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LETTER TO THE EDITOR

Cerebral venous thrombosis: an unexpected Brissaud-Sicard syndrome mimicker

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Dear Editor-in-Chief,

The constellation of hemifacial spasm (HFS) and contralateral hemiparesis was initially mentioned in the literature by the distinguished French physician and pharmacologist Adolphe-Marie Gubler (1821–1979) in 1856. Nevertheless, Brissaud-Sicard syndrome (BSS) bears the name of two later French physicians, Édouard Brissaud (1852–1909) and Jean-Athanase Sicard (1872–1929), who in 1908 described in detail three patients that presented with HFS and contralateral hemiparesis in the context of syphilis and secondary pontine vascular involvement [1].

BSS semiology is typically attributed to brainstem pathology. More specifically, it constitutes an extremely rare pontine-crossed syndrome [2] as since its original publication only 2 cases of pontine stroke and one of diffuse brainstem glioma causing BSS are mentioned [1]. We hereby present a unique case of BSS mimicker that does not abide to the aforementioned classic localization.

Case report

A 55-year-old male presented to our emergency department with a 24-h history of new onset headache and altered level of consciousness (GCS 11/15). His past medical history was unremarkable. Recent COVID-19 infection or vaccination was not mentioned. He was afebrile with no signs of meningeal irritation. Neurological examination revealed right HFS not accompanied by facial paresis or diminished corneal reflex and contralateral hemiparesis (MRC grade 3/5) alongside brisk tendon reflexes (+3) and Babinski's sign. Sensory examination including light touch, pinprick, vibration, and proprioception was unremarkable.

Due to the fact that BSS was suspected, urgent brain MRI was performed in order to trace relevant pontine pathology. Interestingly, an extensive right cerebral hemorrhagic infarct sparing brainstem secondary to transverse, sigmoid sinus, and internal jugular vein thrombosis was imaged (Fig. 1). Thus, the patient was diagnosed with cerebral venous thrombosis (CVT). In an attempt to exclude epilepsia partialis continua causing right facial cramps urgent EEG was also ordered, but did not reveal epileptiform activity.

Lower extremities' venous ultrasound was unremarkable. Hypercoagulable workup including homocysteine, antithrombin III, protein C, and protein S deficiency; lupus anticoagulant, antiphospholipid, and anticardiolipin antibodies; factor V Leiden gene mutation and resistance to activated protein C; and prothrombin G20210A mutation was negative. Tumor markers (CA 125, CA 15.3, CEA, PSA), thorax, and abdominal CT scans were negative. Blood testing for autoimmune rheumatologic diseases (RF, C3, C4, pANCA, cANCA, ANA), serum protein electrophoresis, and immunofixation were also normal.

HFS did not respond to treatment with either carbamazepine or levetiracetam. Anticoagulant treatment, initially with therapeutic dose of tinzaparin (14,000 IU daily) and oral warfarin afterwards, was commenced. On 3-month follow-up, the patient's symptoms, including HFS, were fully resolved.

Discussion

We present a rare case of BSS mimicker due to CVT resulting in extensive right cerebral hemorrhagic infarct able to explain contralateral hemiparesis, but not ipsilateral HFS.

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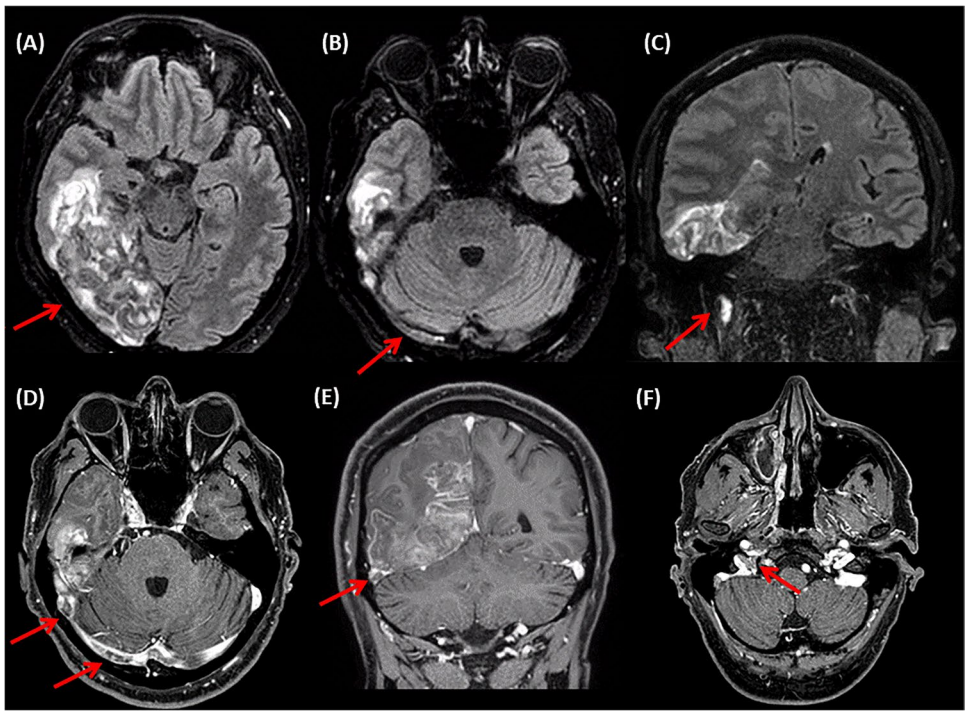

Fig. 1 Axial and coronal FLAIR (top row) and T1W post-CM (low row) images demonstrating extensive right temporo-occipital infarct (**A**), absence of expected flow void in ipsilateral transverse sinus (**B**) and internal jugular vein (**C**), and intraluminal enhancement defect in right proximal/distal transverse (**D**), sigmoid sinus (**E**), and internal jugular vein (**F**)

HFS is most often due to aberrant/ectatic blood vessels in the posterior cranial fossa causing facial nerve root exit zone (FNREZ) compression. Offending vessels in descending frequency are the anterior inferior cerebellar artery, posterior inferior cerebellar artery, and vestibular artery. Dual compression of FNREZ by arteries and veins is mentioned; however, HFS caused solely by a venous offender is extremely rare, as only few cases associated with brainstem developmental venous anomaly [3] and a single case of HFS related to straight sinus occlusion in a 10-month infant [4] are mentioned. Congested and dilatated brainstem veins lying in close proximity to the FNREZ could be the irritants. Such is the vein of the middle cerebellar peduncle that successively drains into the superior petrosal vein, the superior petrosal sinus, and the transverse sinus, the latter being thrombosed in our case [5].

Conclusion

To our knowledge, this is the first case report of a patient presenting with BSS semiology related to CVT and not intristic pontine pathology. Moreover, it constitutes the first reported case of HFS complicating CVT in adults.

Supplementary Information The online version contains supplementary material available at https://doi.org/10.1007/s10072-022-05977-1.

Data availability Available upon reasonable request.

Declarations

Ethical approval All procedures performed in the studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards.

Informed consent Informed consent was obtained from the patient.

Conflict of interest The authors declare no competing interests.

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