

Iatrogenically Induced Cortical Blindness Associated with Leptomeningeal Enhancement

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Summary: Leptomeningeal enhancement is usually infective or neoplastic in origin. We present a case in which a patient received total parenteral nutrition via a catheter unknowingly placed within the right vertebral artery. We postulate that the hyperosmolar nature of the infused solution induced temporary osmotic disruption of the blood-brain barrier, resulting in cortical blindness associated with localized leptomeningeal enhancement.

Abnormal meningeal enhancement may take two forms: dura-arachnoid enhancement, which follows the inner contour of the calvaria, or pia-subarachnoid space enhancement, which extends into the depths of the sulci. The latter, also referred to as leptomeningeal enhancement, is often caused by an infectious or neoplastic process (1, 2). We present a case of marked regional leptomeningeal enhancement accompanied by cortical blindness thought to be due to the inadvertent infusion of a hyperosmolar solution into a vertebral artery.

Case Report

A 37-year-old HIV-positive man was admitted for investigation of hyperbilirubinemia associated with fevers and chills. Early in his hospital stay, a central venous catheter was inserted via a right infraclavicular approach. The tip of the catheter, which extended up the right side of the neck, was assumed to lie within the right internal jugular vein. The catheter was initially perfused with a dextrose-saline solution. Six hours after placement of the line, total parenteral nutrition was administered via the catheter. Over the ensuing 8 hours, the patient reported light-headedness and blurred vision, which progressed to complete binocular loss of vision. Clinical examination revealed cortical blindness without other focal neurologic signs. A lumbar puncture showed a pressure of 310 mm of water. The protein content was also elevated, at 84 mg/dL. No abnormal cells were present.

A contrast-enhanced MR examination 5 hours after complete loss of vision showed marked, patchy pial-subarachnoid space and subpial enhancement over the cerebellum, the occipital lobe, and the thalami, suggesting involvement of the vertebrobasilar arterial territory (Fig 1A-C). Review of a portable chest radiograph showed the tip of the central catheter extending cephalad over the right transverse processes of the

lower cervical spine. Contrast material injected into the central catheter confirmed the position of the catheter in the right vertebral artery (Fig 1D). The catheter was removed and a new catheter inserted via the left infraclavicular approach into the left innominate vein.

Over the ensuing 2 weeks, the patient's symptoms improved and he was left with a mild right homonymous hemianopia. A repeat MR study showed only minimal high signal intensity on T2-weighted images within the left optic radiation. The pial-arachnoid enhancement had completely resolved (Fig 1E).

Discussion

The arachnoid and pia mater compose the leptomeninges. Breakdown of the leptomeningeal blood-brain barrier (BBB) from a variety of causes, but especially from infection and neoplasia, is responsible for the abnormal contrast enhancement seen on contrast-enhanced MR images.

Animal studies suggest that in infectious meningitides, breakdown of the leptomeningeal BBB is due to the bacterial cell wall components inciting an inflammatory reaction that results in opening of the tight intercellular junctions in the arachnoid capillary bed (2, 3). In carcinomatous meningitis, it is suggested that there is neoplastic invasion of the meninges, which disrupts the tight capillary junctions of the outer layer of the arachnoid, resulting in pial-subarachnoid enhancement after administration of contrast material (4). The presence of leptomeningeal enhancement in our HIV-positive patient, although localized over the cerebellum, occipital lobes, and portions of the mesial temporal lobes, raised the possibility of tuberculosis, cryptococcosis, or lymphoma (5, 6). However, the presence of enhancement in the anterior thalamus and the choroid plexus, in addition to the localized leptomeningeal enhancement, suggested a process involving branches of the vertebrobasilar system. This prompted a search for the source, which led to the discovery of the malpositioned catheter.

The BBB may be opened experimentally by introducing hyperosmolar solutions into the bloodstream (7, 8). Osmotic dehydration of the capillary endothe-

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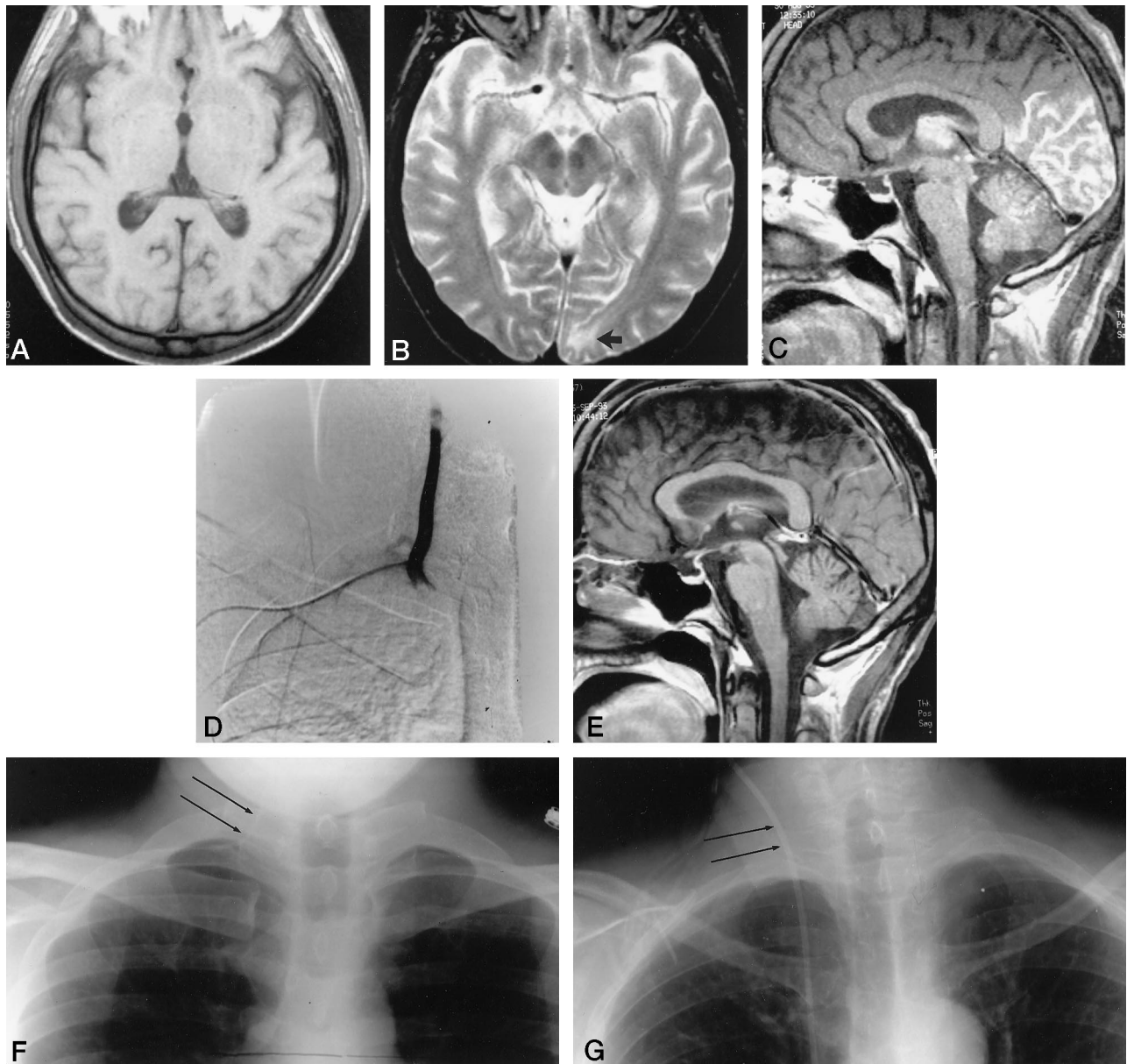


FIG 1. 37-year-old man with cortical blindness.

A, Noncontrast axial T1-weighted MR image (540/15/2) at the level of the thalami and occipital lobes appears normal.

B, Axial T2-weighted MR image (2500/90/1) shows high signal intensity within the left optic radiation (arrow).

C, Sagittal contrast-enhanced T1-weighted MR image (538/15/2) shows leptomeningeal enhancement over the cerebellum and occipital lobe. Enhancement is also seen in the thalamus.

D, Contrast injection into the catheter in the right side of the neck opacifies the right vertebral artery (arrow).

E, Sagittal contrast-enhanced T1-weighted MR image (580/15/2) 2 weeks later shows no remaining abnormal enhancement.

F Plain radiograph of the lower neck and upper chest shows the abnormal medial course of the catheter (arrows).

G, Plain radiograph in a different patient shows normal course of a catheter within the right internal jugular vein. Note that the catheter does not overlie the transverse processes of the lower cervical vertebrae (arrows).

lial cells may cause them to shrink, thus pulling apart or “unzipping” the tight junctions (9). Intentional injection of hyperosmolar solutions of urea and mannitol into the cerebral arteries has been used to open the BBB, after which chemotherapeutic agents may be delivered to those portions of brain tumors with an otherwise intact BBB (10, 11).

Our patient had been inadvertently receiving vertebral intraarterial total parenteral nutrition that contained 30% dextrose together with 20% fat and a

mixture of trace elements and electrolytes at an estimated osmolality of 1500 mOsm/L through a catheter thought to be located intravenously. We postulate that this hyperosmolar solution induced osmotic disruption of the BBB, which led to the patient’s blindness and the subsequent MR findings. The presence of the catheter within the vertebral artery may have caused periodic impairment of vertebral artery flow. It is possible that inadvertent air emboli from the central line or fat emboli from the injection of the

total parenteral nutrition may have resulted in scattered cerebral and cerebellar infarction, but the onset of enhancement in cerebral infarction is nearly always delayed for several days after the event and therefore seems unlikely (12).

Our patient received 16 mL of gadopentetate dimeglumine through the indwelling catheter. Although there have been a few reports on the intraarterial use of gadolinium (13, 14), no mention was made about the possible neurotoxic effects. The direct intraarterial injection of a gadolinium complex in our patient's case occurred after his symptoms developed and cannot be incriminated, but the possibility exists that it may have caused the enhancement pattern seen. Lantos (15) in 1989 reported four cases of reversible cortical blindness occurring as a complication of cerebral arteriography. In his study, abnormal parenchymal enhancement was seen on CT scans within 1 hour of the event and he postulated that this was related to the hyperosmolar nature of the contrast material. Although his cases and ours suggest osmotic disruption of the BBB, physical or chemical chemotoxicity may have contributed.

Central venous catheters are inserted via an internal jugular or subclavian vein approach. An infraclavicular approach is used for the subclavian vein access and the catheter is ideally positioned at the junction of the superior vena cava and the right atrium. However, in some situations, the catheter may not be negotiated into the subclavian vein and will be directed cephalad and placed within the internal jugular vein. On plain radiographs of the neck, catheters within the internal jugular vein should be seen lying lateral to the transverse processes of the lower cervical vertebra, as opposed to the situation in our patient, in which the catheter was overlying the transverse processes (Fig 1F and G).

Normally, the vertebral artery arises as the first branch of the subclavian artery. Although the vertebral artery can be cannulated via a subclavian artery puncture, vertebral artery catheterization is usually performed via a transfemoral approach. Retrograde brachial or axillary angiography or direct percutaneous vertebral angiography are seldom used but may be employed in certain patients with severe, generalized occlusive vascular disease or extremely tortuous great vessels, or in some therapeutic embolization procedures (16).

Many new devices are available for use with central venous catheters. A stop-cock with a one-way valve was used in our patient to facilitate safe and bloodless exchange of infusion bags. This device, although a

help to nursing personnel, prevented the timely recognition of the arterial location of the catheter.

Conclusion

Osmotic disruption of the BBB should be added to the already extensive causes of pial-arachnoidal enhancement. Diligent care should be undertaken in the evaluation of neck catheters. Any catheter that overlies the transverse processes of the lower cervical vertebra may well lie within the vertebral artery rather than within the internal jugular vein.

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