The Journal of Laryngology & Otology
March 2002, Vol. **116,** pp. 227–229

Bilateral cortical blindness: an unusual complication following unilateral neck dissection

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Abstract

We present the case of a 50-year-old man who developed bilateral cortical blindness and confusion following a seemingly uneventful right-sided radical neck dissection. Computed tomography (CT) scans confirmed bilateral occipital lobe infarctions.

To our knowledge, there are no previously documented reports of this clinical event following head and neck surgical procedures. Although this is a rare occurrence, otolaryngologists should be aware of this potential post-operative complication.

The possible aetiologies of this condition are discussed.

Key words: Blindness, Cortical; Neck; Post-operative Complications

Case report

A 50-year-old man was referred to our Head and Neck Oncology clinic with a month's history of a painless lump in the right side of his neck. He had no symptoms referable to his upper aerodigestive tract. He was a non-smoker. His past medical history revealed ongoing arthritic problems with his neck and back, for which he received osteopathic treatment. He also had had a myocardial infarction at age 30, and had had surgical treatment for a spontaneous retinal detachment at age 35 years.

Clinical examination revealed a firm mobile jugulo-digastric lymph node of 3 cm diameter on the right side of his neck and induration of his right arytenoid, but no other significant abnormality. Fine needle aspiration cytology of this neck lump was unhelpful, while repeated biopsies of his arytenoid swelling revealed no significant abnormality. Excision biopsy of the enlarged node was performed; this was reported as a poorly differentiated non-small cell carcinoma, which was positive for cytokeratin but negative on immunostaining for anti-melanoma antigen and CD30. Further investigations to detect a possible primary including bronchoscopy, flexible gastroscopy, bone isotope scans and CT examination of the neck, chest and abdomen were all unremarkable.

The patient was admitted electively for a right-sided radical neck dissection, which was done in routine fashion. There were no intra-operative surgical complications, and anaesthetic monitoring showed no hypoxic or hypotensive episodes. His immediate recovery period was uneventful, and he was transferred to the Head and Neck unit for his post-operative care.

On the first post-operative morning, the patient was noted to be confused and apathetic. He complained to nursing and medical staff about being unable to see. Physical examination showed no cardio-respiratory insult and a normal peripheral nervous system. He was, however, unable to detect light on visual field examination. Cranial nerve examination was otherwise normal and fundoscopy revealed no new retinal abnormality. The presumptive

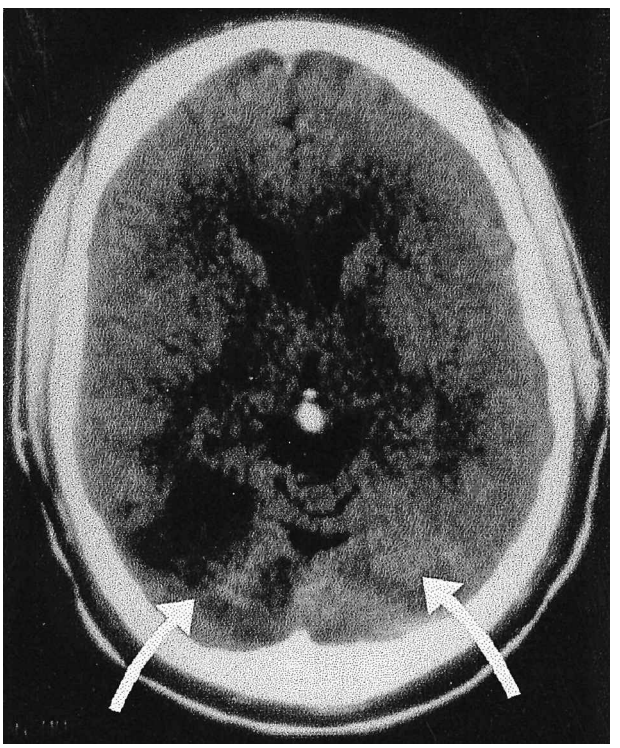

Fig. 1
CT scan of the brain showing bilateral occipital infarction.

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Accepted for publication: 18 September 2001.

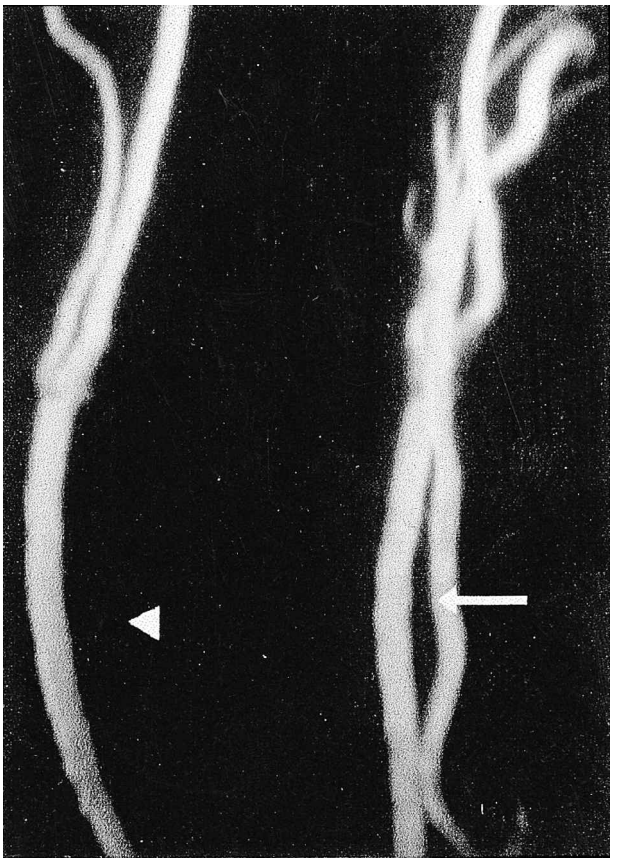

FIG. 2

Magnetic resonance angiography showing a dominant right vertebral artery (white arrow) and the rudimentary left vertebral artery (white arrowhead).

diagnosis of central blindness was confirmed after specialist ophthalmological and neurological consultations. An urgent CT scan (Figure 1) revealed large low-density areas in both occipital cortices consistent with infarction. Subsequent investigations revealed no haematological, biochemical or immunological abnormalities and a carotid Doppler study was also normal. Echocardiography showed evidence of previous infarction, but no mural thrombus.

Magnetic resonance angiography was performed and showed a dominant right vertebral artery with a rudimentary contralateral counterpart (Figure 2). There was a normal sized basilar artery completing the circle of Willis (Figure 3).

Fortunately, the patient's vision has partially recovered, and at his last ophthalmology appointment central vision in his left eye was 5/6, but he still has no peripheral vision in that eye. This is consistent with macular sparing of the left visual field. He has no useful vision in the right eye.

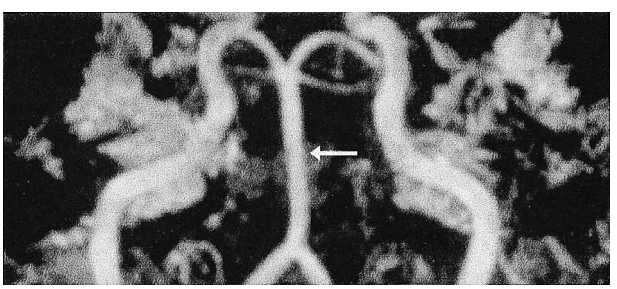

FIG. 3

Magnetic resonance angiography showing a normal sized basilar artery (white arrow).

TABLE I

CAUSES OF CEREBRAL BLINDNESS

Cerebrovascular disease
Cardiac surgery
Cerebral angiography
Craniotomy
Cardiac arrest
Head trauma
Seizures
Bacterial meningitis

Adapted from Aldrich *et al.*[2]

Discussion

The most common cause of cortical blindness is cerebral vascular disease.[1] Aldrich *et al.* found this to be the cause of central blindness in 32 per cent of cases in his series;[2] other causes are listed in Table I. The prognosis was better in cortical blindness due to these latter causes than when it was a result of a stroke. But patients with bi-occipital CT lesions never showed complete recovery of vision.

In some cases of cortical blindness there may be a denial of the visual loss and elaborate descriptions of the visual environment may be presented. This is known as Anton's syndrome, and may mimic functional vision loss as the fundi and pupillary reflexes are normal in both conditions.[3]

In younger patients, approximately one-fourth of all cerebral infarcts are located in the vertebro-basilar territory[4] and approximately one-third of these are exclusively occipital infarcts, suggesting an overall frequency of eight per cent for occipital infarcts.[5]

There are numerous anecdotal reports in the literature about vertebro-basilar ischaemia and consequent occipital lobe infarction due to a variety of causes. These are summarized in Table II. Of special interest is the report of visual field loss resulting from cervical chiropractic manipulation.[6] Since our patient underwent right radical neck dissection, his head and neck were extended and turned to the left. Diminution of blood flow in the vertebral and basilar arteries has been demonstrated with rotation of the head and extension of the neck.[7,8]

Kuether *et al.* reported that the symptoms of vertebro-basilar insufficiency were reproducible with rotational head movement and conclude that this is an important cause of cerebral ischaemia and consequent neurological deficit.[9] The decrease in blood flow in the basilar artery seems to be more significant in individuals with asymmetry of the vertebral arteries of more than 75 per cent.[7] Ischaemia in the territory of a hypoplastic vertebrobasilar system has also been demonstrated by Chaturvedi *et al.*,[10] Matsuyama *et al.*[11] and Morimoto *et al.*[12]

In our patient, magnetic resonance angiography showed the left vertebral artery to be hypoplastic. We, therefore, hypothesize that sluggish flow in this vessel could have

TABLE II

Mechanism of occipital lobe infarction	Reference
Cervical chiropractic manipulation	Donzis and Factor[6]
Acute migraine	Ganji *et al.*[13]
Persistent trigeminal artery	Gasecki *et al.*[14]
Cerebral angiography	Hansen and Stenbjerg[15]
Tentorial herniation	Sato *et al.*[16]
Acute transient hypotension	Strandgaard *et al.*[17]
Cardiac catheterization	Thomas and Troost[18]

https://doi.org/10.1258/0022215021910410 Published online by Cambridge University Press

resulted in the formation of an intraluminal thrombus, which later embolized and obstructed the blood supply to both occipital lobes, resulting in bilateral cortical blindness. Another equally plausible scenario could be the temporary occlusion of the dominant right vertebral artery, with subsequent occipital hypoperfusion via the hypoplastic left vertebral artery.

Conclusion

Our patient developed bilateral occipital infarcts in a hitherto unreported situation, namely following a head and neck surgical procedure. Rotation of the head and neck is often necessary for adequate positioning of the patient during these procedures. By reporting this unfortunate complication we hope to highlight the risk of ischaemic injury to the brain, in particular the areas supplied by the vertebro-basilar system as a result of rotational obstruction of an already precarious blood supply.

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Mr P. Moore takes responsibility for the integrity of the content of the paper.
Competing interests: None declared

https://doi.org/10.1258/0022215021910410 Published online by Cambridge University Press