

Acquired Crossed Aphasia in a Dextral

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حبة متصالبة مكتسبة في شخص أيمى

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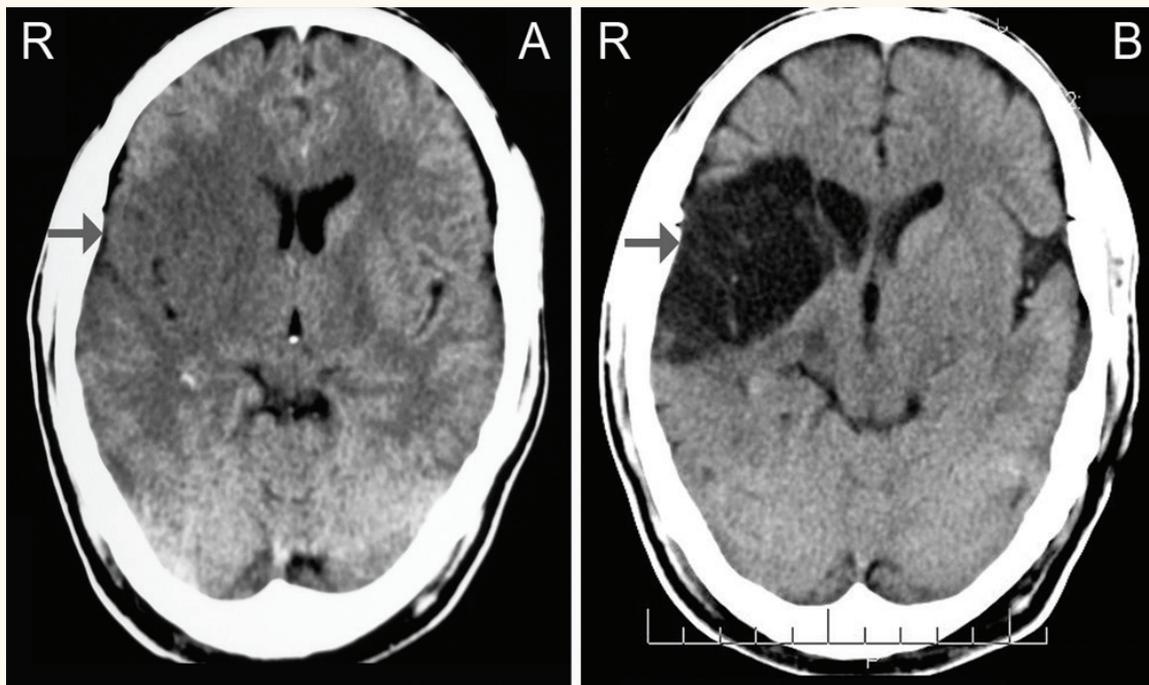


Figure 1: A: Cranial computed tomography scan obtained on the day of the stroke demonstrates ill-defined hypodensity (arrow pointing to the low signal) in the right fronto-parieto-temporal region, corona radiate and capsulo-ganglionic area consistent with a recent infarct in the distribution of right middle cerebral artery. B: The lesion is well delineated with a density similar to that of cerebrospinal fluid (arrow) along with dilatation of ipsilateral ventricle in the scan performed 7 months after the event indicating chronic infarction. Note the absence of an additional lesion in the left hemisphere in both figures A and B.

A 52 YEAR-OLD RIGHT-HANDED MAN, from South India, presented with left hemiparesis of one day duration. He had vascular risk factors such as hypertension, ethanol consumption and cigarette smoking. There was no family history of left handedness. Neurologically, he demonstrated left faciobrachial (muscle power: grade 0–1/5), crural weakness (grade 3–4/5), global aphasia, left hemisensory

impairment and left hemianopsia, with a National Institute of Health (NIH) Stroke Scale score of 19. The cardiac evaluation did not disclose valvular lesion/vegetation, mural thrombus, or significant ischaemic lesion. The computed tomography (CT) scan of his brain [Figure 1A] showed recent infarction in the distribution of the right middle cerebral artery, but there were no additional left hemispheric infarcts (also confirmed subsequently

by a diffusion weighted magnetic resonance imaging scan). As he presented beyond the time window for thrombolytic therapy, he was treated with an antiplatelet drug, statins, antihypertensive medication, speech therapy and physiotherapy. A repeat computed tomography CT brain scan [Figure 1B], performed 7 months later, demonstrated a chronic infarction in the same location with no additional left hemispheric lesion. Although his left hemiparesis had significantly improved (arm power: grade 3–4/5), his global aphasia continued to pose significant problem for verbal communication over the subsequent years.

Crossed aphasia (CA) refers to a language (symbolic communication with words) disorder resulting from a unilateral right hemispheric lesion in dextrals (right-handers).¹⁻³ CA is rare with an estimated prevalence of 0.4–3.5% of all aphasic syndromes.² In right handers, language function is often lateralised to the left hemisphere. Atypical cerebral dominance for language in our patient accounted for the right hemispheric stroke-related aphasia that was persistent and functionally disabling. The pattern of his clinical course suggested complete lateralisation of language to his right hemisphere. Apart from ischaemic strokes, other aetiologies include aneurysmal subarchnoid haemorrhage, multiple sclerosis, direct cortical stimulation, migraine with aura and focal

dementia.⁴⁻⁸

References

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