SHORT REPORT

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

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

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.

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.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.⁴⁵ 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.

(A) Day 1 at 2300. CT showed a large left frontal, intraparenchymal haematoma with subfalcine 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 interpeduncular 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.

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Department of Clinical Neurological Sciences
R Chen
R Sahipaul
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

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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 lobeectomy and clot removal were performed.

The next morning (hospital day 3) he did not obey commands, showed left sided semi-purposeful 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 extracocular movements were normal. He had right hemiparesis 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.

<table>
<thead>
<tr>
<th>Event</th>
<th>Day 1</th>
<th>Day 2</th>
<th>1500</th>
<th>1530</th>
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<th>1645</th>
<th>1800</th>
<th>Day 1</th>
<th>Day 2</th>
<th>Day 3</th>
<th>Day 4</th>
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<tr>
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<td>2300</td>
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<td>CT</td>
<td>Angiogram</td>
<td>Mannitol</td>
<td>CT</td>
<td>ET, HV, OR</td>
<td>6 mm NR</td>
<td>4 mm NR</td>
<td>6 mm R</td>
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<td>Right: extend to pain</td>
<td>Right ptosis</td>
<td>Mild right</td>
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<td>ptosis</td>
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<tr>
<td>EOM</td>
<td>Full</td>
<td>Normal power</td>
<td>Abducted right eye *</td>
<td>Normal power</td>
<td>Bilateral extensor posturing</td>
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<td>Limbs</td>
<td>Normal power Right hyper-reflexia</td>
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* Absent vertical and horizontal eye movements with oculocephalic manoeuvre. EOM = extracocular 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.

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**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, whereas the ipsilateral eye behaved in the manner described for the contralateral eye.

Several authors have described dilatation of the opposite pupil with extra-axial lesions. Browder found that in 38 out of 289 cases of subdural haematoma the contralateral pupil was larger than the ipsilateral pupil. Pevehouse et al 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. Gassel 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 and extra-axial compression. 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 fascicle within the midbrain is unlikely.
The most widely accepted mechanism of extra-axial third nerve compression is
transient dural 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.9 8 The uncal may compress the third nerve directly, cause compression by the
posterior cerebral artery,1 8 or push the nerve against the pteronial ligament.3 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 con-
tralateral 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 if the nerve is slackened by such dis-
placement.

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 mechanism3 but is
unlikely in our case because there was clearly compression of the ipsilateral posterior cere-
bral 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 ten-
torial opening may lead to contralateral before ipsilateral uncal herniation. The oppo-
site 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 ini-
tial upward displacement of the brainstem
that has been described in a case of putaminal haemorrhage.8 Paradoxical pupillary dilata-
tion 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 poste-
rior 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 compres-
sion in cats produced bilateral uncal hernia-
tion 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 local-
isation of lesions. Such information is often
crucial in the interpretation of neuroradiolog-
ical investigations and in patient manage-
ment.

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