

CASE REPORT

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Symmetrical necrosis of the solitary tract nuclei as a contributory cause of death

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Abstract A 64-year-old man died in spite of surgery 4 days after attempting suicide. He first tried to hang himself with a rope and when the hanging did not succeed, he cut his throat with a knife. The autopsy showed four sutured cervical wounds with laryngeal wounds but without associated important vascular injury. The neuropathological study revealed two watershed-type haemorrhagic infarcts, involving the left occipital lobe and the left cerebellum. It also showed a symmetrical necrosis of solitary tract nuclei in the medullary tegmentum. Such a lesion is likely to result from sudden acute transient circulatory failure and might have played a role in the secondary autonomous cardiac and respiratory dysfunctions following a non-lethal trauma.

Keywords Solitary tract nucleus · Acute circulatory failure · Forensic neuropathology · Suicide · Hanging

Introduction

Symmetrical necrosis of the brain stem nuclei has been described as a consequence of severe transitory cerebral hypoxia mainly in neonates [1, 2, 3] and more recently in young adults who experienced an episode of acute ischaemia due to transitory acute heart failure [4]. Some of these brain stem nuclei are more vulnerable to ischaemia, such as the solitary tract nuclei. Rossi highlighted the role of bulbo-spinal pathology in neurocardiac sudden death and the possibility of significant pathological findings in the solitary tract nuclei [5]. We report one suicide autopsy case with selective bilateral lesions of the solitary tract

nuclei, which may have played a causal role in delayed death following a usually non-lethal trauma.

Case report

A 64-year-old man died in spite of surgery 4 days after attempting suicide. He first tried to hang himself with a rope and as the hanging did not succeed, he cut his throat with a knife.

Autopsy findings

An autopsy was performed and showed four recent cervical wounds measuring up to 17 cm in length. All were sutured and no ligature mark was present. The dissection revealed bruising of cervical muscles and two deep laryngeal wounds of the thyroid cartilage without significant associated vascular injury. One of the laryngeal wounds was sutured, the lungs were oedematous and the lower left lobe was full with blood. No pulmonary embolism was found. The other organs including the heart showed no significant lesions.

Toxicological tests only showed traces of benzodiazepines in the urine.

Neuropathology findings

The neuropathology examination revealed two haemorrhagic infarcts, one in the cerebral cortex involving the left occipital lobe and the other in the left cerebellum. Both infarctions were located in watershed regions. The cerebellar infarct was associated with a mild swelling of the cerebellum. Gross examination of the medulla revealed bilateral brown discoloration of the anterior aspect of the fourth ventricle particularly at the level of the solitary tract nuclei, bilaterally (Fig. 1A). There was a severe cerebral atherosclerosis in the arteries of the circle of Willis, particularly in the basilar artery. The calibre of the right vertebral artery was much bigger than that of the left artery. Atherosclerosis was also severe in the neck vessels.

The microscopical study confirmed the haemorrhagic infarcts. In addition, the dorsal portion of the solitary tract nuclei showed a symmetrical bilaterally eosinophilic appearance surrounded by a rim of oedema and vascular dilatation (Fig. 1B). The neurons in the nucleus were shrunken, with eosinophilic cytoplasm and picnotic nuclei suggestive of ischaemic changes (Fig. 1C). This was confirmed by an *in situ* end labelling technique which positively stained the nuclei of some of the neurons and neighbouring glial cells in the same area. These changes were aspects which were

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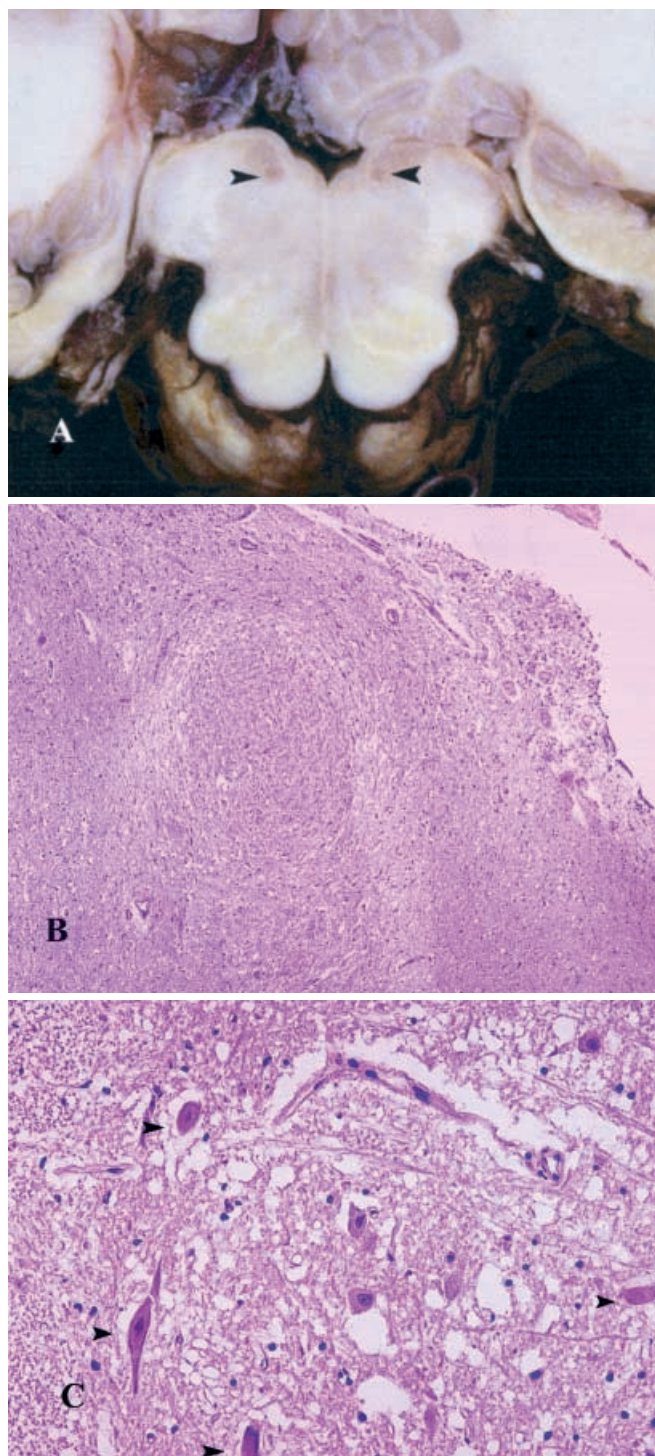


Fig. 1 A–C **A** Horizontal section of the medulla 2 mm under the pontomedullary junction shows brown discoloration of the dorsal part of the medulla particularly at the level of the solitary tract nuclei (*arrows*). **B** Section of the medulla at the level of the left solitary tract nucleus shows central ischemia necrosis surrounded by a rim of oedema (hematoxylin and eosin $\times 40$). **C** Right solitary tract nucleus. Presence of ischemic neurons with shrunken eosinophilic cytoplasm and picnotic nuclei (*arrows*) very different from preserved neurons in the centre of the figure (hematoxylin and eosin $\times 250$)

suggestive of ischaemic coagulation necrosis. The astroglial response was limited to slight nuclear swelling and occasional cytoplasmic hypertrophy. The other brain stem nuclei were normal.

Discussion

The haemorrhagic infarct in our case occurred in at least two steps. Firstly, the suicide attempt by hanging led to ischaemic watershed-type lesions by hypoxia, mechanical obstruction of arterial blood flow in the neck with additional occlusion of venous drainage and low regional cerebral blood flow was probably worsened because of cerebral atherosclerosis [6]. Secondly, the revascularisation of these lesions could explain the haemorrhagic appearance of the infarcts. These infarcts could not explain the death in our case because of their limited size, their location and the absence of brain herniation. On the other hand the cervical wounds were not lethal because of successful neck surgery and of the absence of significant vascular traumatic injury.

Symmetrical necrosis of the brainstem has been described as a consequence of severe transitory cerebral hypoxia or hypotension after an episode of acute heart failure [4]. These brainstem lesions could be directly related to the sudden acute transient circulatory failure and not to hypoxia [7, 8]. In the present case reported, the sudden decrease of the cardiac output and the fall in systemic blood pressure because of blood loss, caused an important diminution of the cerebral blood flow. The medullary tegmentum is a region that is considered resistant to ischaemic insults in adults. However, the subependymal portion of the bulbar tegmentum, mainly the solitary tract nuclei, could be particularly vulnerable to ischaemia due to its intense metabolic activity [9]. C-fos immunoreactivity in the neurons in the solitary tract nucleus has been showed under noxious stimuli, particularly asphyxia [10]. The site of the lesions at the level of the solitary tract nuclei can be explained by vascularisation of the medullary tegmentum. The solitary tract nucleus is localised at the watershed zone between the terminal branches of three medullary arterial sources: the paramedian, the short and long circumferential arteries [11]. Ischaemic lesions are observed when a critical decrease of the vertebro-basilar blood flow occurs. In our case, the morphological evolution of these lesions to gliosis was prevented due to the short interval of survival.

The nucleus of the solitary tract is presumed to act presynaptically to regulate respiratory reflexes [12]. It also plays an important role in the regulation of cardiovascular functions [13]. In the present case, ischaemic lesions of the solitary tract nucleus might have played a role in the secondary autonomous cardiac and respiratory dysfunctions despite successful surgery and resuscitation attempts. Thus, these secondary dysfunctions could have contributed to the delayed death of the patient. Consequently such lesions should be searched in autopsy cases with a history of delayed death after ischaemic hypoxia or acute heart failure.

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