

Complete Binasal Hemianopsia

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Abstract

The authors describe a case study of a 21-year-old female patient suffering from a tumour in the pineal region and dorsal part of the 3rd cerebral ventricle (pineocytoma) who developed complete binasal hemianopsia 11 months after the tumour's removal. These perimetric changes did not show any progression for seven years. In discussing the question of the pathogenesis of hemianopic defects, the authors are inclined to the opinion that both of the optic nerves are compressed by the circle of Willis vessels.

Keywords: Complete binasal hemianopsia; Tumour in the pineal region and the third cerebral ventricle; Pathogenesis of binasal hemianopsia

Introduction

The existence of complete binasal hemianopsia has been disputed since the introduction of the first perimeters. In 1877, Foerster, one of its inventors, wrote about the relationship between the nervous system and the visual organ in the first edition of the Graef-Saemisch Compendium: "there is no binasal hemianopsia" [1].

After the introduction of automated static perimetry, the results of visual field changes became more precise and a large number of incomplete binasal hemianopsias were eliminated.

The occurrence of a typical case of this symptom in clinical practice is therefore even more surprising.

Case study

In November 2002, a 21-year-old nurse suddenly lost consciousness during her night shift, which was accompanied by limb cramps, without wetting or biting. A similar situation repeated itself in December 2002.

During her examination in December 2002, she was conscious, cooperative, well-oriented, with a slight bilateral ataxia and medium-grade paleocerebellar symptomatology. Her palpebral apertures were symmetrical; the eyeballs were in the central position, freely able to move sideways, without nystagm. The pupils were isocoric, reactive to light and convergent. The visus did not seem to show any disorder, she wears glasses (3.5 and 2.5 D) and the perimeter seemed to be without any disorder.

The MRI examination of the brain carried out on 16 December 2002 revealed a hypoinvasive structure in the pineal region sized 21x17x19 mm, slightly ventrocaudally compressing the quadrigeminal bodies, hyperintensive in the T2 weighing, with a narrow nonenhancing border; the ventricular system was narrow and the central line was without any overpressure (Figure 1). The pituitary stalk was localized behind the chiasm (Figure 2).

Eye examination (13 January 2003): eye finding within standard limits; excavation of the fundus with C/D ratio = 0.4; no signs of congestion. V: with -3.5, -2.5 respectively = 1.0. The visual field was not examined.

On 14 January 2003, a tumour was removed from the pineal region through suboccipital craniotomy. During the surgery, the tumour

widely adhered and pressed against the colliculi superiores et inferiores of the upper stalk burrowing into the caudal part of the 3rd ventricle. The tumour was partially cystic, some parts had brittle structure while other parts had rigid bodies. The tumour was gradually sharply separated from the base respecting its boundary against the upper stalk and was radically removed. The histological examination established a pineocytoma. The postoperative MRI of the brain (16 January 2003) confirmed the radical removal of pinealoma with no obstruction of the liquor ways.

In the postoperative period (from the 3rd postoperative day), impairment of the consciousness occurred repeatedly with no objective correlation in repeated imaging examinations (twice brain CT, once brain CT-AG). Eighteen days after the surgery the patient was released to home care. She did not appear to have any problems, apart from slight paleocerebellar symptomatology.

Outpatient follow-up

Her neurological state did not change until December 2003, including normal visus and provisionally examined perimeter. The ocular findings at the follow-up examination carried out by an ophthalmologist in April 2003 were normal, including the objectively performed perimeter examination. In 27 May 2003, the patient suffered a consciousness disorder (approx. 12 minutes), with clonic-tonic spasms and wandering motion of the eyes. During her hospitalization in the neurological department her problem did not recur. ECG, examination of the eyegrounds and laboratory examination showed no pathological findings. CT and MRI examinations proved reparative gliosis around the aqueduct, otherwise without any other pathology.

Early in December 2003, the patient experienced subjectively impaired visus. The eye examination revealed increased intraocular pressure. During her hospitalization at the eye clinic, the first scotomas

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in the nasal halves of both visual fields were found on the perimeter, further to the right. The monthly follow-up examinations of the perimeter showed that until the end of June 2004 the finding progressed gradually into complete binasal hemianopsia, having persisted until now (Figure 4). In July 2004, the patient was examined at the lead author's clinic, where the examination of electrical retinal functions did not reveal any changes in the flash or pattern electroretinograms. The last ophthalmological examination was carried out in November 2010. Both the external and intraocular findings were normal, while the binasal hemianopsia in the perimeter still persisted.

Repeated MRI examinations of the brain (annually until 2010) did not show any recurrence of the pinealoma, the reactive changes disappeared (= reactive gliosis) and the cerebral finding was normal (Figure 3).

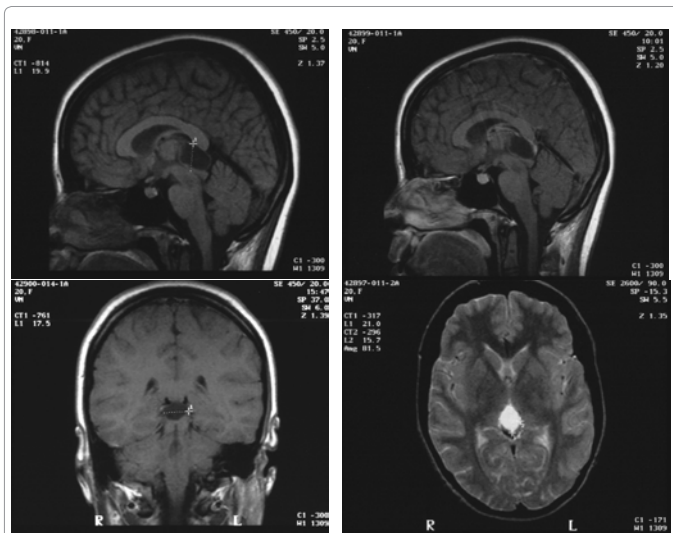


Figure 1: MRI before surgery – cystic expansion in the pineal region (T1W SAG and COR, T2W TRA) with parietal saturation after administration of Gd (T1W SAG).

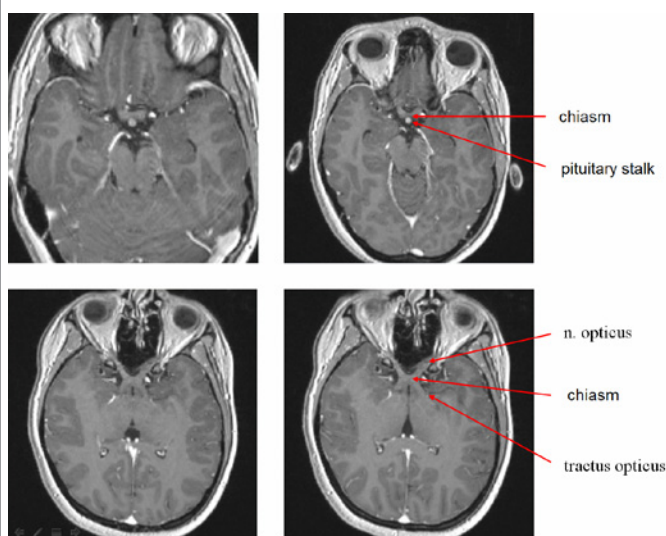


Figure 2: Relationship between pituitary stalk and optic chiasm; optic tracts.

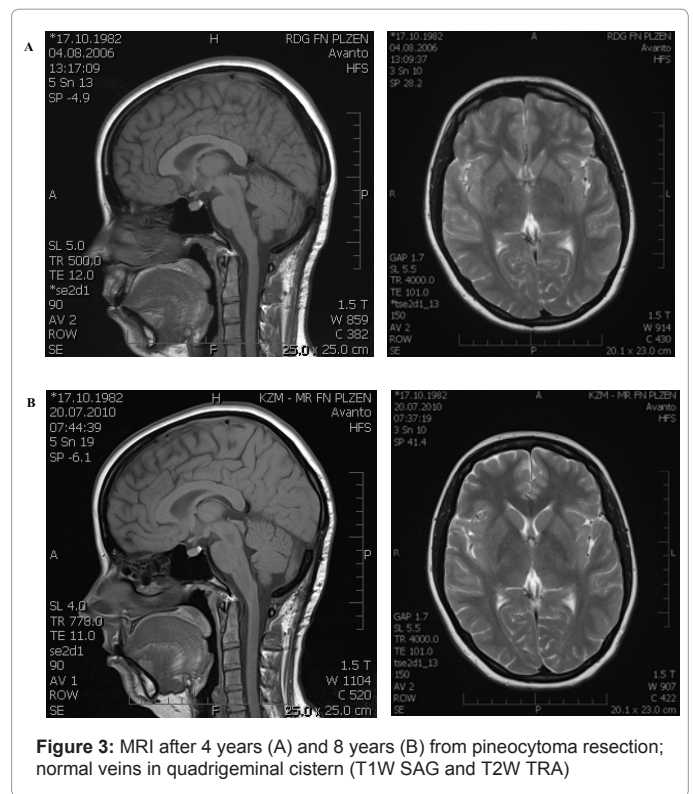


Figure 3: MRI after 4 years (A) and 8 years (B) from pineocytoma resection; normal veins in quadrigeminal cistern (T1W SAG and T2W TRA)

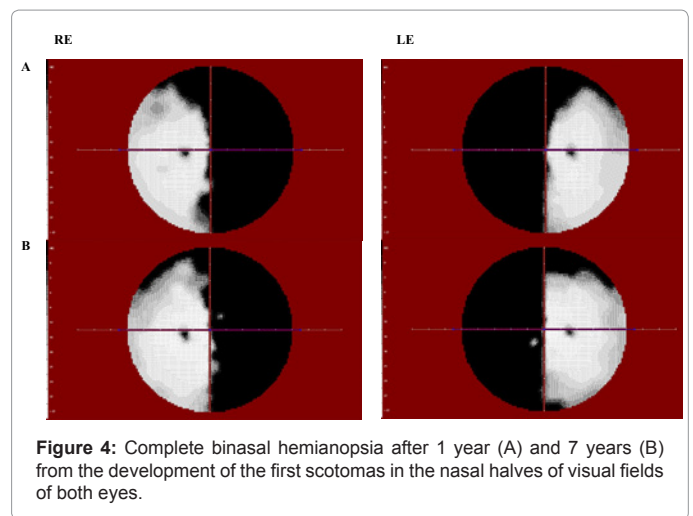


Figure 4: Complete binasal hemianopsia after 1 year (A) and 7 years (B) from the development of the first scotomas in the nasal halves of visual fields of both eyes.

Discussion

The most outstanding post-war Czechoslovak neuro-ophthalmologist, associate prof. Otradovec, wrote in “Clinical Neuro-ophthalmology” that binasal hemianopsia is a theoretical counterpart to bitemporal hemianopsia and they both represent so-called heteronymous hemianopsia. This explanation, however, does not cover the causal relationships of these defects’ development. Binasal hemianopsia, unlike its other types (bitemporal heteronymous; unilateral homonymous), is not explained by a single visual tract lesion. Binasal hemianopsia is also relatively rare and its image nearly always assumes a more or less symmetrical lesion in the optics, chiasm and optical tracts in two locations simultaneously as it can only develop from the lesions in direct, noncrossing fibres [2].

Changes in the nasal halves of the visual fields can also occur in the case of a lesion in the eye region, e.g. when corneal changes such as keratoconus [3] are involved, or retinal changes, sickle-cell anaemia, etc [4].

The antiepileptic drug vigabatrin has an impact on retinal functions and its use can result in changes in the visual fields. The prevalence of these changes is 15-31% in infants, 15% in children and 25-50% in adults. Perimetric defects begin as bilateral nasal scotomas progressing in concentric narrowing of the visual fields. The development time is 3.1 months in infants, 11 months in children and 9 months in adults [5].

Gonzales et al. [6] proved on a group of 204 epileptic patients that they had visual field constriction; 59% used vigabatrin simultaneously, 43% were patients who had stopped using the drug and 24% of patients had never used it. Electrophysiological changes in the retina were proven in 48% of the patients taking vigabatrin, in 22% of the patients who had stopped taking it and in none of the epileptic patients without a history of taking vigabatrin.

In the overview paper by Salinas-Garcia and Smith [7] we can find that out of 100 neuro-ophthalmologically revised patients, 8 patients suffered from binasal hemianopsia. Two of them had ischaemic neuropathia and there were single cases of disc druses of n. II, glaucoma, papilla pit and retinitis pigmentosa sine pigmento. This means that 75% of the binasal hemianopia cases were of intraocular origin. Only two patients had an intracranial pathology – congenital hydrocephalus.

Other possible etiological alternatives include post-infectious arachnoiditis [8] or empty sella syndrome [9]. In theory, we can also consider the fusiform, dolichoectatic, sclerotic or aneurismal dilatation of one or both of the internal carotid arteries.

Czech literature contains a report from 1962 on incomplete binasal hemianopsia by Ms. Vladykova and Mr. Vladyka [10] describing the case of a 20-year-old patient with a cystic tumour in the vicinity of the frontal horn of the lateral ventricle. Histological examination showed a proliferating ependymoma with numerous mitoses. The authors challenge the idea of compression of noncrossing fibres in the chiasm region as an explanation for the causes of visual field changes. This assertion is based on the fact that the noncrossing fibres from the temporal retinal halves nearly reach the chiasm centre and their compression would also cause changes in the temporal halves of the visual fields. Therefore, there must be another explanation focused on searching for changes in the long section of the optical nerves known as the postfixed chiasm.

According to the MRI examination, our patient did not have a postfixed chiasm and most probably pulling the optics during the period of the postoperative reparative processes caused the compression of their external parts against the anterior vessels of the Circle of Willis. The finding has been stabilized and with no progression for seven years since their occurrence.

In this conclusion, we are in agreement with the above-quoted authors while disagreeing with Mr. Otradovec. Due to the rare occurrence of the above-mentioned symptom, the authors recommend interdepartmental cooperation in which an ophthalmologist should exclude an ocular origin of the changes in the visual fields.

Conclusion

The authors describe complete unclear binasal hemianopsia developed nearly one year after the removal of pineocytoma from the pineal region and the 3rd cerebral ventricle.

Objective

To prove the existence of a rare, complete binasal hemianopsia based on a case study.

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