

Transient Cortical Blindness Post Angiography

A Case Report

TR Clarke², P Johnson¹, D Webster¹, DT Gilbert², EN Barton²

ABSTRACT

A 56-year old female reported having had a fall two weeks prior to presentation. Computed Tomography (CT) scan showed an acute right-sided convexity subdural haematoma. A computed tomography angiogram revealed no vascular anomaly. One hour post procedure she had bilateral cortical blindness. Her vision subsequently was fully restored. A diagnosis of transient cortical blindness was made. Transient cortical blindness is a rare but recognized complication of intra-arterial injection of iodinated contrast agents.

Keywords: Angiography, blindness, cortical, transient

Ceguera Cortical Transitoria Luego de una Angiografía

Un Reporte de Caso

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RESUMEN

Una mujer de 56 años de edad reportó haber tenido una caída dos semanas antes de acudir al médico. La tomografía axial computarizada (TAC) mostró un hematoma subdural agudo en convexidad derecha. El angiograma de tomografía computarizada no reveló ninguna anomalía vascular. Una hora después del procedimiento, la paciente experimentó ceguera cortical bilateral. Su visión fue restaurada totalmente con posterioridad. Se le diagnosticó ceguera cortical. La ceguera cortical transitoria es una complicación rara, pero reconocida de la inyección intra-arterial de agentes de contraste yodados.

Palabras claves: Angiografía, ceguera, cortical, transitorio

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INTRODUCTION

Transient cortical blindness is a rare but recognized complication of intra-arterial injection of iodinated contrast agents. The symptoms are typically transient and involve visual disturbances with no impairment of ocular reflexes and extra-ocular muscle function. The fundus is also normal. There is a predilection for the vertebro-basilar arterial territory and likely involves a disruption of the blood-brain barrier with resultant transient neurotoxicity. The mechanism of causation is still not understood. We present the first reported case from the West Indies.

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CASE REPORT

A 56-year old female reported having had a fall two weeks prior to presentation. She stated that there was no associated head trauma. Subsequently, she developed headaches that acutely worsened on the morning of presentation. She denied having any loss of consciousness, seizures or visual disturbances. She, however, had nausea and vomiting. She has a longstanding history of well controlled hypertension and a three-year history of Type 2 Diabetes. She denied having migrainous headaches. On arrival, she was alert with a GCS of 15/15, she had equal and reactive pupils, normal visual acuity and field of vision, her higher mental function was intact and she had no focal neurological deficits. Initial investigations included CT brain scans as well as a CT angiogram (CTA). The plain CT scans demonstrated acute subarachnoid blood in the basal cisterns as well as a small acute right-sided convexity subdural haematoma. There was

also a small area of hypo-attenuation involving the anterior limb of the right internal capsule, which was thought to be ischaemic in nature. The CTA demonstrated no aneurysm or other vascular malformation. She was managed conservatively with phenytoin, pravastatin, nimodipine, laxatives and analgesics. Her antihypertensives were continued.

She did well for the subsequent seven days but then reported an increase in the severity of her headache. Her neurological examination remained normal. Repeat CT brain scan showed that the subdural collection was larger and now subacute. She had uneventful evacuation of the subdural collection by Burr Hole. Her headaches gradually resolved.

On her third postoperative day, she was referred for a catheter angiogram. Catheter angiography was done *via* a right common femoral arterial puncture using an 18 G needle utilizing the double wall puncture (Seldinger) method under sonographic guidance. A 5F arterial sheath was introduced. Selective catheterization of the right and left common carotid arteries and right and left vertebral arteries was done *via* a 5F JB 1 catheter over a hydrophilic wire. The contrast agent used was Ultravist 300 (iopromide 300 mg/ml); 100 mls of contrast was used in total. The procedure was uneventful until the end where the patient began experiencing nausea and hot flashes. This subsided partially after a combination of intravenous hydration and reassurance. Angiography demonstrated an approximately 1mm infundibulum at the origin of the right posterior communicating artery. No aneurysm or vascular malformation was, however, identified. The patient remained stable throughout the procedure and demonstrated no focal neurological symptoms or signs.

One hour after the procedure on returning to the ward, she had rapid deterioration in her visual acuity. This progressed to complete blindness. Associated with the visual changes were headaches, nausea and vomiting. She has no history of migraine. Her headache was generalized and there was no associated phonophobia or preceding aura. Her pupils were both equal and reactive to light, extra-ocular range of movement was normal. Her higher mental function including her level of alertness was unaffected. Her haemodynamic status was unchanged, her blood pressure remained within the high normal range (systolic pressure of 135–140 mmHg) and there was no associated bradycardia. She had no papilloedema or retinal changes. She denied having vertigo or ataxia. She had no other neurological deficits. The clinical scenario was consistent with transient cortical blindness. Other differentials such as complicated vasospastic migraine, malignant hypertension with papilloedema and an acute embolic/ischaemic event were all considered. A CT scan was done immediately after the onset of deterioration in her vision. This did not demonstrate any interval change in the appearances compared to the previous study. Transient cortical blindness secondary to angiography was considered, though an acute embolic event may have normal CT findings. A short course of steroids was initiated.

Ophthalmology consultation was sought; fundoscopy

revealed a normal optic disc and surrounding retina. Her vision began to improve 12 hours post angiography and it was fully restored after 24 hours. The diagnosis of transient cortical blindness was made. Emboli (transient ischaemia) and post angiography cortical blindness were both considered as causative factors. Magnetic resonance imaging (MRI) was not available at the time.

DISCUSSION

Transient cortical blindness represents a rare but recognized complication of catheter angiography using iodinated contrast. It has an incidence of approximately 0.3%–1% (1). It was first described in 1970 following coronary angiography (2). Many of the original case reports involved coronary angiography (1–5). A slightly higher incidence is reported with vertebral angiography (6). This rare entity is characterized by partial or complete visual disturbance (7, 8). There is characteristically no dysfunction of ocular reflexes or extra-ocular muscular function and the fundi are also typically normal (7, 8). There have been reports of headaches, changes in mental state and memory disturbances (9). Interestingly, some patients initially deny the symptoms.

Most reports documenting the imaging findings associated with transient cortical blindness involve CT (9). The positive findings typically include a gyriform hyperattenuation in the parietal and occipital regions attributed to contrast extravasation (10). There may also be associated hypo-attenuation attributed to oedema (5).

There are few reports describing MR findings (8, 9, 11). The typical findings reported describe hyper-intense changes in the cortices of the parietal and occipital lobes. One report of three cases highlighted the absence of restricted fluid motion on diffusion weighted imaging, suggesting that ischaemia is not a factor in pathogenesis (9). In fact, it is the imaging findings which have largely contributed to the current school of thought in terms of pathogenesis. Particularly, the presence of hyper-attenuation on CT, said to be secondary to contrast extravasation, is thought to be due to the effects of intra-arterial contrast agents causing disruption of the blood-brain barrier (9, 12, 13). This proposed disruption in the blood-brain barrier allows contrast to result in reversible neurotoxic effects and is therefore thought to cause the observed transient disturbances (7, 14). This phenomenon has been observed with both ionic and non-ionic contrast agents (5, 15). It is not clear why intra-arterial contrast causes this disruption of the blood-brain barrier. It is also not clear why this phenomenon has a propensity to affect the vertebro-basilar arterial territories. The majority of cases that have occurred post coronary angiography were in patients who had undergone angiography of bypass grafts where angiography of the internal mammary arteries is performed (5).

The clinical features of transient cortical blindness are similar to posterior reversible encephalopathy syndrome (PRES) which is a recently described syndrome associated

with hypertension, immunosuppression and renal impairment (16). It commonly presents with transient headache, mental disturbance and visual disturbances. The MRI findings are also similar. Though the mechanism of causation is not understood in this entity, it has been proposed that the relative lack of development of the regulatory vaso-regulatory system in the vertebro-basilar system results in the posterior hemisphere findings in that entity (17). It may be that this may also account for the findings in transient cortical blindness as well.

Though the index case did not demonstrate the positive CT findings, her clinical findings and course are typical of this rare entity.

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