Iatrogenic benign paroxysmal positional vertigo: review and personal experience in dental and maxillo-facial surgery

Vertigine parossistica posizionale benigna iatrogena: revisione della letteratura ed esperienza personale in chirurgia odontoiatrica e maxillo-facciale

G. Chiarella, G. Leopardi, L. De Fazio, R. Chiarella, C. Cassandro, E. Cassandro
Chair of Audiology and Phoniatrics, Regional Centre for Cochlear Implants and Otorhinolaryngologic Disorders, Dept. Experimental and Clinical Medicine “G. Salvatore”, “Magna Graecia” University, Catanzaro; 1 ENT Unit, USL 11, Empoli; 2 Dept. Maxillo-facial Surgery, University of Perugia, Perugia, Italy

SUMMARY

The post-traumatic origin of benign paroxysmal positional vertigo remains the most likely, from a patho-physiologic point of view. Benign paroxysmal positional vertigo due to surgical “traumas” has been described in the medical literature. According to personal experience, these iatrogenic cases represent a rare possibility and may be the consequence of surgical interventions differing according to the anatomical district involved and surgical technique performed. The temporal relationship with the surgical action and clinical features may be involved in some of these cases, even if it is not possible to define any real cause-effect link. Herewith some cases of paroxysmal positional vertigo are described, strongly held to be of iatrogenic origin, focusing on dental and maxillo-facial surgery as risk factors for benign paroxysmal positional vertigo.

KEY WORDS: Benign paroxysmal positional vertigo • Aetiology • Surgical trauma • Dental surgery

Introduc­tion

The traumatic origin of benign paroxysmal positional vertigo (BPPV), representing the most frequent cause of labyrinthine vertigo 1 2, appears to be the most likely, from a patho-physiologic point of view. Cervical or head traumas lead to the separation of otoconial debris from the macula and the possible sedimentation inside one of the semicircular canals. Shifting of these particles, due to the movements of the head on the neck, produces an endo-lymphatic stream which stimulates the ampullar receptors resulting in typical BPPV symptomatology 3 4. Post-whiplash trauma or any head traumas due to road accidents are often the main cause of this disease. On the other hand, various descriptions of post-trauma BPPV associated with “surgical trauma” have been reported in the literature 5 8. According to our experience, there is a possibility of BPPV following surgical procedures on the ear and so-called “iatrogenic” cases are not frequent (4.2% of stapes surgery performed in our Unit). Furthermore, they can occur following different surgical interventions, depending on the anatomical district and the surgical technique. In particular circumstances, such complications are possible, for instance in the case of dental and maxillo-facial surgery. Moreover, dental and maxillo-facial surgery seems to be one of the most frequent causes of iatrogenic BPPV due to the anatomical features of the districts involved and traumatic potential of the surgical technique. This hypothesis is strongly supported by the temporal relationship with the surgical action and by clinical features, although it is not possible to define any real cause-effect link. In our opinion, this possibility leads to the hypothesis of BPPV being a possible, although not frequent, complication of surgical interventions, such as dental or maxillo-facial surgery.
Review of the literature

Few cases of BPPV, following orthodontic treatment or dental or maxillo-facial surgery, have been reported in the International literature. Perez Garrigues et al. 9 reported a case of BPPV following orthodontic surgical intervention on the superior maxilla. The case described by Kaplan et al. 10 is particularly interesting: bilateral posterior semicircular canal (PSC) pathology following dental implant treatment. In fact, the bilateral involvement remains, also in this field, a rather rare occurrence. Andaz, Whitsett and Ludman 11 reported a case of BPPV subsequent to neurosurgical removal of a parietal osteoma, also describing the traumatic role of the removal by hammer and chisel, even if not in a proximal area. Nigam et al. 7 agree with this hypothesis: in fact, they describe a case of BPPV following maxillo-facial surgery for the removal of a voluminous carcinoma of the superior maxilla, with implantation of a maxillary prosthesis. Flanagan 12 describes the onset of BPPV associated with osteotomy on account of multiple dental implants, suggesting caution for instruments and techniques to prevent this complication. Galli et al. 13 reported another case of BPPV in a 55-year-old patient submitted to oral implant surgery in the 2.3 area. Saleni et al. 14 have described two cases of BPPV following aesthetic surgery on the nose.

Personal experience

For this study, we selected BPPV cases, from those observed between January 2003 and September 2005, in which the onset of symptoms occurred immediately after dental or maxillo-facial surgery. Patients with surgical treatment of previous traumas were excluded, as well as those affected by BPPV, diagnosed more that 7 days after the surgical treatment or affected by another concomitant or previous disorders of the posterior labyrinth.

Moreover, we have excluded patients with BPPV risk indicators (dyslipidaemia, high cholesterol levels, vascular problems, endocrinological disorders, peri-menopausal age, cranial trauma, neurologic disorders, migraine) 4 as well as males over the age of 45 years and females over 40.

The diagnosis of BPPV has been made, under video-oculoscropy control, using the typical positioning manoeuvres Dix Hallpike’s 15 for the PSC and Pagnini McClure’s (slow positioning on the sides) for the lateral semicircular canal (LSC) 16 17. The criteria used for the diagnosis were those commonly approved for this pathological condition: a paroxysmal nystagmus with brief latency, accompanied by vertigo, exhaustible, repeatable, fatigable. The features of the nystagmus, in the various positions, related to the various channels, are those described in the literature. 18

BPPV was treated with Epley’s canalith repositioning manoeuvre. 20 Vestibular functionality of all patients, at the end of the treatment, was evaluated using Fitzgerald Hallpike’s 19 caloric tests in order to exclude any concomitant posterior labyrinth disorder. Little attention was paid to the audiometric results since we did not have any evaluations of the earlier hearing situation.

Features of selected patients are outlined in Table I.

All patients underwent surgical extraction of impacted teeth through the erosion of the incarcerating bony wall with the aid of a rotating tool.

The first patient, after previous extraction of some teeth (2nd premolar, 1st-2nd right upper molars), underwent orthodontic treatment consisting of introducing a metal pin to support the prosthesis at upper-maxilla level, on the right side. Only one female showed right LSC BPPV. In all the other subjects, the posterior semicircular canals were affected.

Vestibular pathology has been recorded, in all cases, on the side submitted to surgical treatment. There was no evidence of bilateral pathology or simultaneous involvement of multiple semicircular canals. The mean onset time of this pathology was 4.1 days. The most rapid onset was reported 8 hours after surgical treatment and the most remote after 7 days.

The treatment used for PSC is a modified Epley’s repositioning manoeuvre, with checks being made after 15 minutes and within 7 days of treatment. In the only case involving the LSC, forced position on the opposite hip 21 has been selected. All other cases were resolved with the first treatment.

Follow-up of patients was scheduled for 3, 6 and 12 months after treatment: a negative clinical pattern was confirmed in all cases.

Discussion

Based on the international literature and on the strict inclusion criteria adopted, our cases are particularly interesting as far as concerns the hypothesis of a iatrogenic origin of BPPV following dental or maxillo-facial surgery.

In the cases observed by us and by other Authors, the more likely pathophysiologic mechanism is represented by an indirect trauma of the posterior labyrinth due to the use

<table>
<thead>
<tr>
<th>Age (yrs)</th>
<th>Sex</th>
<th>Pathology</th>
<th>Onset (days after surgery)</th>
<th>Affected side</th>
</tr>
</thead>
<tbody>
<tr>
<td>35</td>
<td>M</td>
<td>Implant</td>
<td>1</td>
<td>PSC ipsilateral</td>
</tr>
<tr>
<td>22</td>
<td>M</td>
<td>3.8-4.8 Dysodontiasis</td>
<td>3</td>
<td>PSC ipsilateral</td>
</tr>
<tr>
<td>24</td>
<td>M</td>
<td>4.8 Dysodontiasis</td>
<td>7</td>
<td>PSC ipsilateral</td>
</tr>
<tr>
<td>30</td>
<td>F</td>
<td>3.8 Dysodontiasis</td>
<td>4</td>
<td>PSC ipsilateral</td>
</tr>
<tr>
<td>36</td>
<td>F</td>
<td>2.8-3.8-4.8 Dysodontiasis</td>
<td>8 hours</td>
<td>PSC ipsilateral</td>
</tr>
<tr>
<td>35</td>
<td>F</td>
<td>3.8-4.8 Dysodontiasis</td>
<td>7</td>
<td>PSC ipsilateral</td>
</tr>
<tr>
<td>25</td>
<td>F</td>
<td>4.8 Jaw cyst</td>
<td>4</td>
<td>PSC ipsilateral</td>
</tr>
<tr>
<td>21</td>
<td>F</td>
<td>3.8-4.8 Dysodontiasis</td>
<td>3</td>
<td>LSC ipsilateral</td>
</tr>
</tbody>
</table>
of rotating tools or hammer and chisel on the maxilla and on bony structures in relation to the close proximity to the temporal bone. Vibrations would propagate through the bony structures themselves, up to the posterior labyrinth. At this level, mechanical energy would be transferred to the endolymphatic liquids resulting in macular trauma able to determine the otoconial separation. It is also likely that the mechanical traumas of the facial bone are propagated through preferential lines, in which the temporal bone is often involved.

Membranous structures of the inner ear, contained in hollow bones the walls of which are separated only by the perilymph, are particularly subject to traumatic lesions due to the simple propagation of a mechanical wave involving the temporal bone. Traumas, even if not particularly intense but repeated, especially if determined by rotating instruments the vibration of which is prolonged in time, can represent the causal event of the disorder. Another hypothesis includes the tilting of the head, particularly in patients who undergo maxillary surgery or in those requiring intubation during general anaesthesia. We agree on the hypothetic existence of favourable and triggering factors for BPPV and on potentially taking into consideration a multi-factorial genesis. On the other hand, idioopathic BPPV forms are probably such due to our being unable to find the exact mechanism that has provoked them. As already mentioned, post-traumatic forms are those in which it is simpler to hypothesize the pathophysiological mechanism. In our opinion, among the possible traumas to be taken into consideration, there are surgical traumas, not only involving ear surgery. It is possible to hypothesize that the surgical traumatic event acts as a triggering factor on a pre-existing substratum. But, at least in our cases, rigorously selected without risk indicators for BPPV, the cause-effect relationship appears very solid. This stresses the need to consider this complication, even if not frequent, especially in surgery involving districts in continuity or being in contiguity with the labyrinth. It should not be forgotten that BPPV and, more generally, post-trauma vertigo, represents, today, of the various ENT disorders, that which is more subject to compensation in road accidents, reaching a percent disability of approximately 2% or even 5%. Discussion on BPPV as a possible complication following dental or maxillo-facial surgery, with predictable medico-legal consequences, therefore remains open.

References