Case report

Middle ear barotrauma causing transient facial nerve paralysis after scuba diving

Matthew Lee Carmichael and K Paul Boyev

Abstract

(Carmichael ML, Boyev KP. Middle ear barotrauma causing transient facial nerve paralysis after scuba diving. *Diving and Hyperbaric Medicine*. 2016 September;46(4):260-261.)
Middle ear barotrauma is a well known entity with typical injury occurring when diving or ascending in a commercial jetliner. Patients often present with symptoms of acute onset otalgia, hearing loss and sometimes haemotympanum (with or without tympanic membrane perforation). On rare occasions, facial nerve paralysis can occur when the tympanic segment of the facial nerve is dehiscent within the middle ear. We present a case of spontaneously resolving facial nerve palsy associated with middle ear barotrauma following a brief, shallow dive. Prompt and astute diagnosis leads to proper management with simple myringotomy and can prevent unnecessary testing and other misguided treatments.

Key words
Injuries; ENT; Diving incidents; Case reports

Introduction

With the increase in recreational diving, there has been a concomitant increase in reported diving injuries.[1] In particular, the middle ear is a frequent site of injury. Typical injury occurs during descent when the diver fails to actively open the normally closed Eustachian tube (ET) to equalize pressure across the tympanic membrane (TM). If a diver fails to do this, the surrounding hyperbaric pressure forces blood and tissue fluid into air-filled spaces (middle ear) until ambient pressure is equalized. As most divers will report, the greatest change in pressure is within the first 10 metres and if equalization of middle ear pressure does not occur within this depth, injury is likely to occur. In general, middle ear barotrauma (MEBt) presents as acute onset otalgia, subjective hearing loss and at times haemotympanum (with or without TM perforation). This typically resolves spontaneously and without sequelae. However, facial nerve paralysis has been reported as a rare complication of MEBt. Herein, we report a case of transient facial nerve paralysis following a brief, shallow dive.

Case report

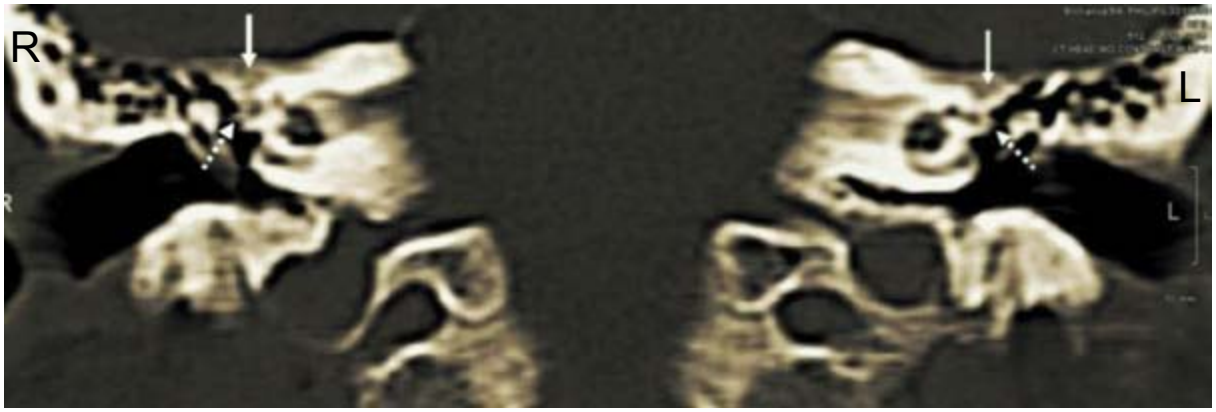
A physically fit, 37-year-old, male commercial diver presented to the emergency room (ER) with right facial droop and oral incompetence. Approximately 45 minutes prior to presenting, the patient reported diving to a depth of 3–4 metres' sea water for five minutes. He reported onset of right ear pain on decent; however, he continued the dive. Upon exiting the water, symptoms of right-sided otalgia, tinnitus, decreased hearing and a "*tin*" taste began abruptly. On reaching the dive shop, his wife noticed he had developed right facial droop and brought him immediately to the ER. At presentation, he had complete right-sided facial paralysis and right haemoptympanum with a bulging TM. He denied ever having previous difficulty equalizing pressure during dives, but could not remember if he had adequately cleared his ears on this dive. A CT head was obtained to rule out any acute cerebral events. Whilst being placed onto the scanner table, he reported "*finally able to clear my ears*" and immediately noted his symptoms abating. Subsequent examination revealed complete resolution of right facial paralysis and of the right-sided haemotympanum with an intact TM. The CT showed no evidence of acute hemorrhagic stroke and no middle ear effusion but showed a thin to absent bony covering of the tympanic segment of the facial nerve on the right as compared to the left (Figure 1). MRI was subsequently performed and was without evidence of acute cerebral infarction. Total time of onset to resolution of symptoms was within two hours.

Despite multiple attempts to obtain permission to report his case, the diver was lost to follow-up.

Discussion

Transient facial nerve paralysis is a recognized but rare complication of MEBt. It manifests as a lower motor neuron lesion with corresponding complete loss of ipsilateral facial tone, as compared to an upper motor neuron lesion causing forehead-sparing facial paralysis, as seen in cerebral vascular accidents. Barotrauma in scuba diving results from the interaction between the internal physiologic pressure of the middle ear and the pressure levels exerted by water surrounding the diver.[2] The ability to ventilate the middle ear space via the ET is essential, and, if not done early and frequently enough upon diving, the ET becomes ineffective because it irreversibly blocks at pressure differentials of

Figure 1
CT head without contrast; solid arrows indicate the geniculate ganglion of the facial nerve; dashed arrows identify the anterior tympanic segment of the facial nerve as it begins its course within the middle ear space; note the thin bony covering of the anterior tympanic segment of the facial nerve on the left as compared to the right where only soft tissue (facial nerve) is identifiable



approximately 90 mm Hg; equivalent to 1.5 metres' depth.[2]

For MEBt to cause facial nerve paralysis, the facial nerve must be compromised within the middle ear. During its course through the temporal bone, the tympanic segment of the facial nerve is found within the middle ear space typically covered by a very thin bony wall called the fallopian canal. However, increasing evidence suggests a large portion of the adult population has a dehiscence within this canal, exposing the facial nerve to middle ear pathology. In a 1971 anatomical study, 55% of normal adult temporal bones showed dehiscence of the facial nerve canal.[3] In a 2004 study, 29.3% of adult temporal bones revealed facial nerve canal dehiscence; a much higher incidence than intraoperative findings suggest.[4] The theory of ischaemic compression of the facial nerve resulting in neuropraxia was confirmed in a study using guinea pigs.[5] As middle ear pressure was increased, blood pressure to the TM and dehiscent facial nerve decreased as compared to monitored blood pressure within the femoral artery. When middle ear pressure exceeds the capillary blood pressure, blood flow to the facial nerve will decrease and induce ischaemic neuropraxia when facial nerve canal dehiscence is present.[5]

Although an experienced commercial diver, we believe this man neglected to equalize adequately the pressure within his middle ears. In doing so, he developed significant MEBt of the right ear. The subsequent middle ear pressure was sufficient to overcome the capillary blood pressure to the facial nerve causing neuropraxia. Once the patient was able to equalize the pressure in his right ear (approximately one to two hours after onset of facial paralysis), his symptoms and facial paralysis resolved.

In conclusion, the tympanic segment of the facial nerve is vulnerable to injury when a dehiscence occurs within the fallopian canal. As such, middle ear pathology has the potential for injury to the nerve. Our case represents the rare occasion when middle ear pathology can lead to potentially serious symptoms. When confronted with this rare phenomenon, careful history and physical examination of the TM is of utmost importance as patients can endure unnecessary diagnostic testing and misguided treatments. Recognition of the conglomeration of symptoms, physical exam findings and historical facts will lead to this astute diagnosis. By and large, treatment is focused on relieving middle ear overpressure. If the patient is unable to equalize pressure through opening of the ET (as was eventually done by our patient) then simple myringotomy and evacuation of the middle ear space is performed with curative intent.[6]

References

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Submitted: 22 June 2016; revised 02 August and 15 October 2016
Accepted: 15 October 2016

Matthew Lee Carmichael, K Paul Boyev
Department of Otolaryngology-Head and Neck Surgery, The Morsani College of Medicine, University of South Florida, Tampa, Florida

Address for correspondence:
M L Carmichael, MD
12901 Bruce B Downs Blvd, MDC 73
Tampa, Florida 33612, USA
mcarmic1@health.usf.edu