Brain SPECT in a Case of Cortical Blindness

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Background
Published reports on patients with cortical blindness describe bilateral brain hemispheric lesions visualized in radiological and functional imaging studies. Published reports of functional imaging studies on patients with cortical blindness are limited. Cestea et al.1 report severely decreased regional cerebral blood flow over the entire posterior parieto-occipital region of both hemispheres on single-photon emission-computed tomographic (SPECT) scanning in a patient with cortical blindness secondary to a cerebrovascular accident. Cestea et al.1 later reported bilaterally decreased N-isopropyl[123I]-p-iodoamphetamine (IMP) concentration in both occipital lobe areas on SPECT scanning in two patients with total blindness, and small spared unilateral areas 17 in two patients with rudimentary residual vision. All the cases reported had bilateral occipital structural lesions visible in either magnetic resonance imaging (MRI) or computer-assisted tomographic scan studies.

We describe a patient who became cortically blind after suffering a penetrating traumatic unilateral lesion of the right occipital lobe. Extensive radiological evaluation failed to reveal bilateral occipital involvement, but SPECT scan showed prominently decreased metabolism of both parieto-occipital areas. We suggest that SPECT scanning can be a precise indicator of cortical dysfunction despite normal radiological studies. We also hypothesize that the clinical and imaging findings in this patient could be attributed to diaschisis.

Case Report
A 26-year-old male policeman was admitted to a large trauma center after suffering a tangential gunshot wound to the head. He was found unresponsive at the scene and was flown by helicopter to a trauma center, where his Glasgow Coma Score (GCS) was 3 (intubated). His admission neurological examination was significant for equal and reactive pupils, conjugate gaze on doll’s eyes maneuver, absent corneal reflexes on the left, and no spontaneous or withdrawal movement of any extremity.

Initial noncontrast computed tomography (CT) of the brain showed an open fracture in the right parietal region and bone and small ballistic fragments in the right parieto-occipital hemispheric region (Fig 1). The patient underwent a right parieto-occipital craniotomy with debridement of the gunshot wound, placement of an intracranial pressure monitor device (Camino Catheter), and repair of the dura. No attempt was made to excise the intracerebral bone or bullet fragments.

On the day after admission the patient’s GCS was 9, with withdrawal and spontaneous movements of all extremities, but less so on the right side. An enhanced cranial CT showed no significant intraparenchymal changes from the previous day. The following day his GCS was 11, and the patient began following simple commands. On the ensuing days the patient became progressively more alert and complained of not being able to see. Examination was remarkable for blindness to all modalities in all four fields, normal visual fundoscopic examination, pupils equal and reactive to light, and spastic quadripareis that was more pronounced on the right side.

Seven days after admission an unenhanced cranial MRI study showed a right parieto-occipital contusion with multiple bone and bullet fragments and the presence of minimal surrounding edema (Fig 2). Brain stem lesion, mass effect, midline shift, herniation, and brain
A SPECT scan of the brain was performed 5 weeks after injury. After intravenous administration of $^{99m}$Tc-labeled hexamethylpropyleneamine oxime (HMPAO), imaging was performed using a Toshiba 9300 triple header Angel camera with a fanbeam, low-energy, ultrahigh-resolution collimator. Sensory input was minimized during imaging by performing the procedure in a quiet, dimly lit room, with the patient wearing a blindfold and headset. Thirty-second images were acquired at 4° intervals throughout a rotation of 360°. The tomographic images demonstrated a pattern of normal high activity in the peripheral cortical gray matter, the deep gray matter structures (the basal ganglia and thalamus), and the cerebellar cortex. Prominently decreased radiopharmaceutical accumulation was seen posteriorly in the visual cortex of both occipital lobes (Fig 3). This decrease in regional cerebral blood flow to the occipital cortical areas was more extensive on the right than the left.

Discussion

Our patient was unusual in that extensive radiological studies, including unenhanced and enhanced cranial CT and MRI, failed to show the extensive bilateral lesions usually found in reported cases of cortical blindness. In contrast, a SPECT scan showed involvement of a much more extensive area than radiological studies and correlated much more closely with the clinical picture. The residual vision in the right homonymous hemifield correlated clinically with a small area of preserved function in the left posterior occipital cortex on SPECT scanning.

The etiological basis of our patient's clinical status is unclear. A vascular lesion involving the posterior circulation could explain the clinical findings; however, the absence of radiographic evidence of ischemia would make this unusual. Axonal shearing injuries could also account for his motor deficits but would be unlikely to result in blindness.
An alternative explanation for bilateral cortical involvement with a unilateral structural lesion would be that of diaschisis. Von Monakow initially proposed the concept of diaschisis in 1905, suggesting that after focal brain injury neurons in regions of morphologically intact brain, remote from but anatomically connected to the damaged area, become functionally depressed because of a loss of excitatory input from the injured area. Feeney and Baron propose that a modern concept of diaschisis should include the following criteria. (1) Symptoms should correlate with physiological measures of functional disturbances; (2) both symptoms and physiological measures must respond to the same manipulations or show parallel changes with time after injury; (3) functional disturbances due to the prolonged lack of neural input may progress to morphological changes; (4) diaschisis must occur in an area after loss of its major afferent input and may not occur or be easily detected in structures with multiple inputs; (5) diaschisis could also result in disinhibition after loss of inhibitory input; and (6) some seemingly permanent symptoms may represent an enduring diaschisis if they are reversible by surgery or drug treatment.

Crossed cerebellar diaschisis has been described after brain injury. Transhemispheric diaschisis, manifested by decreased cerebral blood flow in the opposite hemisphere to the lesion, has also been described in patients with unilateral stroke. In this study, however, patients did not display clinical evidence for transhemispheric dysfunction. Other authors have also reported bihemispheric decreased blood flow and metabolism in patients with unilateral cerebral infarctions and have attributed this finding to diaschisis. In our patient, the normal morphology displayed by radiological studies in the left hemisphere, which contrasted with the severe deficit of blood flow to that same area, could be accounted for by transhemispheric diaschisis. Additionally, clinical evidence of transhemispheric dysfunction...
in our patient was manifested by severe bilateral visual impairment.

Radiological studies are frequently normal in patients with traumatic brain injury despite prominent clinical abnormalities. In this scenario, SPECT scanning is a promising technique for defining focal brain dysfunction.

References
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