Operculum infarction and abducens nerve paresis in an infant

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INTRODUCTION

Paresis of the sixth nerve is frequently seen in the routine practice under manifold etiologies, both in adults and in children. It is nevertheless unusual that an emotional stress acts as a trigger for such a disorder in an otherwise healthy subject. The following report deals with a child who developed strabismus after a sudden state of fear. The clinical investigation disclosed an unexpected pathological finding and it is felt that such a condition deserves communication.

CASE REPORT

A thirteen month old white female was attacked by a dog on March 8, 1981 at 12:00 noon. Although the animal did not touch the child she was scared became very pale, and wept profusely for approximately two hours. After a period of agited sleep the mother noticed before dinner that the patient's eyes looked different but she could not describe what was wrong. The next morning a left eye squint was observed. No other motor and/or speech disturbances were noticed.

The patient was seen two days latter by the referring ophthalmologist and 30 esotropia was present in primary position, with a larger angle in levoversion and orthotropia in dextroversion. She fixated with the right eye and in binocular rotations a -3 left lateral rectus was noticed, with remainder normal muscles. Pupillary and fundoscopic examinations were normal. Retinoscopy under cyclopentolate + 1.00 sph. OU. Patient was put under alternating patch. A neurological assessment was normal except for the left abducens paresis.

The same ophthalmologist re-examined on May 5, 1981 and an unchanged angle was found in primary position, but with —2 left lateral rectus. A second neurological assessment was normal.

She was referred to us for evaluation on May 13, 1981 when the mother informed the deviation seemed intermittent and abduction OS had improved. Gestation had been full term, cesarean delivery, birth weight 4.1 kg, height 53 cm, postpartum period uneventful. With 6 weeks, ccphalic perimeter 38 cm, and thoracic perimeter 37 cm. Patient had bilateral otitis age 4 months. The neuropsychologic development had been normal. No epilepsy history. Patient was the product of mother's fifth pregnancy; there are two brothers and one sister in good health; there was one abortion. Two grandaunts and one granduncle from the maternal side had squint.

On examination, binocular fixation for distance revealed a constant esotropia with preference for OD but could maintain fixation with OS; for near an intermittent esotropia deviating OS.

Prism and cover test with OD fixating:

$$\text{ortho} \leftarrow \text{ET} \quad \begin{matrix} 16 \uparrow \\ 20\Delta \rightarrow 20 \\ \downarrow 12 \end{matrix}$$

E(T)'14

Binocular rotations: -1 right lateral rectus; -2 left lateral rectus.

Pupillary reflexes and fundus were normal.

Retinoscopy under 1% cyclopentolate: OD + 0.75 sph. OS + 1.25 sph.

A third neurological assessment was normal. A lumbar puncture revealed a clear cerebrospinal fluid with cytological, biocheing spontaneous sleep was within normal limical, and other tests within normal limits; cerebrospinal fluid protein electrophoresis was also normal. Electro-encephalogram durmits for the patient's age.

A pediatric assessment showed blood pressure 9 X 6 mm Hg in the upper limb and 10 X 6 mm Hg in the lower limb. The pulse of both dorsalis pedis arteries was normal. X rays skull, dorsolumbar vertebrae, long bones and hands with no signs of mucopolysaccharidoses; the Dorfman — Steiness' test was negative. Blood cells, coagulation time, bleeding time, and glycemia were within normal limits. No stool parasites.

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A computorized tomographic scan of the head (Figure 1) revealed a less dense posterior opercular area in the left side. The remainder encephalon and ventricular system were normal. After injection of 15 ml of contrast intravenously this was retained for a longer period of time in the affected area. An operculum infarction was diagnosed.

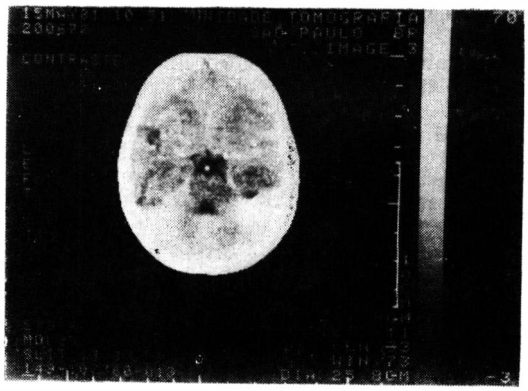

Fig. 1 — Computorized tomographic scan shows a left operculum infarction with retained contrast in the area.

No therapy was indicated.

Patient was seen again on November 10, 1981. Binocular fixation, alternating esotropia for distance and near, with preference for OD fixation; deviation was intermittent for distance and constant for near.

Prism and cover test with OD fixating:

12
↑
12 ← E(T) 14 → 22
↓
10

ET' 25

Binocular rotations: -1 right lateral rectus and left inferior oblique; -2 left lateral rectus, + 1 both medial recti.

A second computorized tomographic scan of the head was essentially unchanged as compared with the previous.

Under 1% cyclopentolate the fundus was normal and retinoscopy showed + 0.75 sph. OU. A full optical correction was prescribed and a 3 hour per day patch of the right lens indicated.

On March 19, 1982 the patient was alternating both for distance and near. Prism and cover test with glasses:

14
↑
12 ← ET 16 → 16
↓
10

ET' 33

Binocular rotations: -1 left lateral rectus and right inferior oblique: + 1 right medial rectus; + 2 left medial rectus.

On March 22, 1982 the left medial rectus was recessed 3 mm and the left lateral rectus resected 4 mm.

On March 26, 1982 a left carotid angiogram (Figures 2 and 3) and a vertebrobasilar angiogram (Figures 4 and 5) were both within normal limits.

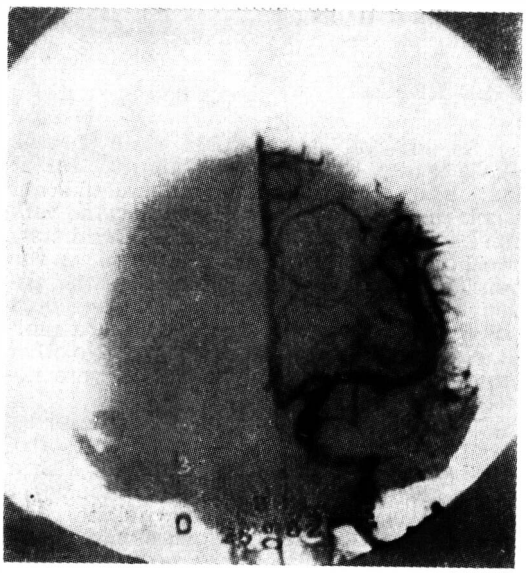

Fig. 2 — Normal left carotid angiogram, frontal view.

rectus and -1 right lateral rectus.

The patient was seen again on June 3, 1982. With glasses she had X (T) 5 and X 1. Without glasses:

ortho
↑
ortho ← ortho → ortho
↓
ortho

X' 1

Binocular rotations: + 1 right medial rectus and — 1 right lateral rectus.

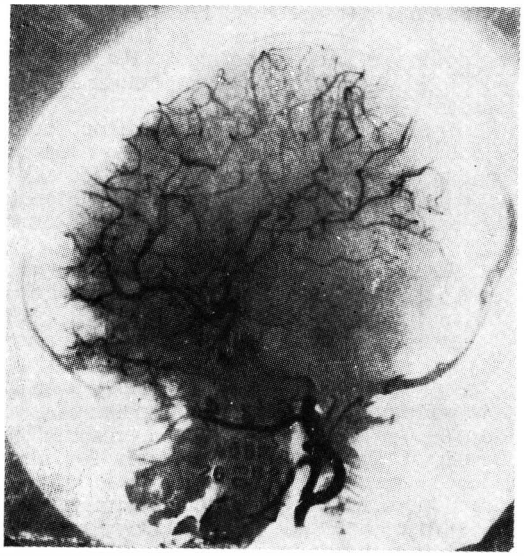

Fig. 3 — Normal left carotid angiogram, lateral view.

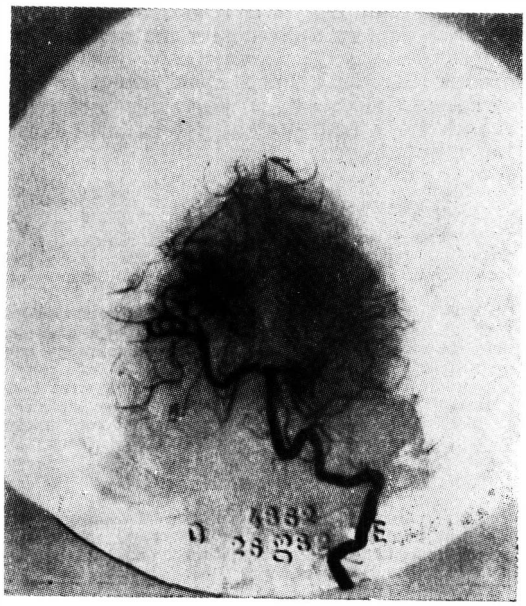

Fig. 4 — Normal vertebrobasilar angiogram, frontal view.

The glasses were discontinued and an appointment given.

DISCUSSION

In this patient a relation between the sudden state of fright and the onset of strabismus can be established. Althoug the ophthalmological, neurological, pediatric, hematological, radiological, and laboratory investigations disclosed only a left abducens paresis, the computorized tomography scan unexpectedly showed a left operculum infarction. A left carotid angiogram and a vertebrobasilar angiogram did not disclose other abnormalities. The question naturally arises on the events connecting the emotional stress, the cortical infarction, and the ipsilateral sixth nerve paresis.

Fig. 5 — Noormal vertebrobasilar angiogram, lateral view.

On account of a dreadful pale patient a cerebrovascular spasm could be considered. This condition is frequently associated with subarachnoid hemorrhage, head trauma or meningeal infection, which were not observed in the patient; her cerebrospinal fluid was normal and so the neurological examination. The question arises as to whether cerebrovascular spasm can cause transient ischemic attacks in the absence of subarachnoid hemorrhage. Gurdjian & Thomas [1] feel such a vasospasm has not been demonstrated, although not excluding the possibility that reversible vasospasm may produce a reversible clinical focal abnormality. They also question whether spasm can actively cause an infarction.

Feigin & Budzilovich [2] stress that myelinated and non-myelinated peripheral nerves are rarely seen in the smaller arteries or within the substance of the central nervous tissues. When present they are less common in the brain itself than in the brain stem. They conclude that the responsiveness of the muscle of an artery to the activity of autonomic nerves in their walls remains dis-

puted. In our patient, a spasm of a normal vessel leading to a cortical infarction does not seem an appealing explanation.

A cerebrovascular malformation would be another possible cause. These thin-walled anomalies, which vary in size, are visible in the surface of the brain, whence they extend as a wedge into the subcortical white matter (Pool [3]). The majority of cerebral arteriovenous malformations shows a preference for the centro-parietal cortex or to a lesser degree, the temporal and frontal areas. Luyendijk [4] fells that venous angiomas within the head show a tendency to be sited about the fissure. There is a preference for arterial nutrition from the sylvian vessels in more than 50%.

An arteriovenous malformation, according to Pool [3], most commonly becomes manifest as the result of epileptic seizures (absent in our patient) or hemorrhage. Bleeding can either spread into the subarachnoid space or evolve as an intracerebral hematoma. Minor bleeding may result only in a small area of well localized brain destruction (as seen in the computorized tomographic scan of our patient) with or without a neurological deficit, depending on the site of the hemorrhage. McCormick et al. [5] point out that a cerebral arteriovenous malformation is more apt to bleed than other types.

According to Feigin & Budzilovich [2] the gray matter is far more frequently affected than is white matter, but the superficial portion of the last, subjacent to the infarcted cortex, is also infarcted, so that the normally sharp line of demarcation between cortex and white matter is obscured. Luyendijk & Schoen [6] found that the prognosis of the superficially located hematomas is more favourable than that of deeper hematomas.

In our patient the left carotid angiogram (Figures 2 and 3) and the vertebrobasilar angiogram (Figures 4 and 5) did not show other abnormalities and we are left with the supposition of an isolated vascular malformation.

Pool [3] points out that exertion may precipitate bleeding from an arteriovenous malformation. Hypertension, both apart from and in connection with vascular abnormalities and diseases, is thought to play an important part in producing quasi-spontaneous hematomas. Taken as a pathogenic condition, it is the most frequent cause (Luyendijk [4]). In our patient, an elevation of blood pressure and an obstacle in venous circulation, both probably accompanying fear and weep, may have been responsible for the vessel (s) burst.

Edema is almost always present in cerebral infarction and is frequently observed in the tissues of the white matter adjacent to cerebral hemorrhages (Feigin & Budzilovich [2]). The accumulation of fluid may be very marked and, according to Feigin & Popoff [7], 1g of white matter can retain 2.5 g of water.

In our patient, the presence of edema which followed infarction may have led the brain tissues to compress the ipsilateral abducens nerve against the sharp border of the petrous portion to the temporal bone, with subsequent paresis.

The absense of other neurological symptoms and signs is probably related to site of the vascular accident.

In this patient a paresis of sudden onset was observed, followed by a spread of comitance with time as is frequently seen in the late stage of a paralytic strabismus (von Noorden [8]). There was also a marked increase of the angle of esotropia for near. With surgery the results obtained after a short term follow-up were encouraging but the patient deserves further control.

It is not uncommon that parents present themselves with a squinting child informing that strabismus followed a frightening situation. It is not suggested, of course, that all these children have a neuropathological basis to explain their ocular motor disorder. Nevertheless, with nonaggressive methods of examination that have been added to our armamentarium, it is certainly worth to search for a neurological determinant in some selected cases.

SUMMARY

A thirteen month old female was attacked by a dog. Although the animal did not touch the child she presented the next day with a left abducens nerve paresis.

Ophthalmological, neurological, pediatric, hematological, radiological, and laboratory assessments disclosed only the strabismus. A computorized tomography scan showed a left operculum infarction. A left carotid angiogram and a vertebrobasilar angiogram were both normal. The possible pathogenetic causes are discussed.

The patient was submitted to surgery and the results after a short term follow-up were satisfactory.

RESUMO

Uma paciente de 13 meses foi atacada por um cão, sem contudo que o animal a tocasse. No dia seguinte apresentou esotropia incomitante, com paresia de nervo abducente esquerdo

Investigação oftalmológica, neurológica, pediátrica, hematológica e laboratorial revelaram apenas o estrabismo. A tomografia axial computadorizada mostrou infarto do opérculo à esquerda. Angiografia carotídea esquerda e vertebrobasilar foram ambas normais. As causas patogenéticas possíveis são discutidas.

A paciente foi submetida a cirurgia e os resultados após um curto seguimento foram satisfatórios.

ACKNOWLEDGEMENTS

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Oftalmia simpática ou irritação simpática

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INTRODUÇAO

Oftalmia Simpática é uma Uveite generalizada, de origem desconhecida, granulomatosa, bilateral. Caracterizada clinicamente por um início insidioso e um curso progressivo com exacerbações e histopatológicamente por uma infiltração nodular ou difusa do trato uveal com linfócitos e células epiteliodes; que ocorre típicamente após injúria ocular penetrante.

A finalidade deste trabalho é chamar a atenção da classe Oftalmológica do reaparecimento da Oftalmia Simpática, quadro realmente presente e em ascenção na Oftalmologia Moderna; quer pela infortunística ocular, quer pela agressão ao olho por técnicas cirúrgicas como a Vitrectomia.

RELATO DO CASO

Em agosto de 1980, um homem (A.T.P.) de 22 anos de idade sofreu um traumatismo ocular causado por explosão de uma lâmpada que acarretou em perfuração do olho direito. No mesmo dia foi submetido à cirurgia ocular: remoção de vários fragmentos de vidro intra-ocular e sutura de córnea e esclera afirmando que a acuidade visual era de percepção luminosa desde o dia da infortunística ocular.

Dois meses após o acidente notou baixa da acuidade visual e visão turva no olho não traumatizado (olho esquerdo), quando lhe foi prescrito atropina e corticosteroide tópico e sistêmico.

Durante 6 meses esteve em uso de tal medicação com exacerbação do quadro ocular nas tentativas de redução ou suspensão da medicação quando foi encaminhado ao Serviço de Uveites do Hospital São Geraldo.

Este paciente foi por nós examinado em 28 de maio de 1981 e apresentava acuidade visual de percepção luminosa no olho direito e 20/40 no olho esquerdo. O estudo Biomicroscópio do traumatizado (olho direito) revelou extensa cicatriz horizontal em toda extensão corneana, hiperemia conjuntival moderada, câmara anterior rasa e seclusão pupilar.

No olho não traumatizado (olho esquerdo) notamos hiperemia conjuntival discreta; pequena quantidade de precipitados ce-

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