Acute Corticonuclear Tract Ischemic Stroke with Isolated Central Facial Palsy

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Objective: The clinical distinction between habitual facial asymmetry, early stage peripheral facial palsy, and isolated central facial palsy is sometimes difficult. The diagnosis of acute central facial palsy is of importance to identify patients for stroke work-up and appropriate treatment. We aimed to evaluate the prevalence and localization of acute ischemic lesions associated with isolated central facial palsy. *Methods:* We screened our stroke database for patients presenting with isolated central facial palsy related to ischemic stroke between 2012 and 2017. All identified patients were comprehensively characterized including magnetic resonance (MR) diffusion-weighted imaging (DWI). *Results:* We identified four out of 5169 patients (one male; 62-83 years) with isolated facial palsy as a result of acute ischemic stroke (NIHSS 1-2). All four had circumscribed DWI lesions in different regions of the corticonuclear tract in different areas with different etiologies. *Conclusion:* Isolated central facial palsy is a rare manifestation of acute ischemic stroke and may be missed if clinical suspicion is not raised. MR-DWI identifies small ischemic lesions in the corticonuclear tract, which results in appropriate diagnostic work-up and secondary prophylaxis.

Key Words: Central facial palsy—corticonuclear tract—ischemic stroke—diffusion-weighted magnetic resonance imaging

Introduction

Patients with acute ischemic stroke often presented with minor symptoms which may be misinterpreted or overlooked. Isolated facial asymmetry is a common finding in patients who undergo careful distinct neurologic examination, and it might be difficult to distinguish between a habitual facial asymmetry, a (not yet fully developed) peripheral facial palsy and isolated central facial palsy.

Some reports have focused on findings of ischemic stroke mimicking peripheral facial palsy. This might occur in acute pontine stroke of the facial nucleus[1,2] or upper dorsolateral medulla infarction.[3] However, knowledge about isolated central facial palsy without additional symptoms is scarce. In a recent report, isolated central facial paresis (± dysarthria) has been described in seven patients with either parietal cortical ischemic lesions or in the corona radiata, which were associated with a stenosis of the middle cerebral artery (MCA).[4] This finding was in contrast to previous reports of facial paresis as a lacunar stroke syndrome,[5,6] however the distinct question of isolated central facial paresis was not addressed. We therefore aimed to evaluate the prevalence, localization, etiology, and outcome in patients with acute ischemic lesions associated with strictly isolated central facial paresis.

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Methods

We retrospectively screened the stroke database in our comprehensive stroke centre for patients with isolated central facial palsy related to ischemic stroke between 2012 and 2017. Acute central facial palsy was defined as an obvious asymmetry with unilateral impaired innervation of the facial lateral perioral muscles with a drop of the corner of the mouth, which had clearly developed within the previous 72 hours. Upper facial innervation of the front and eyelid needed to be intact and no hyperacusis or loss of taste sensation in the anterior two-thirds of the tongue should be reported. Clinical assessment was performed by a resident in stroke neurology, who was on service in the emergency room (ER). Interpretation of findings was therefore recorded according to each neurologist's judgment.

Clinical data and technical investigations from all patients had been collected according to a standardized acute stroke care protocol. Patients matching with the selection criteria were comprehensively characterized according to the National Institute of Health Stroke Scale (NIHSS), modified Rankin scale (mRS), TOAST,[7] ASCOD,[8] and imaging findings including magnetic resonance (MR) diffusion-weighted imaging (DWI). The local ethics committee (Medizinische Ethik-Kommission II, Ruprecht-Karls-Universität Heidelberg, Medizinische Fakultät Mannheim) approved the use of this data for the purpose of the study.

Results

We identified four out of 5169 patients with isolated facial paresis and ischemic stroke (NIHSS 1-2) (Table 1). All four patients had right hemispheric stroke with circumscribed ischemic lesions in the corticonuclear tract: in the precentral motor region, subcortical, and twice in the crus cerebri (Fig 1). All four patients had additional chronic ischemic lesions.

Doppler-/Duplex ultrasound revealed a stenosis of the proximal right posterior cerebral artery in patient A and a proximal vertebral artery stenosis in patient B, which were identified as sources of arterio-arterial embolism for the ischemic lesion of the crus cerebri. The other two patients had mild atherosclerosis. Transthoracal or transesophageal echocardiography revealed a small patent foramen ovale (PFO) without atrial septum aneurysm in patient C. Continuous ECG-monitoring >72 hours was unremarkable in all four patients.

All but one patient had cardiovascular risk factors (Table 1). There was no history of smoking, and no history of previous stroke/TIA. All four patients presented at the ER beyond 4.5 hours onset as reported by themselves.

Final etiologies according to the TOAST criteria were a patent foramen ovale (PFO), twice large artery disease, and small vessel disease, respectively. Using ASCOD, the

Table 1. *Clinical and imaging characteristics of four patients with isolated central facial palsy and acute ischemic stroke of the corticonuclear tract*

Patient	A	B	C	D
Sex	Male	Female	Female	Female
Age	69 years	69 years	62 years	83 years
NIHSS	2	2	1	1
Time window	72 hours	36 hours	6 hours after wake up	4.5 hours after wake up
DWI lesion	Crus cerebi	Crus cerebri; inferomedial thalamus	Cortical precentral motor region	Subcortical
FLAIR	Fazekas °I	Fazekas °III, chronic embolic ischemic lesion right PCA	Fazekas °III	Fazekas °III
T2*	No microbleeds	No microbleeds	No microbleeds	No microbleeds
ToF-MRA	Left vertebrobasilar & right PCA stenosis	Unremarkable (proximal vertebral artery not captured)	Unremarkable	Right PCA stenosis
TOAST	Large artery disease	Large artery disease	Patent foramen ovale	Small vessel disease
ASCOD	A1S3C0O0D0	A1S3C0O0D0	A3S3C3O0D0	A3S1C0O0D0
CVRF	Arterial hypertension Diabetes mellitus (HbA1c 7%) Hyperlipidemia	Hyperlipidemia	Arterial hypertension Diabetes mellitus (HbA1c 6.2%)	None

ASCOD, atherosclerosis/small vessel disease/cardioembolic /other/dissection; CVRF, cardiovascular risk factors; DWI, diffusion-weighted imaging; FLAIR, fluid attenuated inversion recovery; NIHSS, National Institute of Health Stroke Scale; PCA, posterior cerebral artery; ToF-MRA, time-of-flight magnetic resonance angiography.

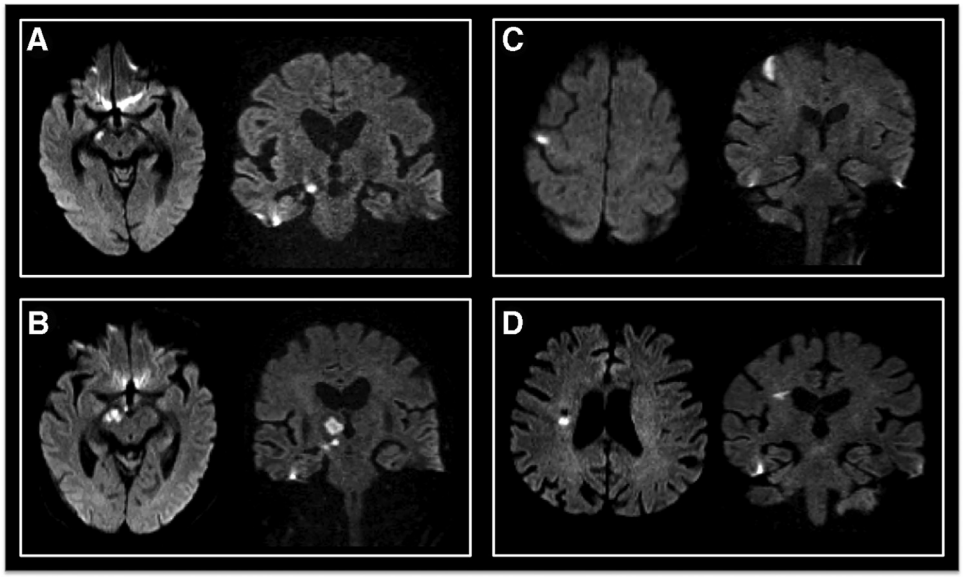

Figure 1. *Transversal/coronal diffusion-weighted magnetic resonance imaging in four patients (A/B/C/D) with isolated central facial palsy. Patient (B) has an additional lesion of the right inferomedial thalamus.*

presence of additional concomitant etiologies of lower evidence grade was found.

A secondary prophylaxis with Aspirin 100 mg and Atorvastatin 40 mg per day was initiated in all four patients. All patients had a good outcome (mRS 0 in 3, preexisting mRS 1 in patient D with macular degeneration) and could be discharged at home.

Discussion

Acute ischemic isolated central facial palsy exists, although it seems to be rare. Since we did not prospectively evaluate all patients with facial asymmetry in our ER, more precise epidemiological interpretation is difficult. This might be assessed in a prospective study in view of the very mild focal deficit that might be overlooked, when not paying attention in a specific way. These considerations might also explain, why the distinct clinical characterization of patients with acute ischemic isolated central facial paresis has only been performed scarcely. One series with 7 out of 2000 acute stroke patients has been reported,[4] however the definition included potential additional relevant dysarthria and therefore was not as strict as in our study.

The assumption that facial paresis in stroke is attributed to lacunar lesions of the pons or lenticulostriate infarcts, has recently been questioned with the identification mainly of cortical lesions associated with this clinical presentation.[4] These findings suggested that circumscribed ischemic lesions with isolated central facial paresis as a correlate of anatomical facial representation from the cortex through the corticonuclear tract clinically exists and should not be missed. In addition to cortical lesions, we now identified lesions in the crus cerebri matching with more distal affections of this tract. This adds some more detailed information of localization of the ischemia with pure central facial paresis, which had been attributed to the MCA-territory only in a previous CT-based and a more recent MRI-based study.[4,9] Considering the anatomical representation we believe, that the discussed acute lesions in our study explain the clinical presentation and are not asymptomatic concomitant lesions (as e.g., the thalamic lesion in patient B), which could lead to misinterpretations.

Although, such a mild deficit is generally not associated with a long-term relevant deficit for daily life activities, its identification is important to select concerned patients for a comprehensive stroke work-up and adopt a best medical treatment and secondary prophylaxis to prevent further major stroke events. Indeed, in all four patients a secondary prophylaxis was started and optimization of cardiovascular risk factors was enhanced. The mild clinical deficit might explain that all affected patients presented to the ER beyond 4-5 hours after onset. Different etiologies were identified in our patients, therefore central facial paresis as a clinical stroke syndrome was not especially associated with a focal MCA stenosis as had been reported previously. [4]

In cases of clinical doubts, MR imaging with DWI therefore is a useful tool to identify small ischemic lesions responsible for this particular presentation, as lesions may be missed by CT scan.

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