The second specificity is related to the morphology of the aneurysms. The two aneurysms reported here were sacciform and larger than 20 mm. A combination of these characteristics could increase the tendency of intra-aneurysmal thrombus formation and further thromboembolism. Indeed, it is likely that the larger the vascular cavity, the higher the risk of thrombus generation. Besides, the sacciform shape creates a vascular cavity connected to the normal arterial flow with a higher tendency to induce flow stagnation than a fusiform dilation. In their studies, Guillon et al. [2] and Touzé et al. [3] reported a saccular shape in 11 of 16 and in 13 of 36 aneurysms, respectively; however, the size of the aneurysms was not specified.

Technique of ICA Repair
Because surgical carotid repair carries an important risk of injury to the lower cranial nerves [11], endovascular treatment was preferred in both patients. Case 1 was treated in 1997, and at that time, we did not have long-term follow-up of carotid stenting for dissection. Because the patient had shown recurrent thrombus migration, parent artery occlusion appeared the surest way to protect her against further embolism. However, in light of results obtained in stenting of ICA dissection [12–15], a carotid stenting would certainly be proposed now at our institution, as was the case for patient 2.

Conclusion
Although the number of cases is limited, our observations show that late thromboembolic complications may occur in ICA dissecting aneurysms. Accordingly, a persisting aneurysm justifies at least lifetime antiplatelet treatment. Endovascular carotid repair could be considered in the case of large sacciform aneurysms, especially when they contain thrombus.

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References
The patient had no significant comorbidity, did not smoke, use alcohol or take illicit drugs. He had chewed khat regularly since the age of 30 and continued this habit after moving to Belgium. His father died at the age of 70 and had severe arterial hypertension. One brother died in his fifties due to uncontrolled arterial hypertension. Another brother had a left hemiparesis of unknown cause. They all had been chewing khat regularly.

On admission, the patient had a normal electrocardiogram and normal routine blood tests. The blood pressure was 190/95 mm Hg. A brain CT scan disclosed a right-sided infarction in the territory of the middle cerebral artery (fig. 1). Routine cardiovascular work-up (echocardiography, Doppler neck vessels and monitoring) was unremarkable. Thorough further investigations for causes of stroke in young patients were all normal, including lactate, haemoglobin electrophoresis, lupus anticoagulant, anticardiolipin antibodies, cryoglobulins, homocysteinaemia, protein-C or protein-S deficiency, APC resistance and factor V Leiden mutation. Hepatitis C, HIV serology, VDRL and TPHA were negative. Hepatitis B virus-PCR was positive (6,100 genomes/ml). Aspirin therapy was started. The arterial hypertension normalised during the hospital stay, partly by stopping khat, partly by association of amlodipine. He recovered after months, with a left hemiparesis of upper limb MRC 4 and lower limb MRC 5.

Eighteen months later, he was re-admitted with a complete left hemiplegia. Medication intake had been irregular. The patient confirmed that the new episode also occurred shortly after khat chewing. A CT scan showed no changes. MRI of the brain 11 days after the second stroke episode show a larger middle cerebral artery infarction and diffuse white matter abnormalities.

The patient completely recovered. Since stopping khat chewing, no further cerebrovascular events have occurred during 3 years of follow-up.

**Discussion**

**Prevalence and Pharmacology of Khat Chewing.** Khat chewing has been a social habit in African countries for centuries: 5–20 million people are regular khat users [1–3]. In Yemen, 92% of the khat chewers consume khat daily [2, 3]. Even women consume khat. The prevalence of khat use in Western Europe is unknown, though it is becoming more common due to immigration.

Khat consists of cathedulines, phenylpentenyl-amines and cathines. One of the cathines is (–)-S-cathinone. This most important component of khat is a sympathomimetic amine with similar structure and pharmacological properties as amphetamine [4].

**Pathogenesis of Khat-Related Disease.** Amphetamine causes a release of neurotransmitters in the dopaminergic and serotonergic synapses [1]. Kalix [4] found cathinone to stimulate the release of dopamine and serotonin.

The main central nervous system effects of khat are comparable to those of amphetamines: euphoria, hyperactivity, hyperexcitability, insomnia, anxiety, paranoia and psychosis [1, 4]. In the peripheral nervous system khat causes release of norepinephrine, which leads to arterial hypertension, and increases heart rate, body temperature, respiration rate and causes renal vasoconstriction [5].

The most frequently reported cardiac complication of amphetamine-like substance abuse is acute myocardial infarction [6] by coronary vasospasm, catecholamine-mediated platelet aggregation and increased myocardial oxygen demand [3, 6]. Arrhythmias also occur. Similar cardiac abnormalities have been reported after abuse of 3,4-methylenedioxyxymethamphetamine, cocaine [3] and khat [2, 5].

Because of the resemblance between the central and cardiovascular complications of khat and amphetamine, one can assume that there is also a higher risk of cerebrovascular events in khat chewers. Bruno [7] and Miranda and O’Neill [8] have observed stroke-like cerebral complications in patients abusing sympathomimetic drugs like cocaine and amphetamines. Nizar [9] alluded to a possible association between khat use and brain disorders, including migraine and cerebral haemorrhage, secondary to bouts of arterial hypertension. There have been no reports on an association between khat use and major ischaemic stroke.

**Fig. 1.** CT scan, performed several hours after the first stroke episode, shows a right-sided middle cerebral artery infarction.

**Fig. 2.** Proton-density images of MRI of the brain 11 days after the second stroke episode show a larger middle cerebral artery infarction and diffuse white matter abnormalities.
There is another argument suggesting that abuse of sympathomimetic drugs like khat is associated with cardiovascular events: the circadian rhythm of people suffering thrombotic events is different from that of normal controls.

Studies such as ISIS-2 have consistently shown a morning peak in the incidence of acute myocardial infarction, which is related to an increased sympathetic outflow and circulating catecholamines at this time of the day [10]. Alkadi et al. [5] studied the occurrence of myocardial infarctions in khat users and found that 59% of myocardial infarctions developed between late afternoon and midnight. They found that the myocardial infarction incidence was increased three-fold [5]. Pharmacokinetic studies have shown a peak plasma concentration of cathinone 2.1–2.3 h after chewing [1, 11].

Present Case. Our patient developed recurrent large vessel cerebral disease. Both strokes occurred in the evening, after khat chewing. Alternative causes for stroke in young adults were carefully excluded. Apart from khat chewing, this young patient had no major risk factors for stroke, although he reported a familial history of severe arterial hypertension and neurologial deficit in family members who also regularly chewed khat. A genetic background that renders people vulnerable to khat-induced vascular changes, as suggested by Alkadi [5], cannot be ruled out. After stopping khat chewing and taking aspirin regularly, no further cerebrovascular events occurred. We therefore conclude that our observation is highly suggestive of khat use being causally associated with ischemic stroke. As we live in a multicultural society, we want to highlight this possible neurological manifestation of a social habit amongst immigrants.

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