Crossed aphasia during migraine aura

Transcallosal spreading depression?

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Transient neurological symptoms in full awareness allow us to understand functional brain organization in “first hand”. Indeed, descriptions provided by patients or their families, are usually accepted as adequate evidence to localize the side of brain dysfunction during transient ischaemic attacks and partial epilepsies.

Language is lateralized to the left cerebral hemisphere in the majority of right handed individuals and crossed aphasia (CA), i.e., a language disorder secondary to a right hemisphere lesion in a dextral, is a rarity (1-4%) in otherwise healthy subjects.

Over the last 10 years we observed 5 patients with crossed aphasia during migraine auras. Patients were examined in a Headache Outpatient Clinic and systematically recorded as a result of the author’s interest in headache and behavioural neurology. None of them had any factor that could account for an atypical dominance for language, i.e., all patients were right handed, with no personal or family history of left-handedness, previous brain injury, epilepsy, developmental language disorders or learning disabilities. All had normal imaging exams (brain MRI in three and CT scan in two subjects) that excluded a structural lesion. Patients were contacted by telephone in 2005/2006. ICDH-II diagnosis criteria (1) were checked, on the account of their usual attacks. New episodes of aphasic auras were sought; visual aura symptoms were evaluated by the visual aura rating scale (VARS) (2) and right-handedness confirmed by the Edinburgh Handedness Inventory (3).

Clinical data is summarized in Table 1.
Table 1
Patients: previous headache history and relevant clinical data

<table>
<thead>
<tr>
<th>N</th>
<th>age (yrs)</th>
<th>handedness</th>
<th>Oldfield index</th>
<th>age migraine onset</th>
<th>Usual migraine attacks</th>
<th>Attacks with Crossed aphasia</th>
<th>Imaging</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>gender</td>
<td>(yrs)</td>
<td>gender</td>
<td>MWA</td>
<td>VARS</td>
<td>MwoA</td>
<td>Duration of aura</td>
</tr>
<tr>
<td>1</td>
<td>19, M</td>
<td>RH 100</td>
<td>18 yrs</td>
<td>visual, sensory aphasic aura</td>
<td>8</td>
<td>No</td>
<td>20 to 90min</td>
</tr>
<tr>
<td>2</td>
<td>46, M</td>
<td>RH 36 yrs</td>
<td>visual and aphasic aura</td>
<td>10</td>
<td>No</td>
<td>10 minutes</td>
<td>anomia</td>
</tr>
<tr>
<td>3</td>
<td>20, F</td>
<td>RH 100 yrs</td>
<td>visual and sensory aura</td>
<td>8</td>
<td>Yes</td>
<td>60 minutes</td>
<td>anomia</td>
</tr>
<tr>
<td>4</td>
<td>23, M</td>
<td>RH 80 yrs</td>
<td>visual aura typical</td>
<td>8</td>
<td>No</td>
<td>4h hours</td>
<td>cannot organize sentences paraphasias</td>
</tr>
<tr>
<td>5</td>
<td>34, M</td>
<td>RH 100 yrs</td>
<td>visual sensory aura</td>
<td>5</td>
<td>Yes</td>
<td>60-120 minutes</td>
<td>stereotype &quot;hum&quot; Paraphasias</td>
</tr>
</tbody>
</table>

M= male; F= female; RH=right handed; MWA=migraine with aura, MwoA=migraine without aura; VARS= visual aura rating scale.
CA = crossed aphasia; ER= Emergency room; CT=computed tomography; MRI=magnetic ressonance imaging
All patients described typical aphasic symptoms (disorders of expressive language, with speech reduced to a stereotype, paraphasias, neologisms, word finding or syntactic difficulties) preceded by left sided, unilateral, visual, sensory or motor symptoms that localized the dysfunction primarily to the right hemisphere. Patients either had one or more witnesses to the attack (including emergency room physicians who observed them during the attacks), that could confirm language impairment and symptom localization, or had repeated and stereotyped attacks, providing consistency about the lateralization of the aura symptoms.

Although all patients fulfilled ICHD-II criteria for migraine (with or without aura), on the account of their usual attacks, in three cases the episode with CA was beyond the expected attack duration or included motor symptoms, suggesting sporadic hemiplegic migraine. Yet, in all subjects aphasic symptoms built up in the sequence of unilateral progressive typical visual or sensory phenomena and reverted completely within 24 hours, making the diagnosis of migraine more likely than stroke. Patients with visual auras, scored above 5 points on the VARS, which predicts the diagnosis of migraine, and those with sensory symptoms had cheiro-oral or brachiolingual topography which is also typical of migraine.

Diagnosis of CA usually requires the demonstration of a unilateral lesion of the right cerebral hemisphere, i.e., it precludes its diagnosis in transient neurological dysfunctions, when patients are often observed after the end of the attack and structural lesions may not be found. This limits its diagnosis in migraine aura but should not prevent it, because there seems to be no reason to reject a type of evidence (report of symptoms, personally or by observers) that is the basis of clinical diagnosis of other conditions (TIAs, for instance).

Assuming that the phenomenon underlying the aura is unilateral, these five cases suggest that crossed aphasia can be diagnosed, on clinical grounds, during migraine auras. That assumption is now possible since functional brain imaging performed during the aura has shown that the underlying phenomenon is primarily neurogenic, unilateral, starts at the visual cortex and spreads slowly forward, corresponding to the timing of clinical symptoms (4, 5). The unilateral nature of aura has also been emphasized by the ICHD-II (1) that requires the symptoms to be unilateral.

The interest of these cases is that they clearly show that aphasia can occur in association with right hemisphere dysfunction during migraine aura. This observation may unveil pathogenic mechanisms involving the aura or the effect of repeated neuronal dysfunction in language organization.

Atypical language organization in migraine has some theoretical support, by analogy with other pathologies. Cerebral organization for language is rather plastic for language may develop in the right cerebral hemisphere in a number of conditions (early left hemisphere lesions, arterio-venous malformations and, particularly, in epilepsy, an independent factor responsible for language shift, even when the epileptic focus is localized outside the classical language areas, possibly by causing repeated transient dysfunctions of left hemisphere activity). Migraine is known to cause repeated episodes of transient neuronal dysfunction, especially during the aura, and this factor could stimulate atypical forms of neural organization. This question is relevant to functional imaging studies given the high prevalence of migraine in the adult population. It can now be clarified by functional imaging, which was not possible to perform in these patients.

An alternative explanation for CA aura is the possibility that the phenomenon underlying the aura travels between the two cerebral hemispheres. Migraine aura has
been attributed to cortical spreading depression (CSD), a phenomenon first described in rodents, where it has been observed to unfold to the other hemisphere through transcallosal fibres. So far, transcallosal spreading has not been observed in migraine. Although functional images of the aura describe the phenomenon as unilateral, or much more pronounced on the symptomatic side, most reports include few patients with typical auras, which does not cover all possible variations of this phenomenon. If CSD also moves transcallosally in humans, then crossed aphasia should not be diagnosed in this specific context.

Both hypotheses may be correct, and we cannot exclude the possibility that this group of patients is heterogeneous and include patients with atypical dominance (those with stereotyped attacks) and cases with atypical spreading (prolonged attacks).

“Bedside to bench” research is necessary and justified to understand possible variations of cortical spreading depression and language dominance in migraine.

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