CASE STUDY - ACUTE INTERNAL CAROTID ARTERY OBSTRUCTION

Clinical History

PMH of hypertension, sudden collapse, generally increased tone, bilateral upgoing plantars, miosis.

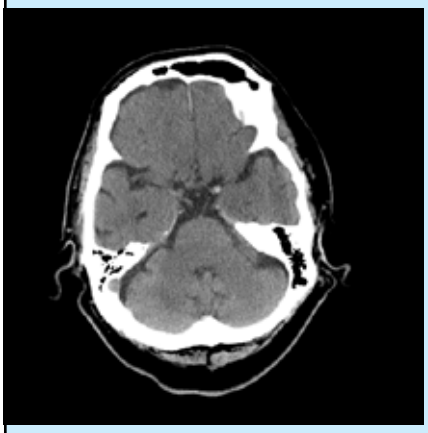

Figure 1.

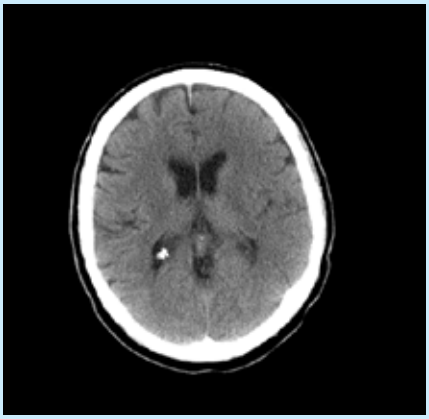

Figure 2.

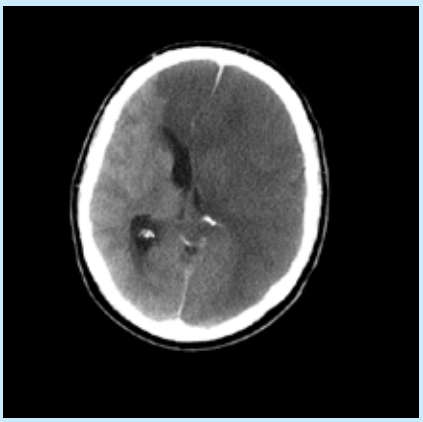

Figure 3.

Picture 1 shows an increased density of the left sided ICA (internal carotid artery) compared to the opposite side. Picture 2 demonstrates a very subtle discrepancy in the appearance of white and grey matter of the frontoparietal lobes bilaterally displaying early signs of sulcal effacement on the left side caused by cerebral oedema.

Picture 3 has been acquired 48 hours later demonstrating now a complete acute infarct of the left MCA (middle cerebral artery) as well as of left and right ACAs (anterior cerebral arteries) caused by complete obstruction of the left ICA and its branches.

Radiological Report

Comparison has been made between two CT scans being acquired in a time interval of 48 hours. The first scan, which has been acquired immediately after onset of symptoms and subsequent hospital admission, shows a dense appearance of the left sided ICA in keeping with either a thrombus or vascular stasis. There is already evidence of a subtle cerebral oedema affecting the left cerebral hemisphere resulting in early sulcal effacement.

The follow up scan 48 hours later shows now an extensive cerebral oedema involving supplying areas of left MCA as well as of left and right ACA. A complete loss of grey and white matter discrimination is noted. The cerebral oedema is resulting in a right sided midline shift with compression of left lateral and third ventricle leading subsequently in a partial obstruction of the right sided foramen of Monro and beginning dilatation of the right lateral ventricle. A generally increased intracranial pressure is noted resulting subsequently in a progressive transfalcial and transtentorial brain herniation. No haemorrhagic infarct transformation is identified.

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