

CASE REPORT

Unusually Deep Intracranial Extension of Instrument with Internal Carotid Artery Injury during Functional Endoscopic Sinus Surgery

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ABSTRACT

Functional endoscopic sinus surgery is a widely accepted technique, performed extensively in conjugation with septoplasty and paranasal sinus disorders, with good results and minimal complications. Even though, extreme care is required during surgery due to the proximity of the sinuses to the eyes, optic nerves, brain and internal carotid arteries. Here we report a rare case of unusually deep intracranial extension of instrument with internal carotid artery injury during functional endoscopic sinus surgery.

Keywords: Functional endoscopic sinus surgery, Internal carotid artery dissection, Stroke.

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INTRODUCTION

Rhinology and sinus surgery have undergone a tremendous expansion since the discourses of Messerklinger and Wigand in the late 1970s.¹⁻³ Imaging advances allowed surgeons to perform more complex procedures with increased safety. Today, functional endoscopic sinus surgery (FESS) is a widely accepted technique, performed widely in conjugation with septoplasty⁴ and paranasal sinus (PNS) disorders, with good results and minimal

complications. Intranasal and intraorbital complications are well described but major complication rate remain low (0.23-0.46%).^{5,6} Advances in FESS technique and equipment have made the procedure safer, faster, more precise and more comfortable. Here, we report an unusually deep intracranial extension of FESS instrument into cerebral parenchyma with intracranial hematoma in the track site and internal carotid artery injury. Internal carotid artery injury due to accidental instrument passage into skull base during FESS is rare grave complication with possible torrential hemorrhage secondary to internal carotid artery injury.

CASE REPORT

A 25-year-old male, with history of endoscopic septoplasty for deviated nasal septum (DNS) one year back, underwent recent revision septoplasty in a private hospital. Post procedure, there was delay in recovery from anesthesia, and on recovery there was complete right sided hemiplegia and he was in drowsy state. Later he had profuse cerebrospinal fluid (CSF) discharge from nose and continuous high grade fever. He was transferred to nearby medical college and later, he was referred to our center for further management. At the time of presentation, he was drowsy, continuously febrile with features of acute meningitis and dense right hemiplegia with grade zero power. He underwent magnetic resonance imaging (MRI) (Figs 1A to H), which revealed two hemorrhagic tracks, one traversing from cribriform plate → orbitofrontal gyrus → thalamus → body of right lateral ventricle → small portion of left cingulate gyrus. The other longer track was traversing from cribriform plate → orbitofrontal gyrus → thalamus and posterior limb of right internal capsule → cranial portion of external capsule → right frontoparietal white matter → right inferior parietal lobule cortex. At the terminal portion of longer latter track, Y-shape division of track seen possibly corresponding to track of two limbs of an open forceps. The both tracks were T1 (Fig. 2A)/T2/FLAIR hyper intense, GRE blooming suggestive of late subacute bleed along the track. Air foci were noted within nondependent portion of bilateral frontal horn and left temporal horn of lateral ventricles (Fig. 2B) suggesting instrumental entry into

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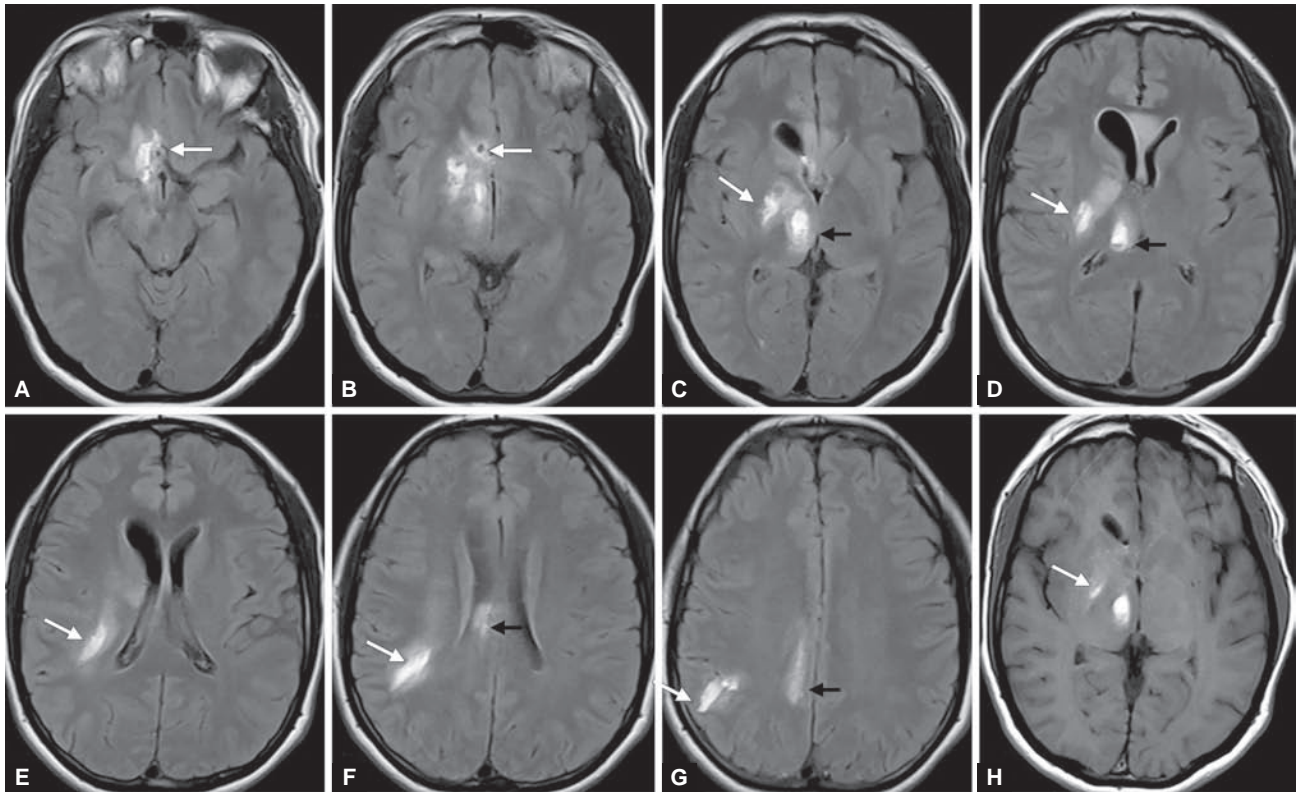
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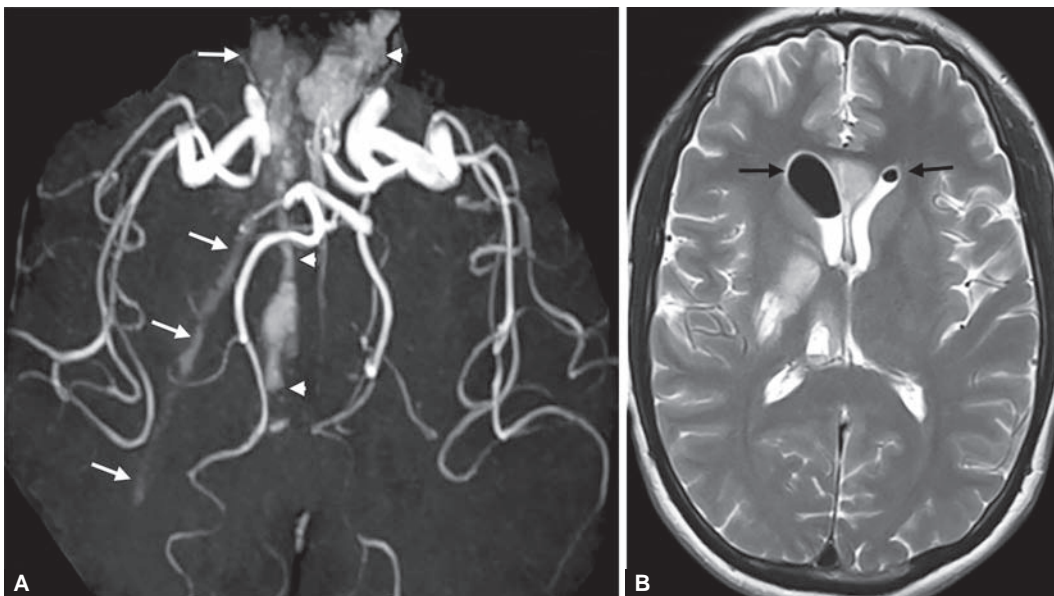
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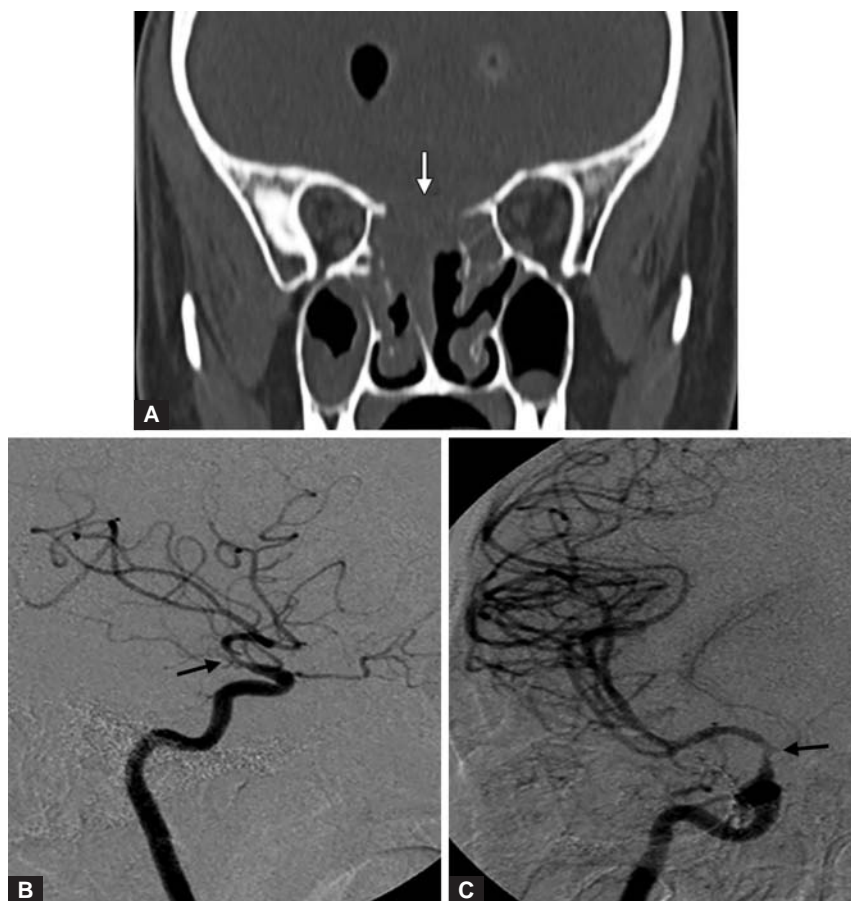
Figs 1A to H: (A to G) Serial cross-sectional FLAIR images revealed two hyperintense tracks, one track traversing from right basifrontal lobe through posterior limb of internal capsule till parietal lobe cortex (white arrows), another track traversing from basifrontal lobe through thalamus to cingulate gyrus (black arrows) and (H) The tracks are hyperintense on axial T1w image suggesting late subacute stage hematoma



Figs 2A and B: (A) Time-of-flight (TOF) image, thick slab maximum intensity projection (MIP) basal view, showing course of two hyperintense tracks (arrow and arrowhead) and (B) Axial T2w image reveals air nondependent portion of frontal horns of lateral ventricles

lateral ventricle. Diffusion restriction of posterior limb of internal capsule corresponding to right anterior choroidal artery territory noted. CT scan (Fig. 3A) revealed large defect through cribriform plate, roof of sphenoid sinuses, bilateral sphenoid hemosinus. Digital subtraction angiogram (DSA) revealed stenosis with suspicious dissection flap in right supraclinoid internal carotid artery (Figs 3B

and C). CSF study revealed acute meningitis. After acute meningitis management and other supportive conservative management, his consciousness improved over next 10 days, and his right hemiplegia improved and CSF rhinorrhea stopped by conservative management. On 3 months follow-up, he became fully independent with no CSF rhinorrhea, and doing same till now.



Figs 3A to C: (A) Computed tomography coronal sections showing large defect in roof of nasal cavity, (B) Lateral and (C) AP projections of right ICA injection during DSA revealed focal stenosis with suspicious dissection flap in right supraclinoid ICA

DISCUSSION

Nasal endoscopy is an excellent method for the precise diagnosis of pathological abnormalities of the nasal septum,⁷ used widely combined with powered instrumentation to perform nasal septal surgery. Septoplasty, which is among the three most commonly performed procedures in otolaryngology,⁸ is particularly well suited to endoscopic application. The combined approach is advisable because during common nasal procedures, the surgeon's view is obstructed due to the narrowing caused by septal spurs or septal deviations.⁹ Endoscopic septoplasty is considered as a minimally invasive and safe technique advisable for the patients those who had previous nasal septal surgery.^{10,11} Most common intracranial complication related to endoscopic surgery being CSF leak secondary to dural tear, others include meningitis, brain abscess, intracranial and torrential nasal hemorrhage secondary to internal carotid artery injury. In our case, patient had profuse CSF leak, acute meningitis, ICA injury and intracranial hematoma. Accidental instrument passage into intracranium is more frequently seen in revision surgeries than in first session of FESS. Patient became left hemiplegic immediately

secondary to right internal capsule damage and right hemispheric intracerebral hemorrhage. The FESS operator should monitor the instrument penetration depth in order to assess its internal movement. More caution is required in revision surgeries, as the bony skull base barrier damage during previous surgery allow passage of FESS instrument easily into intracranial compartment without undue resistance being felt. Also, the distorted anatomical landmarks, two FESS sessions performed by different operators also might have contributed to the current intracranial complication.

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