Don't sue the surgeon: remineralisation of the skull base or a defect that never existed?

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Abstract

Introduction: Intracranial complications are recognised as rare, but serious, sequelae of endoscopic sinus surgery.

Case report: A 56-year-old woman was referred after developing meningitis following elective functional endoscopic sinus surgery. Computed tomography demonstrated a significant defect of the skull base in the right posterior ethmoid, clearly visible on both coronal and sagittal sections. Operative exploration demonstrated the skull base to be intact in the posterior ethmoid area identified on the scan, and the overlying mucosa appeared undisturbed. Scans were reviewed in the light of operative findings; coronal and sagittal images were found to be reconstructions. Directly acquired coronal computed tomography, undertaken three weeks after surgery, demonstrated a complete bony plate in the right posterior ethmoid at the site previously identified as dehiscent.

Discussion and conclusion: We speculate that the posterior ethmoid defect was actually an artefact of reconstruction. We cannot exclude the alternative possibility of remineralisation, but given the time frame this seems unlikely. This case highlights the need for caution when interpreting reconstructed images of the thin bony plates of the skull base and lamina papyracea, as regards both clinical significance and medicolegal reporting. While virtual defects have been reported in the superior semicircular canals as a result of reconstructed images, we believe this to be the first reported case demonstrating a similar problem in the anterior skull base.

Key words: Paranasal Sinuses; Computed Tomography; Complications; Meningitis

Introduction

Intracranial complications are recognised as rare, but serious, sequelae of endoscopic sinus surgery. A study of a large, prospective series of patients undergoing a wide range of sinus procedures and nasal polypectomy estimated the incidence of cerebrospinal fluid (CSF) rhinorrhoea to be 0.04 per cent.¹ Intra-operative recognition of anterior skull base damage may allow immediate repair; however, associated CSF rhinorrhoea may be overlooked in the presence of haemorrhage and saline irrigation, and may present at a later stage with ongoing rhinorrhoea or meningitis.

Case report

A 56-year-old woman was referred from a district general hospital after previously undergoing elective functional endoscopic sinus surgery (FESS). The operation had appeared to proceed without complication, and the patient had been discharged after an overnight stay. However, over the following 48 hours she had complained of increasing headache, and had been readmitted. Following computed tomography (CT), a diagnosis of meningitis had been confirmed by lumbar puncture. Microbial culture had grown *Haemophilus influenzae* sensitive to amoxicillin.

The original CT scan demonstrated a significant defect of the skull base in the right posterior ethmoid, clearly visible on both coronal and sagittal sections (Figure 1a and 1b). There appeared to be a second, smaller defect at the root of the right middle turbinate.

At the point of referral, although the patient had made a full recovery from meningitis, she reported daily headaches together with clear, salty rhinorrhoea.

The patient's nasal discharge tested positive for B2-transferrin, confirming the presence of CSF.

The CT scans from the referring hospital had been acquired on a Phillips spiral CT scanner, with 0.8 mm axial sections; copies had been sent to us as compact disk files.

The patient was taken to the operating theatre for exploration of the skull base, six weeks after the initial surgical procedure. Intra-operative fluorescein was thought to be unnecessary, as the site of the defect appeared obvious on CT; moreover, its use requires lumbar puncture and is associated with potential complications. However, intra-operative findings demonstrated that not only was the skull base intact in the posterior ethmoid area identified on the scan, but that the overlying mucosa appeared undisturbed.

Residual ethmoid cells were opened, exposing the entire skull base, and both senior authors carefully palpated the skull base with a blunt probe. Images were reviewed to confirm correct orientation, and the contralateral side was also examined. Although no CSF leak was identified, a mucosal graft was applied to the root of the middle turbinate, where a second defect appeared on the scan.

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CLINICAL RECORD



FIG. 1

Coronal and sagittal images aquired before (a and b, respectively) and after (c and d, respectively) surgical exploration of the skull base.

The patient was discharged the following day, without complication.

We reviewed the original CT scans again in the light of the operative findings, and found that the coronal images were reconstructions.

Repeated, directly acquired coronal CT scanning was undertaken three weeks after surgery. This demonstrated a complete bony plate in the right posterior ethmoid (Figure 1c and 1d).

At the time of writing, the patient no longer reported rhinorrhoea, and her headaches had improved.

Discussion

Cerebrospinal fluid leakage results from a fistulous communication between the intracranial and nasal cavities, via dural and osseous defects within the skull base. This is recognised as a rare but serious complication of endoscopic sinus surgery, and is associated with a significant risk of developing meningitis.² The most common location for an iatrogenic CSF leak is at the lateral cribriform lamella, at the site of entry of the anterior ethmoid artery, followed by the posterior ethmoids and the posterior aspect of the frontal recess.³

Confirmation of CSF leakage, followed by localisation, usually precedes surgical intervention. The presence of B2-transferrin or B trace protein within suspected CSF rhinor-rhoea fluid confirms an active CSF leak.⁴ Imaging studies may help identify the site of the leak. A variety of methods are available, including nuclear medicine, cross-sectional CT, magnetic resonance techniques and radionuclide cister-nography.⁵ The accuracy of several of these techniques

diminishes when the CSF leak is intermittent, a frequent occurrence. Computed tomography has become the mainstay of CSF rhinorrhoea investigation, due to its ability to demonstrate osseous defects. Intra-operative intrathecal fluorescein studies can help localise the site of the leak, but are not always necessary.

In our patient's case, rhinorrhoea tested positive for B2transferrin. The patient's clear history of meningitis following FESS, and CT images reported by two independent radiologists (one with a specialist interest in ENT radiology, from our own department) as demonstrating an obvious skull base defect in the right posterior ethmoid, were both thought to obviate the use of fluorescein. We were very surprised by the intra-operative findings: not only was the skull base in the right posterior ethmoid intact on bony palpation, but the mucosa appeared undisturbed.

We speculate that the displayed defect in the right posterior ethmoid was actually an artefact of reconstruction, rather than a true bony defect. We cannot exclude the alternative possibility of remineralisation; however, given the time frame we believe this to be unlikely.

Can reconstructed computed tomography images create a virtual osseous defect?

Computed tomography images are often acquired in an axial plane, then coronal and sagittal images produced by threedimensional reconstruction. This reduces radiation exposure, improves patient comfort (as direct coronal acquisition requires the patient to assume a hyper-extended position) and is compatible with image guidance equipment protocols.⁶ Advances in imaging technology have allowed these reconstructed images to be of comparable quality to directly acquired images in most situations.

However, when imaging thin bony plates, such as the lamina papyracea or anterior skull base, it is possible that reconstructed images may provide insufficient clarity. Nonlinear volume averaging with adjacent soft tissues may create a virtual osseous defect. This has been described for mastoid imaging, where false positive reports of lateral semicircular canal defects have been reported.⁷ We found no previously published reports of false positive defects in the anterior skull base; however, this is probably due to such findings being thought to be inconsistent with the clinical history, and consequently viewed as artefacts.

- The presented case illustrates the dangers of using reconstructed computed tomography (CT) images to evaluate fine osseous structures in the anterior skull base
- High resolution, directly acquired, fine-cut CTs are needed to adequately define skull base defects
- Radiological investigations for patients referred from other centres should be reviewed similarly to histological specimens, and repeated if thought to be unreliable

One retrospective study of 19 patients undergoing endoscopic CSF leak repair found that high resolution, reconstructed, coronal CT images accurately identified the site of the leak in 91 per cent of cases.8 There were no false positives. Another study, of 64 patients, directly compared the adequacy of directly acquired and reconstructed images of the paranasal sinuses, including the skull base; no difference was found when using 0.625 mm reformatted images, but 1 mm reconstructions were found to be inferior to directly acquired images.⁶ Again, no false positives were reported. However, a similar study, of 52 patients (albeit published more than a decade ago), did detect statistically significant differences in the diagnostic quality of skull base resolution in reconstructed images, compared with directly acquired sinus images, attributable to step artefacts and poor resolution of thin osseous lamellae, even though thin slice helical CT had been performed.⁹

In our patient's case, the original CT images had been acquired at another hospital, but were reviewed by a senior radiologist in our unit prior to surgery and thought to be of high quality, with fine cuts. There was no suggestion that repeated imaging was required prior to surgery – indeed, this was advised against by our radiology department, due to the risk of additional radiation exposure.

Can skull base osseous defects remineralise?

Remineralisation of skull base defects has been reported following the development of lytic bony lesions and destructive infections, and after radiotherapy for malignant lesions of the skull base.

However, we could find no reports of bony remineralisation occurring within the short time frame involved in our patient's case; indeed, it has been suggested that long intervals are required.¹⁰ Reports of remineralisation of the anterior skull base have involved repeated scans taken six to nine months after discovery of the original defect.¹¹ If this is the average length of time required, it would seem almost impossible for there to be significant, much less complete, remineralisation of our patient's 'osseous defect' in just six to nine weeks.

This belief is supported by our intra-operative discovery of undisturbed mucosa. Mucosalisation of a regenerating skull base defect would occur by migration from the remaining adjacent mucosa. At such an early stage of repair, the mucosa would be expected to appear abnormal.

Therefore, although remineralisation cannot be excluded entirely, we believe it to be unlikely in our patient's case.

Conclusion

In the presented case, the true diagnosis – artefact versus remineralised defect – will never be known. However, this case highlights the need for caution when interpreting reconstructed images of the thin bony plates of the skull base and lamina papyracea, both in terms of clinical significance and medicolegal reporting. While, for most inflammatory conditions of the paranasal sinuses, reconstructed images are comparable to directly acquired images, there is potential for error when viewing the skull base. If doubt exists, high resolution, directly acquired images may confirm continuity of the bony margins.

In the presented case, the original CT images were felt to be of adequate quality. The patient had developed meningitis, and the presence of CSF rhinorrhoea was confirmed. As the visualised defect was in keeping with the clinical history, and plausible in light of the patient's original surgery, we did not question the findings.

What might we have done differently? If the location of the leak had not been so apparent, it is likely that we would have undertaken fluorescein studies preoperatively. However, we had also identified a second possible defect, and repair of this has stopped further CSF rhinorrhoea.

Although the patient did not suffer any adverse effects from the possible imaging artefact, we believe this case has sufficient merit to inform other surgeons dealing with similar conditions.

When accepting patients from other centres, we suggest that radiological investigations should be reviewed in the same way as histological specimens, and repeated if thought to be unreliable.

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