L’IRM est actuellement l’examen de référence pour le diagnostic de spondylodiscite en raison de sa sensibilité et de sa spécificité avoisinant les 95 % [3]. Elle montre précocement l’infiltrat inflammatoire des disques et des plateaux vertébraux adjacents (avec classiquement une diminution du signal en séquence pondérée T1 et une augmentation du signal en séquence pondérée T2) ainsi que l’envahissement des parties molles. Elle permet également de distinguer des remaniements postopératoires normaux et des anomalies dues à une infection du rachis [15]. La détection de collections abcédées péri-vertébrales, épipéridurales et intradurales (en hypersignal T2) ainsi que le rehaussement en miroir du disque et de l’os sous-chondral après injection de produit de contraste confirment le diagnostic [15]. En cas de doute diagnostique, comme chez notre patient, il peut être utile de renouveler l’IRM : les modifications de l’imagerie étant également évocatrices d’une cause septique. À notre connaissance, c’est la première fois que l’on décrit une IRM rachidienne sans signe d’infection à près de quatre mois de la chirurgie et après environ quatre semaines du début de la recrudescence des lombalgies. L’IRM pathologique chez notre patient, il peut être utile de renouveler l’IRM : les modifications de l’imagerie étant également évocatrices d’une cause septique. À notre connaissance, c’est la première fois que l’on décrit une IRM rachidienne sans signe d’infection à près de quatre mois de la chirurgie et après environ quatre semaines du début de la recrudescence des lombalgies. L’IRM pathologique à cinq mois nous permet de souligner l’impression clinique de virulence faible et lente de \( P. \text{acnes} \). Il faut donc savoir évoquer ce diagnostic, lors d’antécédents de chirurgie ou de ponction tardives. Ceci illustre la faible virulence du germe et l’intérêt de demander une IRM, au moindre doute clinique de spondylodiscite.

**Conclusion**

Nous rapportons un nouveau cas de spondylodiscite iatrogène à \( P. \text{acnes} \) chez un sujet sans comorbidités. Cette présentation clinique est originale en raison des modifications IRM tardives. Ceci illustre la faible virulence du germe et l’intérêt de demander une IRM, au moindre doute clinique de spondylodiscite.

**Conflicts of interests : aucun**

**Références**


**Marchiafava-Bignami disease complicating SC hemoglobin disease and Plasmodium Falciparum infection**

Maladie de Marchiafava-Bignami compliquant une crise vaso-occlusive de drépanocytose SC au cours d’un accès palustre à *Plasmodium Falciparum*

SC hemoglobin disease (HbSC) is a prevalent hemoglobinopathy, in which equal concentrations of HbS (beta6Glu6Val) and HbC (beta6Glu6Lys) coexist. HbC enhances the pathogenic properties of HbS, resulting in a clinically significant disorder
All complications that are found in patients with sickle cell anemia (HbSS) including hemolysis, vasoocclusive disease, infection and proliferative retinopathy may occur in individuals with HbSC disease [1]. Neurologic complications include paralysis, seizures and death. Stroke is a leading cause of neurologic morbidity and mortality affecting two to three percent of all HbSC disease patients [2]. Brain magnetic resonance abnormalities may also be present without a clinical history of cerebrovascular accident [3]. Such complications may be triggered by dehydration and fever. Cerebral malaria due to *Plasmodium falciparum* infection is one of the recognized causes of acute vasoocclusive disease in which sequestration of infected erythrocytes into cerebral vessels leads to a severe encephalopathy [4]. We report herein on a SC hemoglobin patient who suffered from Marchiafava-Bignami disease (MBD) in a context of cerebral malaria.

**Case report**

A right-handed 37-year-old man born and living in Burkina Faso was referred because of headache. Past history was remarkable for SC hemoglobin disease. The patient had complained from recurrent episodes of painful crisis and suffered several acute chest syndromes. Multifocal osteonecrosis involved the right femoral head, right humeral head and lumbar spine. He denied alcohol or tobacco abuse. The patient was admitted because of headache and loss of consciousness with a presumed diagnosis of cerebral vasocclusive crisis. Malaria was ruled out by two negative blood smears. The patient had analgesic therapy and erythrocyte support because of anemia (haemoglobin = 84 g/L). Three days later, aphasia and left hemiparesia developed. Blood cell counts were: haemoglobin 93 g/L, leukocytes 10600/mm³, platelets 72 000/mmm³. A cerebral CT scan with contrast medium was normal. Seven days after the admission haemoglobin was 69 g/L, platelet count was 21 000/mm³. Blood smear found 240 *Plasmodium Falciparum* trophozoites/mm³. Quinine therapy and erythrocyte and platelet supports were started. The patient was referred to our hospital. On admission, vital signs were blood pressure (BP) 146/72; pulse 94/min; temperature 38.5 °C (101.3 °F) and respiration rate 24/min. The patient was cooperative and fully oriented. Glasgow Coma Scale was 13/15. Speech was impaired with global intact comprehension. Left pyramidal signs were present.

**Figure 1**

Cerebral magnetic resonance imaging (MRI)

A: numerous T2* susceptibility-weighted hypo-intensities can be elicited (arrows) in the posterior arm of the corpus callosum (arrowhead). B: T2-FLAIR corresponding images.
including spastic hemiparesia, increased tendon reflexes and extensor plantar response. Sensation was intact throughout to light touch, pinprick, temperature sense, vibration, and proprioception. Romberg was negative. Cerebellar signs were absent. Cranial nerves were normal. Incomplete interhemispheric disconnection syndrome with left ideomotor apraxia and left tactile anomia was noted. Lungs were clear to auscultation and percussion bilaterally. Cardiovascular system revealed regular rate and rhythm without rubs, gallops, or murmurs. Abdomen was soft, nontender with normal bowel sounds and no bruits. Spleen was not enlarged. No significant hypo- or hyper-pigmented skin lesions were noted.

Blood tests showed: haemoglobin 92 g/L, leukocytes 8600/mm³, platelets 64 000/mm³, 192 erythroblasts/100 leukocytes, 50 000 reticulocytes/mm³. There was no schizocyte. Haemoglobin electrophoresis found 58% of haemoglobin A1, 14% of haemoglobin S, 14% of haemoglobin C. Mild hepatitis (ASAT = 2N, ALAT = 2N, GammaGT = 10N, Alkaline Phosphatase = 2.5N) was present. Blood clotting tests were normal. Fibrinogen was 7.6 g/L, C reactive protein was 26 mg/dL, haptoglobin was 0.23 g/L (N 0.67-1.79). Thin and thick smears were negative. Cerebrospinal fluid (CSF) examination was normal except for protein level measured at 61 mg/dL. Bone marrow aspiration and biopsy found bone marrow necrosis. Screening for other infectious agents including legionella, salmonella, chlamydiae, syphilis, hepatitis B and C viruses, human immunodeficiency virus (HIV) 1 and 2, parvovirus B 19 was negative.

Brain CT scan with contrast medium was normal, including venous reconstruction pictures. Brain MRI performed 9 days after the first admission found numerous T2* susceptibility-weighted hypointensities associated with an hemorrhage of the posterior arm of the corpus callosum evocative of MBD (figure 1).

The patient was considered having severe vaso-occlusive cerebral crisis complicated with MBD, bone marrow necrosis and probable fat embolism triggered by *Plasmodium Falciparum* infestation. He was then treated with quinine 1500 mg/d, folic acid 10 mg/d, omeprazole 20 mg/d and intravenous thiamine supplementation 1 g/d.

Quick improvement was obtained with stable apyrexia, complete regression of pyramidal tract lesions and slow resolution of interhemispheric disconnection syndrome. Language parameters and gait were back to normal. Cerebral MRI performed at 3 weeks showed partial regression of the lesions. The patient was symptom-free when he was discharged a month and a half after the first admission. He went back to Burkina Faso.

**Discussion**

Acquired lesions of the corpus callosum include stroke, diffuse axonal injury, multiple sclerosis, hydrocephalus, acute disseminated encephalomyelitis, lymphoma, glioblastoma, hemartoma and Marchiafava Bignami Disease resulting in different MRI patterns [5].

Despite radiological heterogeneity, callosal diseases share a common clinical picture. The complete interhemispheric disconnection syndrome observed after callostomy include hemi-alexia, unilateral verbal anomia, ideomotor apraxia, agraphia, tactile anomia, constructional apraxia, lack of somesthetict transfer and dissociative phenomena. Usually there’s a left side involvement in right-handed patients, secondary to disruption of left hemisphere i.e. dominant hemisphere containing language and language dependant memory coming fibers [6].

Marchiafava-Bignami Disease belongs to the spectrum of callosal diseases and was first thought to be a primary degeneration of the corpus callosum. The disease was first noted in middle-aged and elderly Italian men who consumed red wine before being described in the general setting of chronic alcohol consumption [7]. Nutritional deficiencies also have been implicated since cases of MBD have been reported in anorexia nervosa and severe malnutrition. Moreover, substantial clinical and radiological improvement has been observed after high-dose thiamine administration [8].

The *sine qua non* pathological condition of MBD is necrosis of the medial zone of the corpus callosum whereas the dorsal and ventral rims are usually spared. The lesions arise as small symmetric foci that extend and become confluent. Although medial necrosis of the corpus callosum is the main finding, other features are described including degeneration of the anterior commissure, the posterior commissure, centrum semiovale, subcortical white matter, long association bundles, and middle cerebellar peduncles. All these lesions have a constant bilateral symmetry. The internal capsule, corona radiata, and subgyral arcuate fibers are spared. The gray matter is not grossly affected. The microscopic alterations are the result of a sharply defined necrotic process with loss of myelin but preservation of axis cylinders in the periphery of the lesions. There is usually no evidence of inflammation aside from scarce perivascular lymphocytes. Fat-filled phagocytes are common. Gliosis is usually not prominent. Capillary endothelial proliferation may be present in the affected area [7]. Ischemic edema is another pathological hallmark of the disease. However, steroids have no clear-cut efficacy in the treatment of MBD [9].

Our patient suffered from severe HbSC vasoocclusive crisis involving the central nervous system. The triggering event was a *Plasmodium falciparum* infestation. Left tactile anomia was caused by callosal necrotic lesions as seen on brain MRI. Cerebral malaria and fat embolism secondary to bone marrow necrosis could also have contributed to neurological symptoms such as impaired consciousness, left hemiparesia and aphasia. A recent report described three patients suffering from cerebral malaria with T2-weighted foci hyperintensities involving the...
splenium of the corpus callosum and presenting with sensorial impairment [10].

Acute callosal lesions mimicking MBD have not been described in the setting of HbSC disease. In a series of 28 children with sickle cell disease, corpus callosum size assessed by MRI was smaller for children with silent infarcts (n = 8) or overt stroke (n = 8) than for those without visible infarcts (n = 12) or control participants. Moreover, lesions’ volume was a robust predictor of IQ and other cognitive scores [11]. Because it is a silent feature, involvement of the corpus callosum could be under-diagnosed in HbSC patients.

Hence, vaso-occlusive disease seen in HbSC patients may cause MBD particularly in the context of cerebral malaria. To our knowledge, acute callosal lesions have never been reported in HbSC/sickle cell patients [3]. Clinical screening for interhemispheric disconnection syndrome may lead to identify sickle cell/HbSC disease patients who require more aggressive treatment, such as preventive red blood cell transfusions in order to avoid cognitive function decline [12].

Conclusion
We report the first case of acute vaso-occlusive callosal crisis in the setting of HbSC hemoglobin disease and cerebral malaria. Subtle signs of interhemispheric disconnection syndrome should be sought in such clinical setting and brain MRI with coronal views for corpus callosum analysis should be obtained quickly because of potential therapeutic implications. Marchiafava Bignami disease belongs to the phenotype of vaso-occlusive crisis associated with SC hemoglobin disease.

Conflict of interest: none.

References

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