who had frontal lobe syndrome. Disinhibition,

aggression, social dissonance, and inappropriate sexual behaviors were reported especially in ventromedial prefrontal cortex lesions (4). In our patient, an infection originated from sinuses, and infiltrated frontal lobe was diagnosed. Cases with aspergillus infection with central nervous system involvement are rarely reported (5). In conclusion, an underlying organic reason should be considered in schizophrenia patients with regular clinical progression, if they develop a rapid clinical deterioration and treatment non-responsiveness.

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