Chronic rhinosinusitis complicated by intracranial suppuration

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ABSTRACT

Background: Intracranial abscess formation secondary to chronic rhinosinusitis (CRS) is a rare, but life-threatening infection with a poor outcome.

Case presentation: A 63-year old Caucasian male with a history of CRS presented with one day of fever (40.3°C), repeated vomiting, two episodes of generalized seizures, and later became unresponsive. The patient was diagnosed with pansinusitis and a cerebral abscess, and he was treated successfully with surgery and antibiotic therapy. At follow-up nearly 9 years later, the patient had only minor complaints despite severe sinus pathology on the follow-up computed tomography scan.

Conclusion: Early diagnosis and treatment of intracranial complications are essential to reduce subsequent morbidity and mortality. After an acute exacerbation, imaging findings and subjective complaints may differ. Treatment should, therefore, be based on a combination of objective and subjective findings.

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Introduction

Chronic rhinosinusitis (CRS) (with or without nasal polyps) in adults is defined as the presence of two or more of the following symptoms for at least 12 weeks, one of which should be either nasal blockage or nasal discharge with or without facial pain/pressure and reduction or loss of smell [1]. CRS, acute bacterial rhinosinusitis (ABRS), otitis media, head injury, meningitis, and metastatic spread from distant foci are the main causes of intracranial suppuration (ICS) [2]. In immunocompetent patients, bacteria are responsible for most (>95%) intracranial abscess (ICA) [3]. ABRS is defined as three or more symptoms of the following fever (above 38°C), double sickening (worsening of symptoms after initial improvement), unilateral disease, severe facial pain and/or raised c-reactive protein (CRP) [1]. An Acute exacerbation of chronic rhinosinusitis (AECRS) is defined as worsening of symptom intensity with return to baseline CRS symptom intensity [1]. ICS may occur secondary to both ABRS and CRS [4]. However, ICS secondary to CRS is a rare condition that does not appear to be a result of inadequately treated CRS [5]. Usually, ICS develops as a result of the topographical relationship between the paranasal sinuses and the anterior and middle cranial fossae, and include epidural, subdural, and intracerebral abscess, meningitis, and cavernous sinus thrombophlebitis [4].

The frontal sinus is most frequently involved in ICS [4], and the frontal lobe is the most common site of ICA formation. Presenting symptoms are related to the size, location, and virulence of the infecting organism [6]. They include symptoms of increased intracranial pressure, fever, neurological deficit, epileptic seizures, hemiparesis, and cranial nerve paralysis [2–5]. However, ICS can be neurologically silent, or only present with subtle changes in mood or personality [5]. The classic clinical triad of headache, fever, and neurological deficit is only complete in nearly 20% of patients on admission [3].

Here we present a serious case of a patient with an ICA secondary to CRS. The treatment was successfully based on an excellent recovery in contrast to the control computed tomography (CT) findings of the sinuses.
Case presentation

A 63-year-old male with a history of atrial fibrillation, and CRS (through 30 years with daily nasal discharge and intermittent epistaxis), presented with fever (40.3°C), repeated vomiting, and two episodes of generalized seizures. The patient was unresponsive with a Glasgow Coma Scale (GCS) score of 3 on arrival at the emergency department (ED). However, there was no nuchal rigidity or petechiae. An acute lumbar puncture showed no purulence. The patient developed vertical conjugate eye deviation, anisocoria with normal pupillary light reflex, left-sided Babinski reflex, right-sided indifferent response, but had spontaneous breathing. After 30 min in the ED, the patient had a GCS of 10 but sustained left-sided neurologic deficits.

This presentation was initially interpreted as purulent meningitis, and treatment according to guidelines was initiated. However, the results of the cerebral CT (Figures 1 and 2) showed a cerebral abscess in the anterior cranial fossa originating from the right frontal sinus, opacification of all the sinuses, and an intra-orbital abscess. The patient had a history of CRS and a sinus mucocele, and he was diagnosed with ICS caused by either an AECRS or via an infected mucocele.

In the following four weeks, the patient received a total of six surgeries (three neurosurgeries (gathering a microbiological specimen collection, draining of an epidural empyema twice, and evacuating an IC hematoma), and three endoscopic sinus surgeries (exploring and cleaning out the sinuses, and drainage of the abscess in the right orbital cavity)). Culture from the brain and sinuses grew non-hemolytic streptococcus, and the patient received six weeks of intravenous penicillin and peroral metronidazole. After six weeks of antibiotics, a bone scintigraphy and radiolabeled white blood cell scintigraphy showed osteomyelitis. Subsequently, the patient was treated with an additional six weeks of antibiotics.

Due to persistent rhinorrhea and facial pain, the patient was re-admitted seven months later. On nasal endoscopy, the right frontal sinus was filled with fibrotic scar tissue but there were no traces of pus. In the aftermath, the patient resumed his position as an associate professor in mathematics. The patient was followed clinically and with CT scans for the following 3 years.

Nearly 9 years after his initial admittance the patient was seen in the clinic to assess any development in his CRS. In this context, the patient was invited for a clinical control and CT scan. The patient experienced no neurologic sequelae and reported only occasional mild frontal headache, and nasal discharge. On examination, the nasal mucosa was moderately swollen, and the right frontal recess was narrow with only limited access. The follow-up sinus CT (Figure 3) showed stationary conditions with multiple rather large isolated sinus isles in the frontal bone. A moderate mucosal thickness was found in the right maxillary sinus, the left ethmoid sinus, and both frontal sinuses. It was concluded that it would require comprehensive surgery and bone drilling to reach the isolated sinuses and since the patients’ symptoms of CRS were well controlled, they were not addressed.
surgically. The patient is still followed in the outpatient clinic and remain well controlled.

Discussion

This case is important for several reasons. First, it demonstrates that patients, who suffer from a severe ICA secondary to CRS, can return to the same high level of social functioning with no or few sequelae. Secondly, it illustrates that severe pathology on CT does not equal subjective complaints and it is important for the surgeon to carefully consider the indication for surgery if the patient is asymptomatic. Finally, it highlights the importance of clinical suspicion, early diagnosis, and aggressive treatment to accomplish this positive outcome.

The most common presenting symptoms of ICS secondary to frontal CRS are low-grade fever, malaise, and frontal/retroorbital headache or tenderness. Yet, some patients are asymptomatic [5]. When the diagnosis is made ‘in time’, and medical and surgical management is applied as appropriate, the rate of cure is >90% [3].

To our knowledge, no prior studies have examined the prevalence of ICS secondary to ABRS in patients with no previous history of CRS compared to patients with a history of CRS. However, it would be relevant to consider.

The first-line treatment for an ICA <2.5 cm is antimicrobial therapy, while stereotactic aspiration is recommended for those >2.5 cm [3]. No clinical study has specifically addressed the length of antibiotic treatment for an ICA, but most experts recommend six weeks of antibiotics for immunocompetent patients [3].

Conclusion

The formation of an ICA secondary to CRS is rare and unpredictable. Quick recognition of symptoms and fast treatment initiation remains the cornerstones in minimizing morbidity and mortality. Our patient is a perfect example of how severe pathology and subjective complaints may differ. Treatment should, therefore, be based on a combination of objective and subjective findings.

Consent

Written informed consent for patient information to be published was provided by the patient.

Disclosure statement

No potential conflict of interest was reported by the author(s).

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