

Letter to the Editor

Hemiparesis due to ipsilateral supratentorial stroke: Report of two cases☆

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Corticospinal tract
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Dear Editor,

Corticospinal tract (CST) is the main motor neuronal pathway in the human brain. Almost all patients suffering from supratentorial stroke involving the CST exhibit contralateral motor weakness consistent with the contralateral projection of the CST. We report here two rare cases of patients that presented with hemiparesis associated with lesions of the supratentorial, ipsilateral CST and discuss possible pathophysiological mechanisms.

Patient 1: A 74-year-old left handed man with a history of hypertension and hyperlipidemia was admitted to the hospital for sudden worsening of left lower limb weakness that was associated with an earlier infarct involving the contralateral right internal capsule (IC). He was able to walk with a stick but could not use his left hand before admission. At the time of admission, he was unable to walk and neurological examination revealed left spastic hemiparesis, a left hemibody hypoesthesia, and pseudobulbar syndrome. Brain MRI with diffusion weighted imaging sequences (DWI) revealed a small hyperintensity in the left IC compatible with a recent lacunar infarct, ipsilateral to the weakness (Fig. 1A). Fluid-attenuated inversion recovery (FLAIR) sequences also revealed moderate leukoaraiosis, multiple residual lacunes of the brainstem and basal ganglia, and the previous lacunar infarct of the right IC (Fig. 1B). The 2-D echocardiogram disclosed an inter atrial septal aneurysm. The patient's weakness improved but did not completely resolve. The patient was discharged to a rehabilitation center.

Patient 2: A 68-year-old man was referred for sudden worsening of right hemiparesis. His medical history included hyperlipidemia, hypertension, tobacco abuse and alcoholism. He had undergone a left pneumonectomy 19 years before admission for the treatment of a squamous cell lung carcinoma. One year after pneumonectomy, a left rolandic metastasis had been discovered and surgically removed. The patient had undergone brain radiotherapy after surgery. His

neurological condition had worsened gradually for five years after radiotherapy with progressive onset of cognitive impairment, bladder dysfunction, and spastic hypertonia of the right side of the body that resulted in gait difficulties. He had been stable for many years before admission. At admission, he presented with sudden increase of right hemiparesis, preventing him for walking and feeding. Clinical examination upon admission to the hospital revealed a mute patient with a right spastic hemiplegia, bilateral Babinski's sign and swallowing disorder. The patient also demonstrated perseveration and the frontal release grasp reflex. He could not understand simple verbal orders. Brain MRI revealed a large left frontal sequelae with reactive gliosis, and a post-radiation bilateral severe leukoaraiosis (Fig. 1C). DWI sequences demonstrated a recent ischemic lacunar stroke in the right IC (Fig. 1D). Sonographic imaging of the branches of the aortic arch revealed extensive non-stenosing atheromatous lesions. The patient died five days after admission of respiratory failure.

We report here two rare cases of supratentorial stroke that produced a worsening of ipsilateral paresis. Corticospinal tract (CST) mediates voluntary movements. CST fibers course through the rostral half of the posterior limb of the IC, the intermediate three fifths of the cerebral peduncle and the basilar portion of the pons. Most axons of the CST (70–90%) cross the midline in the caudal medulla oblongata at the decussation of the pyramids to form the lateral corticospinal tract (LCST) [1]. Approximately 10% of the CST fibers do not decussate but continue to descend ipsilaterally in the ventral funiculus as the anterior corticospinal tract (ACST), ultimately crossing the midline at the level of termination. Most of the time, patients suffering from supratentorial stroke present contralateral paresis, related to contralateral projection of the CST. However, a subtle ipsilateral motor impairment may be found in patients immediately after stroke and is probably explained by ACST impairment [2]. Stroke is an important cause of long term disability with gradual recovery during the first 6 months. Precise mechanisms of motor recovery after stroke remain unclear. Mechanisms involved in motor recovery are thought to be related to functional reorganization of the affected hemisphere and contributions from projections that arise in the unaffected hemisphere and descend in the ACST [3].

In our cases, patients had previously experienced stroke or complications from metastatic disease that were responsible for contralateral paresis. Saada et al. [4] reported on 10 patients who had experienced ipsilateral hemiparesis following ischemic or hemorrhagic stroke. Similar to our first patient, 5 of these patients had experienced prior strokes involving the contralateral CST that produced hemiparesis in the distribution of the new weakness. We hypothesize that as a result of previous strokes the role of the unaffected ACST, whose fibers were no longer overshadowed by the prevailing influence of the LCST projection, were enhanced and that the new stroke, affecting previously unaffected ACST, led to a worsening of the ipsilateral motor impairment. This hypothesis is supported by fMRI studies in which patients that have shown that when gripping with the paretic hand, ipsilateral, but not contralateral, motor areas were activated [5]. By contrast, our second patient had undergone surgical ablation of a left hemispheric metastasis

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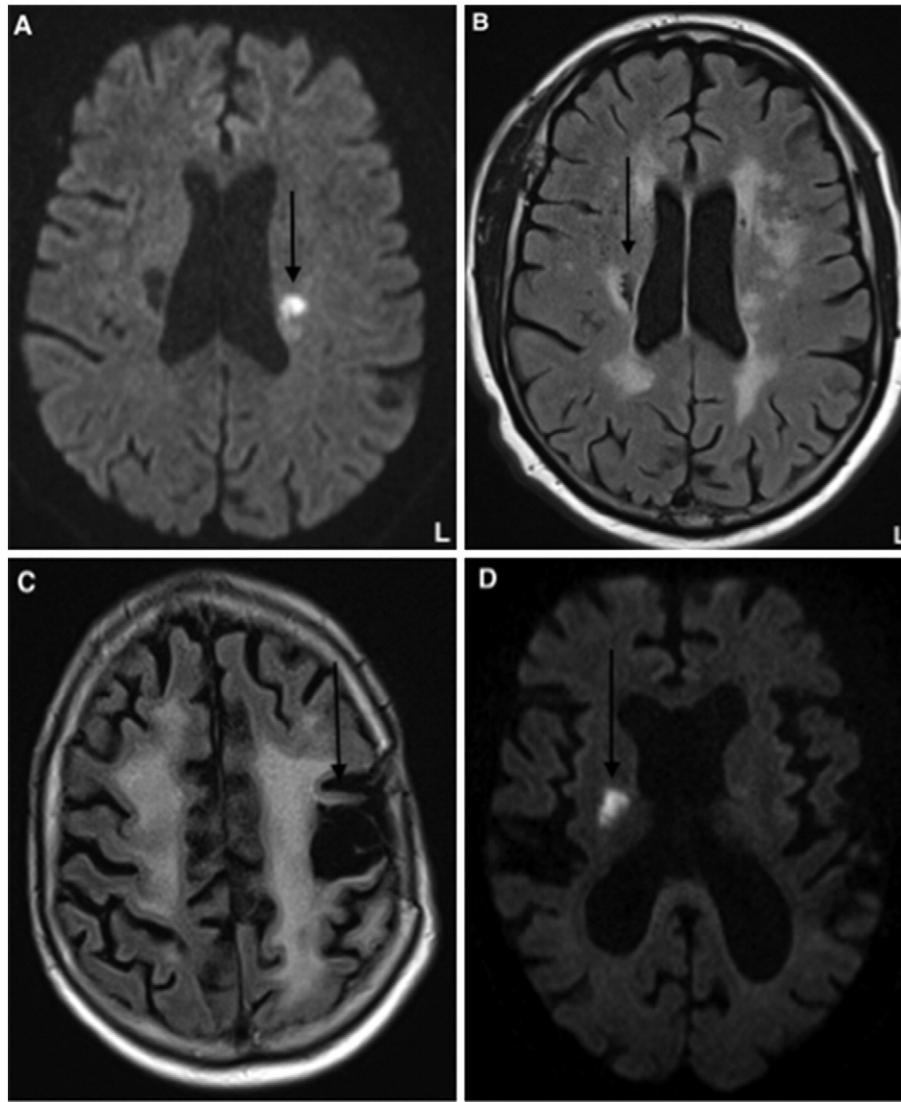


Fig. 1. (A) DWI sequence demonstrates a small area of restricted diffusion in the left IC (arrow) suggesting a recent lacunar infarct. (B) FLAIR sequence shows a previous lacunar infarct in the right IC (arrow). (C) FLAIR sequence shows the left frontal sequelae of metastasis surgery with reactive gliosis (arrow) and bilateral radiation-induced leukoencephalopathy. (D) DWI sequence demonstrates a small area of restricted diffusion in the right IC compatible with recent ischemic lacunar stroke (arrow).

and had suffered from severe radiation-induced leukoencephalopathy prior to the worsening of his right hemiparesis from his right IC infarct. Underlying pathophysiological mechanisms are nevertheless probably the same.

The five others patients described in Saada's article had experienced first onset of paresis revealing ipsilateral supratentorial stroke. In two of them there is no clear explanation for the onset of ipsilateral hemiparesis. In three other patients, associated congenital abnormalities were reported. Patients with congenital lack of decussation of CST have been described and they often presented with associated congenital abnormalities such as scoliosis, agenesis of corpus callosum, posterior fossa malformations, Dandy Walker malformations, and Joubert or Mobius syndromes. In these patients, stroke is responsible for an ipsilateral hemiparesis. In our cases, the hypothesis of an uncrossed CST cannot be raised because our patients had previously presented hemiparesis following contralateral hemispheric injury.

In conclusion, our observations support the explanation that loss of supporting projections in the ACST following infarction are responsible

for the exacerbation of preexisting motor weakness caused by prior damage to the contralateral CST.

Disclosures

All authors report no disclosures.

Authors' contribution

Benjamin Hebat: acquisition of data, analysis and interpretation, critical revision of the manuscript for important intellectual content, study supervision.

Yvan Vaschalde: acquisition of data, analysis and interpretation, critical revision of the manuscript for important intellectual content, study supervision.

Jean-Philippe Cochin: drafting the work or revising it critically for important intellectual content.

Clémence Hardy: drafting the work or revising it critically for important intellectual content.

Romain Lefaucheur: acquisition of data, analysis and interpretation, critical revision of the manuscript for important intellectual content, study supervision.

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