

Case reports

Sphenoid sinus mucocele as an unusual differential diagnosis in diving injuries

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Key words

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Abstract

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Sphenoid sinus mucocele is a rare cystic lesion. It grows gradually and causes visual disturbances, ocular motility abnormalities and headache due to cavernous sinus compression. Sudden change in sinus cavity volume by a barotrauma may compress a mucocele and precipitate symptoms that may easily be confused with decompression sickness. A diver suffering from vertigo, nausea, blurry vision and hearing loss following uneventful dives is presented in this report. He underwent hyperbaric oxygen treatment for inner ear decompression sickness but later was diagnosed as sphenoid sinus mucocele. A high index of suspicion is necessary to capture rare conditions like mucocele in the differential diagnosis for divers with symptoms suggesting vestibulocochlear origin. To our knowledge, only one sphenoid sinus mucocele case presenting as a diving injury has been previously reported.

Introduction

Diagnosis of decompression sickness (DCS) depends mainly on dive history and physical examination. Vestibular symptoms and hearing loss after a dive may suggest inner ear DCS however they are not specific to it. Similar symptoms may be encountered in other dive related injuries like inner ear barotrauma (IEB) or arterial gas embolism (AGE) as well as totally nonrelated diseases like sensorineural hearing loss, so they may be misleading. Differential diagnosis through comprehensive history taking, examination and sometimes imaging methods is essential for accurate and timely treatment.

Mucocele is a rare cystic lesion that may precipitate conditions with these symptoms.¹ We present a patient who underwent hyperbaric oxygen therapy for inner ear DCS but was later diagnosed with a sphenoid sinus mucocele.

Case report

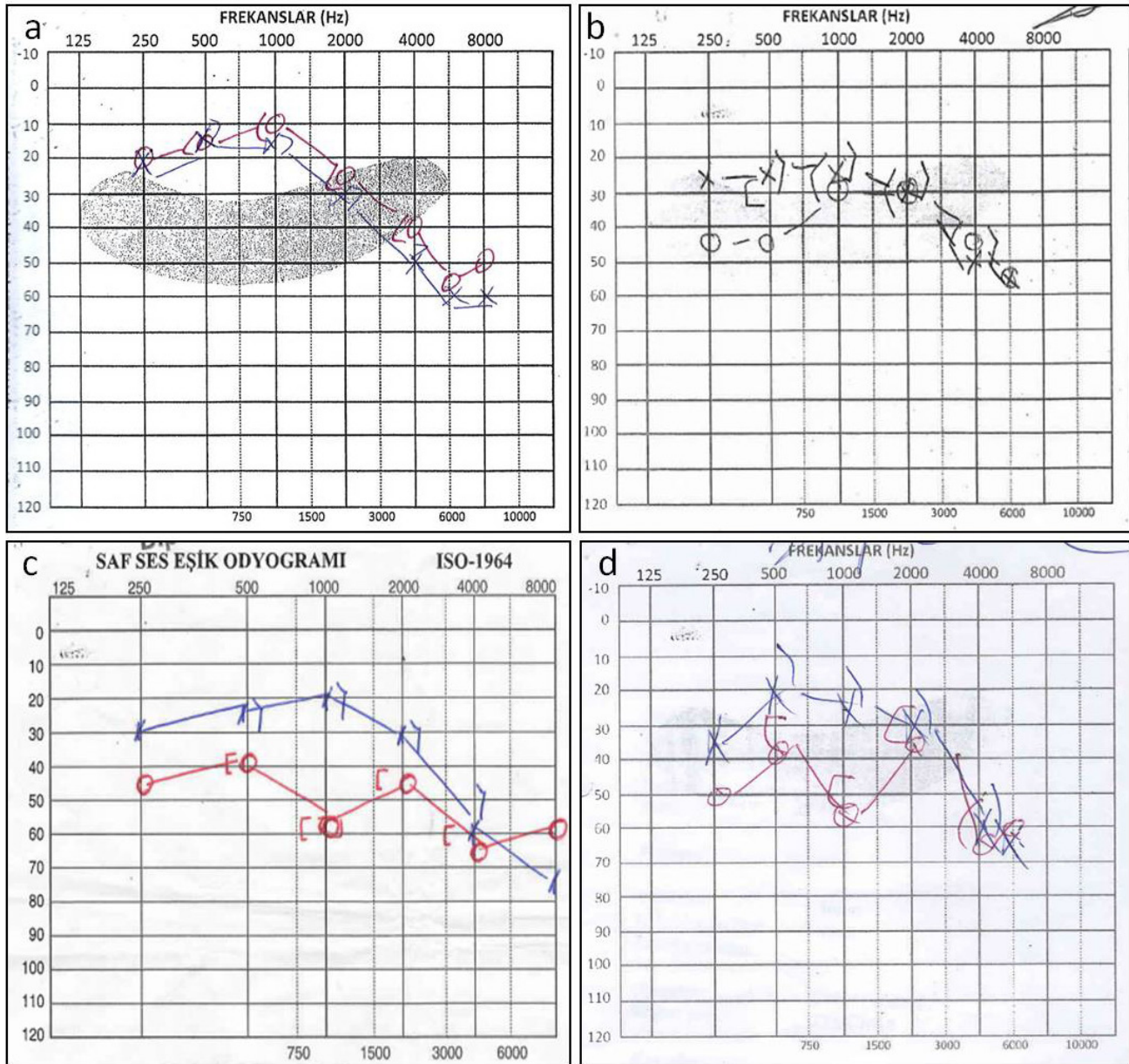
The patient was a 59 year-old male professional diver with over 2,000 dives in 20 years. He was trained to CMAS instructor level but had been working as a harvester recently. He had been assessed for fitness to dive twice previously in our department and was known to have well controlled type 2 diabetes mellitus, mild hypertension and bilateral high frequency hearing loss. (Figure 1a).

In his first incident, he presented to the emergency department of a local hospital with blurry vision, vertigo and headache followed by vomiting, hearing loss and tinnitus after a dive to 32 metres' seawater (msw) for 35 minutes with decompression stops at 12, 9, and 6 msw for 5 min, and a shallower 'safety stop'. He didn't report difficulty in equalising or physical effort during and after the dive. He was referred to a local hyperbaric oxygen treatment (HBOT) unit with a diagnosis of DCS and was treated with US Navy Treatment Table 5 (TT5) 10 hours after the incident. He reported residual visual and hearing complaints after HBOT but no further treatment was planned.

Two days later, he re-presented as his symptoms persisted. It was thought idiopathic sudden sensorineural hearing loss (ISSHL) had developed in addition to DCS so he was prescribed oral steroids and again referred for HBOT. For unclear reasons he did not present to an HBO center for a week. Upon his arrival to our department on the tenth day after the incident he had horizontal nystagmus and a positive Romberg test. The audiogram revealed a pure tone average of 50dB and 30dB for the right and left ears respectively. His tympanic membranes were intact. He was seen by the Ear, Nose and Throat (ENT) department where ISSHL was ruled out and he was administered intratympanic steroid injections for endolymphatic hydrops. Ten days later, his hearing had improved (Figure 1b) and his other symptoms had regressed. Afterwards he was lost to follow up.

Figure 1

Audiograms: (a) during fitness to dive assessment; (b) after the first incident, hearing is almost back to baseline; (c) second incident, moderate decrease in hearing threshold (both air and bone conduction); and (d) when HBO was stopped, minimal change from the previous test. Red is right ear and blue is left ear. 'o' and 'x' show right and left air conduction respectively. '>' or ']' and '<' or '[' show right and left bone conduction respectively

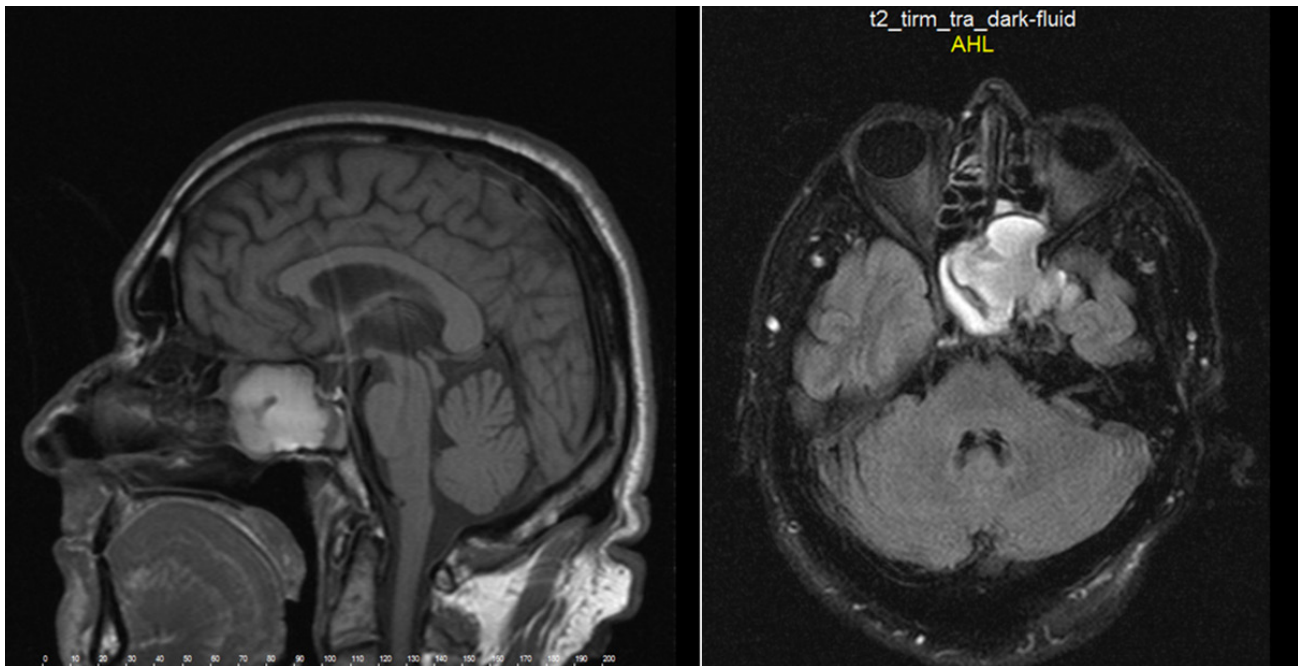


A year later he presented to an emergency department again with blurry vision, vomiting and stomach ache and hearing loss that occurred approximately 10 minutes after surfacing from a dive. He had performed two 75-minute dives to 25 msw with a 4.5 h surface interval. Dive profiles were the same; 40 minutes of bottom time, 10-minute stops at 12, 9 and 6 msw and a shallower safety stop. He was administered 100% oxygen and IV fluids and discharged when he felt better but again symptoms did not resolve completely.

The following day he was evaluated in the ENT department where he was diagnosed with ISSHL and referred for

HBOT. Again, for an unknown reason he presented to our department 10 days after the referral and exhibited vision problems, imbalance, hearing loss and dizziness. He reported that he had experienced similar symptoms four times since the first incident and only after diving. Physical examination revealed bilateral horizontal nystagmus, steady walking and dysdiadochokinesis. Otoloscopic examination was normal. There was a significant deterioration in the mid-range audiometric thresholds in the right ear, compared with the audiogram recorded after the previous incident (Figure 1c). Inner ear DCS was considered the most likely diagnosis.

Figure 2
Cranial MRI showing sphenoid mucocele; sagittal (left) and axial sections



After extensive discussions about treatment choice he underwent US Navy TT6 but didn't improve. It was decided to continue HBOT with daily US Navy TT9 applications and the patient was hospitalised to investigate further for right-to-left shunting, arterial gas embolism (AGE) and possible other causes. There was no evidence of patent foramen ovale (PFO) in transoesophageal echocardiography (TOE) performed with bubble contrast and Valsalva provocation. High resolution computerised tomography (HRCT) of the chest failed to demonstrate any lesions predisposing to pulmonary barotrauma. His ophthalmological examination was unremarkable. In electronystagmography, which was offered by the neurology department, bilateral vestibular hypofunction was observed with caloric test. Finally, cranial magnetic resonance imaging (MRI) revealed a large cystic mass in the sphenoid sinus. It had eroded the temporal bone and was compressing the cavernous sinus. Paranasal CT confirmed the MRI findings but also showed that the left optic nerve was compressed (Figure 2). The lesion was identified as a sphenoid sinus mucocele (SSM) and the hearing deficit was thought to be unrelated. HBOT was stopped. At this time the patient's complaints had not changed and there was a minimal change in hearing. (Figure 1d) Endoscopic drainage was performed by the ENT department. All symptoms except hearing loss resolved after surgery.

Discussion

Mucoceles are rare cystic lesions of the sinuses that are thought to arise following obstruction of the sinus ostium.² Although benign, they can be very destructive due to

their expansile properties.³ Resorption and/or erosion of sinus walls are possible and further expansion may result in compression of adjacent structures. SSM, comprising only 1–2% of all mucoceles, are in close proximity to the cavernous sinus which contains the internal carotid artery, optic chiasm and the 3rd, 4th and 6th cranial nerves. An expansion in a sphenoid mucocele can therefore cause symptoms related to compression of these nerves and vessels. Sometimes inflammation of these structures may supervene.⁴ Headache, visual disturbances, ocular motility abnormalities, endocrine dysfunction and nasal congestion are common symptoms.^{5–8}

Generally, a mucocele grows gradually within the sinus so acute onset of symptoms is unexpected. However, barotrauma of the sinus containing the mucocele can precipitate symptoms. There may be remaining air filled spaces in the sinus until the mucocele fully fills the cavity, but drainage is blocked leaving the sinus prone to barotrauma.⁹ It is probable that our patient had barotrauma. Thus, mucosal edema and bleeding or volume change in the sinus resulted in displacement and compression of the mucocele and consequently the cavernous sinus. Nystagmus and ataxia which are rarely reported in SSM were probably due to unilateral dysfunction of oculomotor nerves. Non-synchronized movements of the eyes are known to produce such symptoms.¹⁰ Interestingly, symptoms became evident after a short lag in both incidents probably because compression developed over time. Furthermore, barotrauma explains why the patient was symptom free in his daily life but had attacks only after diving.

The patient also complained of hearing loss in his incidents however only a moderate decrease in hearing thresholds (both in air and bone conduction) was shown. In fact, a gradual decline especially in higher frequencies was already present. In addition, hearing loss was greater in the right ear during the second incident even though the mucocele was left sided. Therefore, this was considered to be an independent sensorineural hearing loss unrelated to the mucocele. Apparently, the patient did not report his baseline hearing deficit in other hospitals so was diagnosed with ISSHL in both incidents. Yet, ear MRI was not performed for this patient at the diagnosing centres although it is recommended in treatment guidelines to evaluate retrocochlear pathologies.¹¹ The mucocele could have been identified earlier if MRI had been performed.

Our patient presented with symptoms that suggested inner ear DCS. For many years, inner ear DCS was thought to occur primarily after deep mixed gas diving.¹² Later, however, it was seen to be possible following repetitive diving and even recreational air dives.^{13,14} Recent studies have shown that right-to-left shunting which may predispose to certain forms of DCS after less provocative dives was prevalent in inner ear DCS.^{15,16} Our patient had performed air dives within safe decompression limits and surface intervals, so PFO was thought to be a possible risk factor. However, TOE performed with bubble contrast and provocation manoeuvres was completely normal.

Similar symptoms may also be seen in AGE. For example, the presence of visual symptoms could be suggestive of AGE, however the patient did not report rapid ascent or breath holding during ascent. Nevertheless, pulmonary barotrauma can develop despite controlled ascent and correct exhalation especially in the presence of predisposing lung lesions.¹⁷ In this case HRCT did not reveal any lung pathology.

IEB is another diving injury that can present with vestibulocochlear symptoms. It may occur with a forceful Valsalva or a spontaneous opening of Eustachian tube after middle ear equalisation fails.¹⁸ IEB was unlikely in this case as the patient didn't report any difficulty in equalising during any of his dives. Additionally, in the case of an IEB, compression in the chamber could cause deterioration which was not seen. The finding of the mucocele provided a plausible explanation for all the symptoms independent of inner ear pathology, with the exception of the hearing loss.

To our knowledge, this is the second reported sinus mucocele case presenting as a diving injury. A diver presenting with acute vision loss, retrobulbar pain and headache after an uneventful dive was reported previously.⁹ Contrary to our case, his symptoms were less suggestive of DCS but both divers had relatively unexpected visual symptoms in common. Sinus mucoceles may be considered if visual disturbances occurring after a dive don't associate with other

symptoms or the dive profile.

Conclusion

Initial presentation of this patient suggested DCS in both incidents but SSM was found to be responsible for visual disturbances, vertigo and nausea. Hearing loss, on the other hand, was thought to be unrelated. SSM may precipitate symptoms that can easily be confused with other diving related injuries. A high index of suspicion is necessary to include it in differential diagnosis when symptoms suggesting vestibulocochlear origin are accompanied by visual disturbances.

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