Journal of Neurology, Neurosurgery, and Psychiatry 1994;57:1126–1128

SHORT REPORT

Initial enlargement of the opposite pupil as a false localising sign in intraparenchymal frontal haemorrhage

R Chen, R Sahjpaul, R F Del Maestro, L Assis, G B Young

Department of Clinical Neurological Sciences
R Chen
R Sahjpaul
R F Del Maestro
G B Young

Department of Radiology, University of Western Ontario, London, Ontario, Canada
L Assis

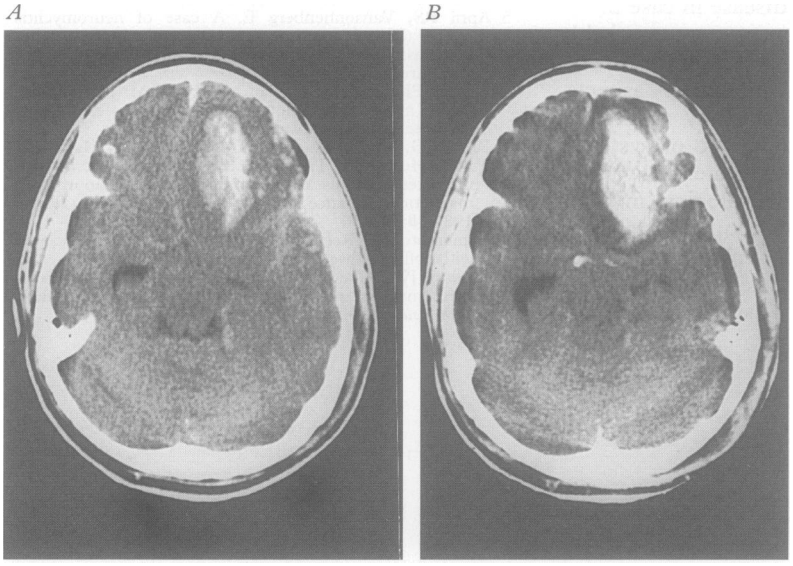
Correspondence to:
Dr G B Young, Room W824, Victoria Hospital, 375 South Street, London, Ontario, Canada N6A 4G5

Received 1 November 1993 and in revised form 25 January 1994.
Accepted 22 February 1994

Abstract

Ipsilateral third nerve palsy with early pupillary enlargement is an important sign of transtentorial herniation from a supratentorial mass lesion. A case of frontal, intraparenchymal haemorrhage is reported in which the first ocular manifestation of transtentorial herniation was enlargement of the contralateral pupil. The ipsilateral pupil dilated only after complete oculomotor palsy of the contralateral eye. After partial frontal lobectomy and removal of blood clot, the ipsilateral third nerve recovered before the contralateral third nerve. Clinical findings localised the contralateral third nerve lesion to an extra-axial site. The possible mechanisms of contralateral third nerve compression are discussed. This seems to be the first example of pupillary enlargement as a false localising sign from a contralateral, supratentorial, intraparenchymal mass lesion.

(*J Neurol Neurosurg Psychiatry* 1994;57:1126–1128)



(A) Day 1 at 2300. CT showed a large left frontal, intraparenchymal haematoma with subfalcial herniation and subarachnoid blood in the interhemispheric fissure. The left temporal horn is compressed and medially displaced. The right temporal horn is enlarged. Note obliteration of the suprasellar and interpenduncular cisterns, but easily identifiable quadrigeminal plate cistern. (B) Day 2 at 1630. The haematoma is larger and the surrounding oedema is now clearly defined. The quadrigeminal plate cistern is no longer visible.

Ipsilateral oculomotor nerve palsy has long been recognised as an important sign of mass effect caused by a supratentorial lesion. The sequence of events is stereotyped with loss of light response, followed by a brief period of pupillary constriction; then pupillary dilatation and finally loss of oculomotor function.[1 2] Changes in the contralateral pupil are usually seen around the time of dysfunction of ipsilateral eye movement.[1] A similar sequence of changes then develops in the contralateral eye.[3]

Enlargement of the opposite pupil as the first ocular sign of herniation is rare but has been described in patients with extra-axial mass lesions.[4–6] The mechanism is uncertain. To our knowledge no such case has been described for intra-axial lesions. We present a case of initial pupillary enlargement contralateral to an acute frontal intraparenchymal haemorrhage. The possible mechanisms of pupillary dilatation are discussed.

Case report

A 29 year old previously healthy man was found unconscious on the restroom floor at 2100 while at work. When examined at our hospital at 2300 his blood pressure was 150/60 mm Hg, pulse 50 beats/min, and respiratory rate 14/min. He opened his eyes to pain, spoke occasional comprehensible words, and moved all limbs spontaneously, but did not follow commands. Both pupils were 5 mm and reacted briskly to light. Fundi were normal, venous pulsations were present, and no gaze preference was noted. Muscle tone was increased and deep tendon reflexes were brisk on the right side. Both plantar responses were extensor. Brain CT showed a left frontal intraparenchymal haematoma (figure (A)).

At 0720 the next day he was drowsy but arousable, opened his eyes to voice, nodded his head to questions, and moved all limbs purposefully. Pupils were 4 mm and reactive. Carotid angiograms did not show a source of haemorrhage. His level of consciousness fluctuated throughout the day. At 1500 both pupils were 3 mm and reactive. At 1530 his right pupil dilated to 6 mm, became unreactive to light, and he developed a right ptosis.

Summary of clinical course

	Day 1 2300	*Day 2 0720*	*1500*	*1530*	*1600*	*1645*	*1800*	*Day 3 0800*	*Day 4 0800*	*Day 29 0800*	*Day 60 0800*
Event	CT		Angiogram		Mannitol	CT	ET,HV,OR				
Right pupil	4 mm R	4 mm R	3 mm R	6 mm NR	6 mm NR	7 mm NR	6 mm NR	4 mm NR	4 mm R	4 mm R	3 mm R
Left pupil	4 mm R	4 mm R	3 mm R	4 mm R	4 mm R	7 mm NR	3 mm NR	2 mm MR	2 mm R	3 mm R	3 mm R
Eyelids	Normal		Right ptosis		Unable to assess			Right ptosis	Mild right	Normal ptosis	
EOM	Full			Abducted right eye *				Full			
Limbs	Normal power Right hyper-reflexia			Normal power Restless		Bilateral extensor posturing		Left: semi-purposeful movement Right: extend to pain	Right hemiplegia		Mild right hemiplegia

* Absent vertical and horizontal eye movements with oculocephalic manoeuvre. EOM = extraocular eye movements, ET = endotracheal intubation; HV = hyperventilation, MR = minimally reactive; NR = non-reactive; OR = operating room; R = reactive.

Blood pressure was 180/100 mm Hg. He was restless and moved all four extremities purposefully, but did not obey commands. At 1600 his right eye was abducted and failed to move across the midline with oculocephalic manoeuvre. The left eye retained pupillary reactivity (4 mm unstimulated) and motility. After treatment with mannitol, a second CT at 1630 showed increased mass effect from the left frontal haematoma (figure (B)). At 1645 he was deeply unconscious with bilateral extensor posturing; both pupils were 7 mm and unreactive. By 1800 after endotracheal intubation and hyperventilation, the left pupil size decreased to 3 mm but was still unreactive; the right pupil remained dilated (6 mm) and fixed.

At 1900 a left frontal craniotomy disclosed a contused frontal lobe with a large intraparenchymal and smear subdural haematoma. A partial left frontal lobectomy and clot removal were performed.

The next morning (hospital day 3) he did not obey commands, showed left sided semipurposeful movements, and extended his right side to pain. The right pupil was unreactive at 4 mm whereas the left was 2 mm and reacted sluggishly to light. No horizontal or vertical eye movements could be shown with oculocephalic manoeuvre in the right eye. On hospital day 4 right ptosis was noted on spontaneous eye opening. The pupil sizes were unchanged but both now reacted to light. Oculocephalic manoeuvre showed complete conjugate eye movements. Follow up CT showed bilateral infarcts in the posterior cerebral artery territory.

He made a gradual recovery. Examination on day 29 showed mild right sided ptosis, a 4 mm right pupil, a 3 mm left pupil, and bilateral light reactivity. Voluntary extraocular movements were normal. He had right hemiplegia with normal strength on the left side. Two months after admission he had mild right hemiparesis and the ptosis had resolved. He was transferred to a rehabilitation hospital.

The table summarises his clinical course.

Discussion

Dilatation of the contralateral temporal horn and obliteration of the suprasellar and perimesencephalic cisterns found by CT are characteristic features of transtentorial herniation. Coincident with the loss of light reflex, the opposite pupil dilated and ptosis occurred. These were followed by paresis of oculomotor nerve innervated extraocular movements. The ipsilateral pupil was affected only after contralateral eye movement paresis, by which time the patient was obtunded with bilateral extensor posturing. The ipsilateral pupillary palsy responded to reduction of intracranial pressure with mannitol and hyperventilation. Postoperatively, the ipsilateral eye also recovered before the contralateral eye. Thus the sequence of changes in the contralateral eye is similar to that reported for the ipsilateral eye,[1 2] whereas the ipsilateral eye behaved in the manner described for the contralateral eye.[1 3]

Several authors have described dilatation of the opposite pupil with extra-axial lesions. Browder[5] found that in 38 out of 289 cases of subdural haematoma the contralateral pupil was larger than the ipsilateral pupil. Pevehouse *et al*[6] reported similar findings in nine out of 389 cases. It is unclear, however, whether these patients had third nerve compression as eye movements were not described and anisocoria is found in 20% of the population.[7] Gassel[4] studied 250 patients with meningioma and found 12 cases with the larger pupil on the contralateral side. When anisocoria was combined with other evidence of third nerve involvement only 2 cases of contralateral involvement were found. Although a smear subdural haematoma was found in our patient, the mass effect was almost certainly caused by the much larger intraparenchymal haemorrhage. To our knowledge, no previous case of dilatation of the opposite pupil has been reported as the first ocular manifestation of an intraparenchymal lesion.

The proposed explanations for third nerve dysfunction from a supratentorial mass lesion include both midbrain[3] and extra-axial compression.[1 2 8] Clinical findings in our case localise the compression of the opposite third nerve to an extra-axial site. The unilateral ptosis is incompatible with a nuclear third nerve lesion. The right cerebral peduncle and red nucleus were not affected as neither left hemiparesis nor tremor was noted. Therefore, a lesion of the third nerve fasicle within the midbrain is unlikely.

The most widely accepted mechanism of extra-axial third nerve compression is transtentorial uncal herniation resulting in pressure on the third nerve in the tentorial opening,[1 2 8] which has been reproduced with experimental extradural compression in monkeys and cats.[2 8] The uncus may compress the third nerve directly, cause compression by the posterior cerebral artery,[1 8] or push the nerve against the petroclinoid ligament.[2] As the posterior cerebral artery descends secondary to downward displacement of the brainstem, it may also cause compression of the upper surface of the third nerve near its exit from the midbrain.[1] Another mechanism is the shift and rotation of the midbrain to the contralateral side resulting in compression of the ipsilateral third nerve over the edge of the anterolateral clivus.[9] This, however, cannot explain compression of the contralateral third nerve as the nerve is slackened by such displacement.

We can only speculate on the possible mechanism of paradoxical opposite pupillary enlargement. For it to occur, anomalous anatomical variation is likely to be present. A rudimentary or absent ipsilateral posterior cerebral artery is a possible mechanism[3] but is unlikely in our case because there was clearly compression of the ipsilateral posterior cerebral artery, which resulted in infarction in the left posterior cerebral artery territory. Also, a prominent posterior communicating artery is expected with such an anomaly but was not seen on the carotid angiogram. There is wide variation in the size of the tentorial notch.[1] A narrow ipsilateral and wide contralateral tentorial opening may lead to contralateral before ipsilateral uncal herniation. The opposite third nerve may also be wedged between the posterior cerebral and superior cerebellar arteries as a result of lateral displacement of the midbrain.[3] This may produce initial opposite pupil dilatation if associated with other anomalies such as narrow ipsilateral tentorial opening or the two arteries originating from a common trunk, moving the angle of divergence more laterally. The inferior location of the haematoma may result in initial upward displacement of the brainstem that has been described in a case of putaminal haemorrhage.[9] Paradoxical pupillary dilatation may occur if there is associated rotation of the midbrain in the coronal plane such that the contralateral side is raised compared with the ipsilateral side. This causes traction on the third nerve as it passes under the posterior cerebral artery. An alternative mechanism is an anomalous course for the ipsilateral third nerve away from the ipsilateral uncus, rendering its compression a late phenomenon in uncal herniation.

Experimental frontal extradural compression in cats produced bilateral uncal herniation whereas temporal compression caused unilateral herniation.[8] Therefore, the inferior frontal location of the haematoma is likely important in producing the force vectors involved to cause contralateral third nerve palsy, by contrast with those produced by a temporal lobe haematoma.

In conclusion, we found that pupillary dilatation and third nerve compression can be a "false localising sign" for a contralateral, supratentorial, intraparenchymal mass lesion. Its recognition can be helpful in reconciling conflicting neurological signs and in the localisation of lesions. Such information is often crucial in the interpretation of neuroradiological investigations and in patient management.

1 Sunderland S. The tentorial notch and complications produced by herniations of the brain through that aperture. *Br J Surg* 1958;**45**:422–38.
2 Reid WL, Cone WV. The mechanism of fixed dilatation of the pupil. *JAMA* 1939;**112**:2030–4.
3 Ropper AH. The opposite pupil in herniation. *Neurology* 1990;**40**:1707–9.
4 Gassel MM. False localizing signs. *Arch Neurol* 1961;**4**: 526–54.
5 Browder J. A resumé of the principal diagnostic features of subdural hematoma. *Bulletin of the New York Academy of Medicine* 1943;**9**:168–76.
6 Pevehouse BC, Bloom WH, McKissock W. Ophthalmologic aspects of diagnosis and localization of subdural hematoma. *Neurology* 1960;**10**:1037–41.
7 Loewenfeld IE. "Simple, central" anisocoria: A common condition, seldom recognized. *Transactions of the American Academy of Ophthalmology and Otolaryngology* 1977;**83**:832–9.
8 Jennett WB, Stern WE. Tentorial herniation, the midbrain and the pupil. *J Neurosurg* 1960;**17**:598–609.
9 Ropper AH, Cole D, Louis DN. Clinicopathologic correlation in a case of pupillary dilation from cerebral hemorrhage. *Arch Neurol* 1991;**48**:1166–9.