

Presentation of rhinosinugenic intracranial abscesses

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SUMMARY

Intracranial abscesses secondary to rhinosinugenic disease are uncommon and the incidence is poorly documented. It is generally believed that individuals at risk of developing this complication can be identified by presenting clinical features.

A ten year retrospective Scottish national survey 1976-1985 of intracranial abscesses was carried out. Clinical and or radiological evidence of nasal/paranasal aetiology in abscesses localised to the frontal lobe, extradural or subdural spaces allowed 23 surgery or autopsy confirmed abscesses to be classified as rhinosinugenic.

12 abscesses occurred in individuals aged 0-19 years, two in patients with risk factors for intracranial spread, 8 of 11 adults had similar predisposing features ($p < 0.01$). This is a rare complication and individuals at risk in the first two decades of life can seldom be identified prospectively on clinical grounds.

INTRODUCTION

The proportion of intracranial abscesses attributable to rhinosinugenic disease varies from 0.5% to 16% (Yang, 1981; Nielsen et al., 1982; Bradley et al., 1984). Intracranial abscesses are still associated with significant mortality, current rates being 5-14% (Johnson et al., 1988; Patrick et al., 1988). The need to identify patients at risk of developing this complication thus remains. Blumenfeld and Skolnik (1966) suggest that patients with rhinosinusitis who develop orbital signs or forehead swelling are at risk of developing an intracranial complication. Small and Dale (1984) noted that almost 50% of patients with rhinosinugenic intracranial abscesses first developed frontal osteomyelitis. The presence of another complication may be an indicator for intracranial spread.

PATIENTS AND METHODS

A retrospective survey of intracranial abscesses occurring in Scotland over a ten year period 1976-1985 was carried out. Cases were identified from the data base of the Information and Statistics Division of the Scottish Health Service Common Services Agency and the departmental records of the four neurosurgical centres in Scotland.

Case records for 344 (67%) of the 517 cases identified were obtained. 173 case records could not be retrieved for review. Frontal lobe, subdural and extradural abscesses confirmed at surgery or autopsy were attributed to nasal/paranasal aetiology on the basis of clinical and or radiographic findings.

A subdural or extradural abscess found in a patient with an underlying intracranial abscess was not counted as a separate event, the abscess being classified as intracerebral. When a subdural and extradural abscess coexisted without an underlying intracerebral lesion it was classified as subdural. No patient therefore was counted more than once.

Cases were characterised by sex, age, orbital signs, signs of frontal bone osteomyelitis or erosion on examination or plain radiography and preceding nasal/paranasal trauma including surgery.

RESULTS

86 intracranial abscesses localised to the frontal lobe, subdural or extradural spaces were identified showing a 2:1 male predominance ($p < 0.01$). 18 and 13 of the 86 abscesses respectively were idiopathic or post skull trauma in origin. 32 others of non-rhinosinugenic origin were due to a variety of causes both distant cardio-pulmonary, septicaemia or local meningitis, post-neurosurgery, intracranial haemorrhage.

23 abscesses were attributable to nasal/paranasal infection or surgery. 50% (12) occurred in patients under the age of 20 years. Abscess location and site of origin of infection within the nose or paranasal sinuses is shown in Table 1.

Eleven adults (20 years or older) developed rhinosinugenic abscesses, eight with identifiable risk factors (Table 2). One patient had a frontal bone defect secondary to a frontoethmoid mucocoele, another developed a subdural abscess as a complication of intranasal polypectomy. Four with chronic sinusitis presented with frontal bone osteomyelitis in two cases, a palpable frontal bone erosion in one and periorbital cellulitis in another. Two of the five adults with acute sinusitis as the aetiology presented with periorbital cellulitis.

Table 1. The site of nasal/paranasal sinus disease associated with the development of intracranial abscesses.

origin of infection	intracranial site of abscess		
	frontal lobe	extradural space	subdural space
frontal sinusitis	1	1	3
maxillary sinusitis	1	0	1
unilateral pansinusitis	2	1	3
bilateral sinusitis	4	1	3
nasal	1	0	1
total	9	3	11

Table 2. Clinical details of the 23 cases satisfying the inclusion criteria of the study.
+ = present, - = absent.

source	age	sex	peri-orbital signs	frontal bone osteomyelitis	trauma nose/sinus
sinusitis					
acute	23	f	-	-	-
acute	10	f	-	-	-
acute	9	f	-	-	-
acute	19	m	-	-	-
chronic	48	f	-	+	-
acute	26	m	-	-	-
acute	11	m	-	-	-
acute	23	m	+	-	-
acute	23	m	+	-	-
chronic	60	m	-	+	-
acute	17	m	-	-	-
chronic	29	m	-	+	-
acute	15	m	-	-	-
acute	21	m	-	-	-
chronic	67	f	+	-	-
acute	13	m	-	-	-
acute	10	f	-	-	-
acute	17	m	-	-	-
acute	17	m	-	-	-
septal abscess	10	m	-	-	+
surgery	17	f	-	-	+
surgery	69	m	-	-	+
mucocoele	67	f	+	+	-

In contrast a predisposing condition for intracranial spread was present in only two of 12 juvenile patients (Fishers exact test $p < 0.01$). A septal abscess in a 10 year old and a 17 year old with perennial rhinitis treated by submucous diathermy who developed a subdural abscess 19 days after surgery.

Bacteriology results were available on 15 of the cases. The most frequent bacterial isolate was streptococcus as a solitary isolate in seven cases, and with a gram negative bacillus in one. Haemophilus species were identified in two cases as a solitary isolate in one and with an anaerobic coccus in another. Unclassified gram positive cocci were identified in two cases, staphylococcus in one and no organism in two cases. The nasal/paranasal bacteriology differed from the intracranial bacteriology in the patient with a septal abscess where both staphylococci and streptococci were isolated from the septal abscess but only streptococci from the intracranial abscess.

DISCUSSION

There are difficulties in classifying intracranial abscesses as secondary to rhinosinogenic disease. The potential error in relying on radiographic criteria is illustrated by the false positive and negative correlations between sinus radiography and endoscopy of 35% and 9% respectively reported by Croft (1987). No such figures are available for sinus findings on CT scan. The occurrence of false negative CT scans in the presence of subdural abscesses (Kaufman et al., 1983) suggest that CT sinus findings are not exempt from a degree of unreliability. Notwithstanding the above, radiological and clinical findings are the criteria widely used in the literature to define a case as secondary to paranasal disease thus making the present survey comparable.

The proportion of rhinosinogenic intracerebral abscesses not localised to the frontal lobe is small (Kaplan, 1976) and using frontal lobe localisation in the absence of subdural or extradural collections aims to increase the repeatability of the study. There is no reason to believe that the case records that were accessed were significantly different from those that were not and the bacteriology is similar to that seen in series of sinogenic intracranial abscesses (DeLouvois et al., 1977).

The present survey illustrates an age dependent dichotomy in the presence of signs of impending intracranial spread. This is due to the large proportion of adult cases five out of 11 secondary to chronic inflammatory change in the paranasal sinuses which spread by bone erosion resulting in frontal bone and periorbital signs occurring before or in association with intracranial spread. In the paediatric patients intracranial spread was a complication of acute rhinosinogenic inflammatory processes and intracranial spread is presumed to have occurred through vascular channels phlebitis or as part of a septicaemia.

The practising clinician should therefore be aware that in adults intracranial rhinosinogenic abscesses are most often a complication of chronic sinusitis and occur in patients with signs of other sinus disease complications. Juvenile patients who develop rhinosinogenic intracranial abscesses seldom do so in the presence of other complications of rhinosinusitis, indeed in this series only two of 12 juveniles had identifiable predisposing conditions the majority of cases developed as the only complication of an acute sinusitis. It is recommended that patients who develop complications of rhinosinusitis if adult require urgent paranasal surgical intervention as bone pathways for intracranial spread have probably developed whereas in children such pathways cannot be presumed and each complication should be treated on its own merits.

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