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## Letters to the editor

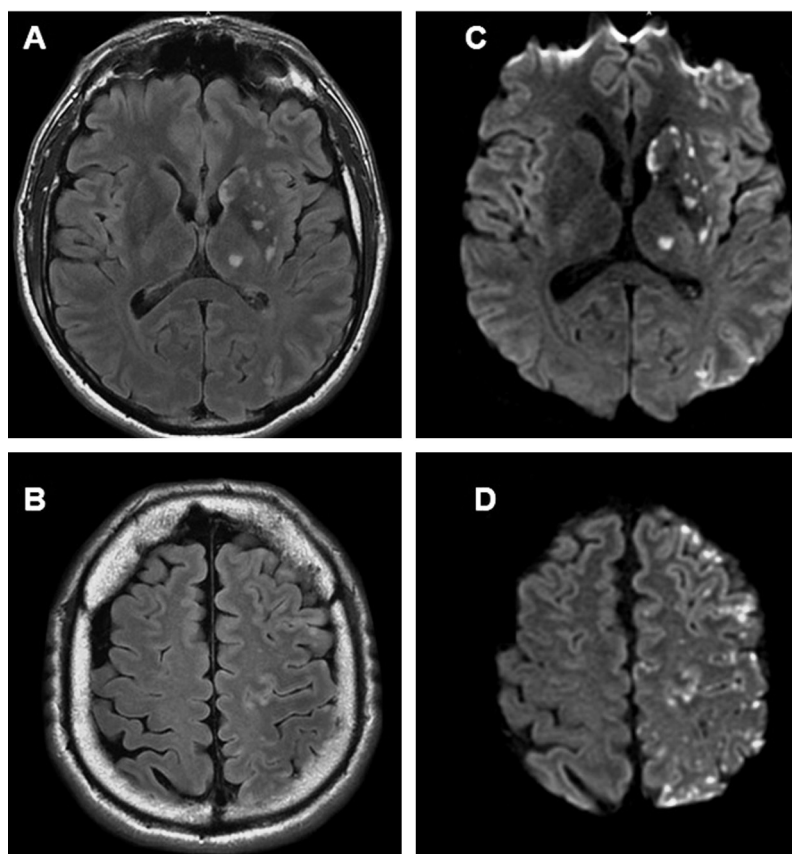
### Embolitic stroke associated with intra-carotid injection of buprenorphine

#### *AVC ischémique secondaire à une injection intracarotidienne de buprénorphine*



A French, 40-year-old Caucasian man was admitted in our stroke unit for right hemiparesis and aphasia. This right handed patient without prior history of atherosclerosis and smoking as the only cardiovascular risk factor was a parenteral heroin and buprenorphine user who admitted to

multiple injections. On admission, toxicology showed the presence of buprenorphine only, despite a large serum and urine screening (other opiates, amphetamine, ecstasy, cannabis, benzodiazepines, and cocaine). The patient had a history of injection into the neck. One of these injections was complicated 1 year earlier by an abscess on the right part of the neck. The day before admission, the patient had lost consciousness and could neither remember nor describe the circumstances of this event. On regaining consciousness, he showed clinical features of hemispheric syndrome including right hemiparesis with severe deficit of the upper limb, hemisensory loss and aphasia (NIHSS = 14). MRI revealed multiple hyperintense abnormalities on T2-weighted images and diffusion-weighted imaging (DWI). These small (less than



**Fig. 1** – A and B. FLAIR MRIs show scattered hyperintense lesions in the basal ganglia, white matter, and cortex of the left cerebral hemisphere consistent with acute embolic infarction in the left internal carotid artery territory. C and D. Corresponding diffusion-weighted imaging shows corresponding hyperintense lesions, without any lesion in the contralateral hemisphere.

0.5 cm diameter), discrete lesions were scattered throughout the cortex, white matter and basal ganglia but strictly limited to the left internal carotid artery territory. The lesions were better visualized on DWI compared to FLAIR images, especially the smallest punctate lesions (Fig. 1). Magnetic resonance angiogram of the carotid arteries and of the circle of Willis was unnoticeable (especially no dissection). Ultrasound did not reveal any plaque on the carotid artery. Although the patient denied injecting into the neck, lesions proved that he did. He also admitted that he was still a parenteral buprenorphine user. Moreover, other etiologies were ruled out (no cardiac valve vegetations, no heart rhythm abnormalities, no hepatitis B or C, no HIV and no sign of autoimmune disease). The patient was treated with antiplatelet therapy (lysine acetylsalicylate, 250 mg/day) and was discharged on day 11 while he was still presenting a clumsy right hand and a mild aphasia. It is noteworthy that 5 months after this neurovascular event, the patient was admitted again in our university hospital for critical lower limb ischemia secondary to intra-femoral crushed zolpidem injection.

Ischemic strokes are usually due to endogenous mechanisms such as cardiovascular or clotting disorders. Nonetheless, exogenous mechanisms cannot be completely excluded. Seven cases of carotid embolism in buprenorphine injectors have been described and an initial loss of consciousness was described in 6 of these patients, as it is the case in our patient [1]. It is common for injecting drug users to prepare injections by crushing tablets, which are not designed for parenteral administration. The injection of insoluble tablet excipients, such as talc or microcrystalline cellulose, can lead to serious local and systemic complications [2]. Accidental intra-arterial injection of drugs is a sporadic complication in IV drug addicts. Its most common ischemic complication consists in acute limb ischemia [3,4]. Inadvertent injection of dirt could also be causative of arterial embolism. To sum up, this means that neurologists and radiologists should be aware of the characteristic MRI findings of carotid artery embolic infarction in parenteral drug users that inadvertently or intentionally inject into carotid arteries.

## Disclosure of interest

The authors declare that they have no conflicts of interest concerning this article.

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## Mydriase unilatérale révélant une neuroborréliose de Lyme



### Unilateral mydriasis in Lyme neuroborreliosis

Un homme de 60 ans, alsacien, présentait une mydriase unilatérale indolore, non fluctuante, notée fortuitement en se regardant dans un miroir. L'interrogatoire ne révélait aucune prise de toxiques ni application de topiques. Il n'y avait pas de céphalées ni aucun autre symptôme associé du point de vue neurologique, ni arthralgie ou éruption cutanée. À l'examen ophtalmologique, on notait une disparition du réflexe photo-moteur de l'œil en mydriase sans autre anomalie et du point de vue neurologique l'examen clinique était normal. Les examens pratiqués ont retrouvé : une sérologie de Lyme dans le sang positive avec des IgM à 21 U et des IgG < 4 U/mL, un western blot avec 10 bandes ; une protéinorachie à 0,46 g/L sans cellules. La sérologie dans le LCR était également positive avec des IgG à 36 U/mL, un western blot avec 1 bande et un index de synthèse intrathécale d'Ig anti-Borrelia burgdorferi à 5,45 (normal < 1,5). Le reste du bilan était normal. Une IRM cérébrale et des troncs supra-aortiques injectée s'est avérée