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CASE REPORT

Benign paroxysmal positional vertigo after dental surgery

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Abstract We investigated the relationship between dental and maxillofacial surgery and benign paroxysmal positional vertigo (BPPV). BPPV represents the most frequent cause of vertigo of labyrinthine origin. BPPV has been reported following surgical trauma from various surgical interventions, regarding anatomical site and technical execution. A surgical origin is, in many cases, supported by the temporal relation to the surgical intervention as well as by the clinical picture. We considered eight BPPV cases of suspected iatrogenic origin focusing our attention on dental surgery with particular reference to surgical extraction of included teeth through rotating tools. The cases taken into account had no other inner ear disease and BPPV risk indicator. We conclude that dental surgery is a risk factor for BPPV.

Keywords Benign paroxysmal positional vertigo · Dental surgery · Surgical trauma

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Introduction

Cupulolithiasis and canalithiasis BPPV represents the most frequent cause of vertigo of labyrinthine origin [12, 13]. Epidemiological studies often make reference to a post-trauma origin of this pathology [16, 17]. Cervical or head traumas can represent the decisive cause underlying the mechanisms of cupulo or canalithiasis [4, 25].

In cupulolithiasis otoconial debris in semicircular canal becomes attached to the cupula, rendering it sensitive to gravity; in canalithiasis the debris are free-floating in the endolymph: the shifting of these particles due to the movements of the head on the neck produces an endolymphatic stream which stimulates the ampullar receptors with the ensuing symptomatology typical of BPPV.

On the other side this pathology can be, more frequently, of non-traumatic origin whereas the debris, responsible for the symptoms, are not necessarily of otoconial origin but they are often simply aggregates of either fibrin or platelets [21, 28].

BPPV can be efficaciously treated by rehabilitation consisting in either liberatory or repositioning manoeuvres, which are chosen according to clinical characteristics, to health worker's experience and to patient's peculiarities [17, 26].

There are many theories on the etiology of this disease and these are continuously evolving but post-trauma origin is still now the most understandable from a physiopathologic point of view.

Post-whiplash trauma or any head trauma from road accidents are often the main cause of this disease. On the other hand, literature contains various descriptions of post-traumatic BPPV associated with ear surgery [2, 3, 5, 19].

According to our experience these so-called "iatrogenic" cases are not frequent in relation to the global incidence of

this pathology. Besides they can be following to surgical interventions very different for anatomical district and technique.

Dental surgery represents a particularly sensible sphere for this complication, and indeed, within iatrogenic cases, it seems to be, for anatomical evaluations and traumatic potential, one of the most frequent causes of BPPV. Such a theory is strongly supported by the time relation with the surgery operation as well as by the clinical picture although it isn't possible to assert any real cause-effect link. Therefore the discussion about BPPV as possible, not frequent, complication after dental surgery is still open with the possible medico-legal lapels.

Patients and methods

Our selection is relating to those BPPV cases we have personally examined, since January 2003 to the September 2005, in which vertigo symptoms have arisen immediately after a surgery operation regarding the dental-maxillofacial districts. All patients were systematically assessed for BPPV. We have been considering a group of 18 patients from 762 (2.3%) examined in our clinic, on the grounds of a very careful anamnesis.

The criteria taken into account for the selection were:

- Surgery operation regarding dental-maxillofacial districts (excluding all treatments of previous traumas).
- BPPV arisen within 7 days after surgery operation without any other contemporaneous pathology affecting posterior labyrinth
- Negative anamnesis regarding previous vertigo episodes
- No presences of BPPV risk indicators (vascular problems, endocrinological disorders, high cholesterol, dyslipidemia, perimenopausal age, cranial trauma, neurotologic pathologies, migraine) [17].
- Age under 45 for men, under 40 for women

Taking these parameters into account eight persons were excluded from this study for the following reasons: BPPV risk factors, especially of cardiovascular origin, old age and previous similar episodes so that we had come to the conclusion that a surgical operation could have been just an aggravation.

Two other cases had been excluded due to a non-valid time relation: BPPV would arise after some months from operation (in one case even after 10 months) thus making the link with surgery trauma appear dubious. Eight cases (1%) show all the characteristics to be taken into account (Table 1).

All patients have undergone surgical extraction of included teeth through the erosion of the incarcerating bony wall by aid of a rotating tool.

The first patient, after previous extraction of some teeth (2 premolar, 1–2 right upper molar) has undergone orthodontic treatment consisting in setting a metal pin in order to support the prosthesis at upper-maxillo level, on the right side.

BPPV diagnosis has been made through typical positioning manoeuvres (slow as well as rapid): by Dix Hallpike [6] for posterior semicircular canals (PSC) and by Pagnini-McClure (slow positioning on hips) for lateral semicircular canals (LSC) [18, 20, 27]. The criteria adopted for this diagnosis were those generally accepted for this pathology: observation of paroxysmal nystagmus preceded by a short latency (in posterior canal BPPV), associated with vertigo lasting for seconds. The characteristics of nystagmus in its various positions and relevant to the different canals are extensively described in literature [13, 14].

Once patients had concluded BPPV treatment, they underwent caloric tests according to Fitzgerald Hallpike [9] in order to exclude coexisting pathology of the labyrinth.

Results

Only eight cases (1%) met our strict selection criteria. Seven patients out of eight had posterior semicircular canals BPPV. Only one female patient had right LSC BPPV. On the other hand there has been no evidence of

Table 1 Characteristics of the sample examined (see text)

Age	Pathology	Onset (days after surgery)	Sex	Anesthesia	Affected canal
35	Implant	1	M	Local	PSC
22	3.8–4.8 dysodontiasis	3	M	Local	PSC
24	4.8 dysodontiasis	7	M	Local	PSC
30	3.8 dysodontiasis	4	F	Local	PSC
36	2.8–3.8–4.8 dysodontiasis	8 h	F	Local	PSC
35	3.8–4.8 dysodontiasis	7	F	Local	PSC
25	4.8 jaw cyst	4	F	General	PSC
21	3.8–4.8 dysodontiasis	3	F	Local	LSC

bilateral pathology or simultaneous affection of semicircular canals. In all cases vestibular pathology has been recorded on the side that had been treated surgically. The average onset of BPPV was 4.1 days after surgery, whereas the most rapid beginning was reported 8 h after surgical treatment and the latest after 7 days. To all cases relating to PSC, repositioning manoeuvres by Epley [8] have been applied as treatment, modified, with a check after 15 min and later on 3–7 days after treatment. The only LSC-case has been treated by forced positioning on opposite hip [27]. Just a single treatment was sufficient to solve this pathology in all patients. Patients' follow-up included checks after 3, 6, and 12 months that confirmed a negative clinical picture in all the cases examined.

Discussion

The international literature contains few BPPV cases after dental treatment or maxillo-facial surgery [1, 10, 11, 15, 19, 22–24]. In all these cases, as in our experience, it is probable that the indirect trauma on the posterior labyrinth is linked with the use of either rotating tools or hammer and scalpel on the maxilla as well as on bone structures and, in any case, sufficiently close to the temporal bone. It is supposed that vibrations are propagated throughout bone structures eventually reaching the posterior labyrinth. At this level mechanic energy would travel through endolymphatic fluids or bone eventually causing macular trauma. Some authors believe that mechanic traumas on the maxilla travel through preferential lines, after involving the temporal bone. The membranous structures of the inner ear, which are contained in bony chambers whose walls are separated by perilymph, are particularly exposed to traumatic lesions due to simply travelling of a mechanic wave, which affects the temporal bone [7]. Even mild trauma, when caused by rotating structures whose vibrations are prolonged in time, can damage semicircular canals: vibrations dissolves otoliths, which then enter canal causing BPPV. This second step may be caused by tilting of the head particularly in patients that undergo maxillary surgery or in those that need intubation during general anesthesia.

BPPV can originate from various causes and the incidence of idiopathic forms is probably overestimated due to the difficulty to single out the individual pathogenic mechanism. There is agreement about the presence of factors, which favour the evolution of BPPV thus leading to a multifactor-genesis theory. As said before, post-trauma forms are those ones in which a physiopathologic mechanism is obvious. However, in the light of what has been reported, we think that the trauma-concept must be widened, because, within all possible traumas, we must also consider surgical trauma and not only in purely otologic context.

It is also possible that surgical trauma works as a trigger factor, maybe on a pre-existing substratum, but at least in the cases reported, the cause-effect relation is unquestionable.

We think, therefore, that it is justified to consider this possible, even if not frequent, complication when operating on structures next to labyrinth, which can be relevant both for treating vertigo symptoms.

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