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Fatal stroke in a young cocaine drug addict: chemical hair analysis and cervical artery examination twenty months after death

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Abstract

We present a case of a 26-year-old female who died of acute cerebral infarction after thrombosis of the left internal carotid artery, conceivably related to cocaine use. The forensic examination was performed only twenty months post-mortem. Revaluation of clinical data was carried out after exhumation and forensic autopsy examination were done, including anatomic dissection of cervical vessels and histological and toxicological analyses. Interestingly, comparative histological examination of cervical arteries was more useful in determining the putative site of vascular damage than gross and histological examination of the brain itself, although the state of preservation of tissues was poor. In conclusion, when a vascular accident is suspected or has to be demonstrated, we suggest performing comparative histological examinations of selected artery samples, even several months after death.

Key words: *cocaine, histological examination, stroke, hair analysis, exhumation.*

Introduction

Cocaine use has been associated with ischaemic stroke due to vasospasm of large arteries and secondary intravascular thrombosis [6,9]. Exposure to cocaine is usually demonstrated by urine toxicological analysis; however, this analysis is not routinely performed in young stroke victims. We present a case of a young female who developed a progressive and fatal stroke. Twenty months post-mortem, toxicological analysis in a forensic exhumation autopsy showed cocaine in hair samples. Moreover, targeted artery sampling for histological examination was performed. The results of histological examination appeared to be helpful in demonstrating the putative anatomic site of vascular damage responsible for the stroke.

Case report

A 26-year-old female with an unremarkable medical history developed a persistent headache and heaviness of her right arm. The patient arrived at the Emergency Department of a local hospital about 24 hours later. Clinical examination revealed minor lower facial weakness and slight drift of the right upper arm when extended. Neurological examination was otherwise normal. Head CT scan demonstrated a focal hypodensity of the head of the caudate nuc-

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leus, probably related to recent ischaemic parenchymal damage (Fig. 1A, arrow). Chest x-ray and electrocardiogram were normal. Routine laboratory tests disclosed mild leukocytosis. No drug screening was performed.

The patient was admitted to hospital and 16 hours later her neurological condition deteriorated rapidly. She became aphasic and presented severe motor deficit of the right upper limb and slight motor deficit of the right lower limb. Anti-oedema (glycerol), anti-inflammatory (dexamethasone), and anti-coagulant (enoxaparin) treatments were rapidly done. Furosemide was administered because of a slight increase in blood pressure. A new CT scan showed a large shaded area of hypodensity in the left frontal lobe with involvement of the adjacent basal ganglia region (Fig. 1B); both the rapid evolution of the lesion and the morphology of the involved cerebral areas suggested a diagnosis of recent ischaemic infarct in the vascular territory of the left carotid artery. Colour Doppler examination revealed a complete obstruction at the beginning of the left internal carotid artery. After a few hours, the patient lapsed into a coma and was transferred to the intensive care unit. She died shortly thereafter. On the death certificate, the cause of death was noted as acute cerebral infarction due to obstruction of the initial segment of the left internal carotid artery. No autopsy was done and the body was put in a sealed zinc coffin and buried.

Twenty months later, a forensic post-mortem examination was requested by the deceased's family because of suspicion of medical malpractice. At the autopsy, the body was in a state of mummification. Cerebral matter was reduced in consistency and quite homogeneous. Blood vessels, on the contrary, were relatively well preserved because of embalmment. When main thoracic and neck arteries were bilaterally isolated from the aorta to the skull, i.e. up the beginning of the petrous segment of the internal carotid artery, thinning and reduced consistency of the first tract of the left internal carotid artery were noted. Furthermore, the artery wall was ravelled and very fragile compared with other vessels. Thrombosis was not evident in the lumen of any artery segments examined. No signs of haemorrhage were visible.

At microscopic examination, brain samples presented poor morphology, with no significant differences between samples taken from the damaged area (left basal ganglia and left frontal lobe) and samples taken from other brain portions. The common tract of the left carotid artery and the distal portion of the left internal carotid artery (cervical portion) showed relatively good histological details (Fig. 1C). Only a few post-mortem transformation spots were observed in the common tract of the artery wall. The first portion of the internal carotid artery presented different histological features (Fig. 1D). The wall structure was irregular, histological details were reduced and numerous post-mortem transformation spots were observed in the wall. Dissection, aneurysmatic dilatation and inflammation were excluded.

In the toxicological analysis, the hair samples spiked with internal deuterated standards were analyzed by liquid-liquid extraction procedures with further analysis by gas-chromatography/mass spectrometry (GC/MS) [3,4,11]. A 2-cm section from the root of the hair, corresponding to a 2-month ante-mortem period, was submitted for toxicological hair analysis on the drug profile for opiates, cocaine and cannabinoids. The analysis showed a positive result for cocaine (20 ng/mg) and traces of benzoylecgonine; no other drugs of abuse were detected.

Based on the forensic investigation, the cause of death was considered to be acute cerebral infarction after thrombosis of the first segment of the internal left carotid artery, conceivably related to cocaine use, although cocaine was only detected in hair samples. The presence of cocaine in hair samples only demonstrated previous drug abuse, giving no information about recent cocaine intake.

Discussion

Cocaine use has been associated with sudden deaths in young adults caused, for example, by cerebral strokes, with a peak in the early thirties [7,8]. In the case presented here, a possible connection between cocaine use and the stroke was presumed but could not be verified because only hair samples were available in the autopsy. However, the patient's medical data contained no cardiovascular malformations, arterial hypertension, atherosclerosis, blood coagulation disorders, alterations of lipid metabolism or other diseases usually associated with ischaemic strokes in the young along with drug abuse.

In general, without a medicolegal autopsy, the underlying cause of death has been estimated to be erroneously defined in 28-35% of deaths [1,10]. Fin-

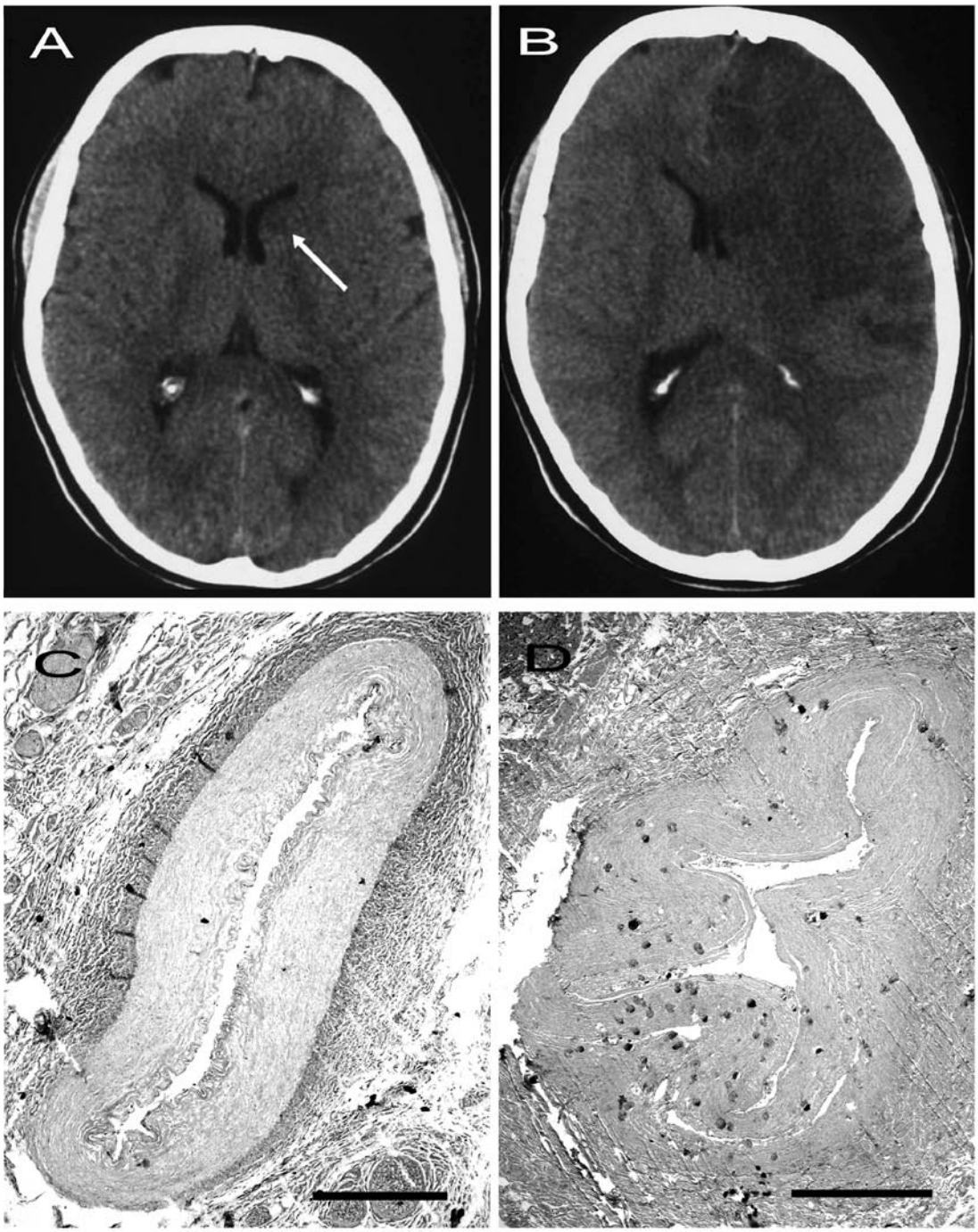

Fig. 1A-D. A. Axial CT image obtained at the level of the basal ganglia one day after onset of symptoms. A focal hypodensity of the head of the caudate nucleus (arrow) is an early sign of an ischaemic infarct in the left internal carotid artery. **B.** Axial CT image obtained at the same level two days after onset of symptoms. The hypodense area is clearly enlarged, diffusely involving the left basal ganglia region and the frontal lobe. Signs of "mass effect" are also evident (left frontal ventricle and cerebral falx are compressed). **C.** Microphotograph of common tract of the left carotid artery with only a few post-mortem transformation spots. **D.** Microphotograph of first portion of the internal left carotid artery with several post-mortem transformation spots, a disrupted wall and collapsed lumen. C and D: haematoxylin-eosin staining; black bar indicates 1 mm

dings of a delayed forensic investigation are often insufficient for conclusive determination of cause and manner of death, especially due to post-mortem transformations. We believe that in the case presented here drug screening in the hospital would have been reasonable considering the patient's young age and her symptoms.

Cocaine is relatively unstable in postmortem biological samples [5,12] because of hydrolytic processes due to variability in pH and temperature. However, cocaine has been detected even in samples of ancient cadavers [2,12]. Also in this case we were able to demonstrate previous cocaine use in a subject deceased 20 months before.

The histological examination of the artery samples obtained in the exhumation autopsy turned out to be of great utility. Different putrefaction features in various segments of the carotid artery were observed. These differences, i.e. the degree of post-mortem changes observed, might be attributable to the state of health of the arterial wall at the moment of death and might therefore be causally related to the stroke suffered by the patient shortly before death. So, by post-mortem histological examination we were able to verify the carotid artery damage seen radiologically ante-mortem. This finding can be significant for cases in which clinical information and ante-mortem findings are unavailable.

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