The Death of a Young Adult due to Wallenberg Syndrome: A Case Report

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Abstract
Wallenberg syndrome which is also known as lateral medullary syndrome (LMS) and posterior inferior cerebellar artery syndrome ((PICA syndrome) is detected relatively rarely among young adults. A 42-year-old apparently healthy male presented with headache, vomiting and vertigo. He was diagnosed to have severe hypertension and type-2 diabetes mellitus. During his first admission his first non-contrast computed tomography (NCCT) scan of the brain had confirmed a cerebellar infarction. With clinical findings, the patient was treated as a possible case of LMS. With the repeat NCCT on the third day, he was diagnosed to have progressive cerebellar infarction and a medullary infarction. In the following day the patient was discharged with reserved dates for vertebral artery duplex and ultrasound scan of abdomen (USS). On the seventh day of the illness he had collapsed and died. Subsequent autopsy revealed a left-sided cerebellar and a brain stem infarction along with generalized cerebral oedema. Important findings deduced by forensic pathologists should be conveyed to the clinicians in order to broaden the treatment options and to prevent premature deaths.

Keywords: Cerebellar infarction, Lateral Medullary Syndrome (LMS), neuroimaging, Posterior Inferior Cerebellar Artery Syndrome (PICA syndrome), Wallenberg syndrome

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Introduction

The clinical importance of cerebellar infarctions is considerable because of the life threatening post-infarction brain stem compression by a post-infarction oedema. The triad of Horner's syndrome, ipsilateral limb ataxia, and contralateral limb numbness reliably indicated the involvement of vascular territory of Posterior Inferior Cerebellar Artery (PICA) resulting in Wallenberg syndrome/LMS. A secondary oedema, herniation, obstructive hydrocephalus, a secondary infarction or a propagation of a thrombus are also to be considered in this young patient especially because of the progressive infarction confirmed with a CT scan at the time of discharge. A magnetic resonance imaging (MRI) of brain would have perhaps saved his life.[1,2] The option of "treatability" of a given death is an important concept to broaden the management options.[3]

Case report

A 42-year-old male, who was in apparent good health presented himself to hospital with a sudden onset of headache, vertigo and vomiting. On the second day of admission, he had experienced a left hemi-facial sensory loss, a right upper and lower limb sensory loss with dysphagia, dysarthria, and left sided cerebellar signs. In his medical history he was diagnosed for the first time to have diabetes mellitus and hypertension. The first Non Contrast Computed (NCCT) Tomography, brain of the patient showed a left cerebellar infarction and with the second NCCT brain it had progressed to the lateral medulla as well (Fig. 1 A & B). The 2D echo had revealed mild concentric hypertrophy of the left ventricle and had excluded possible cardiac thromboembolism. The dysphagia, deviation of the uvula to the right, left-sided cerebellar signs and left-sided Horner's syndrome, horizontal nystagmus also been elicited. By the fourth day, the patient had been discharged along with a nasogastric (NG) tube because of his dysphagia with the plan of being subjected to vertebral artery duplex and an ultrasound scan abdomen to exclude any renal pathology causing secondary hypertension and with a plan of physiotherapy. On the day three after being discharged (almost 7 days after the initial symptoms) in the morning he had indicated that he wanted to defecate and had mentioned that he felt unwell and had suddenly collapsed and died. Upon the inquest, a postmortem examination was performed. No (add internal or external) features of trauma were evident. He died before the due date of the vertebral artery duplex scan.

The brain was oedematous(weight 1400g and the dura was tensed) with no midline shift and there was a marked swelling over the left cerebellar hemisphere with macroscopical area of infarction measuring 3.5 x 4 cm in size mostly involving the inferior surface and also the superior surface. There was no gray and white matter demarcations and the area showed necrotic brain matter (Fig. 4). No brain stem haemorrhages were noted but left -sided tonsillar herniation was noticed (Fig. 3 A & B). The circle of Willis appeared anatomically normal with patchy atherosclerosis mainly in the fourth part of the left vertebral artery, bilateral PICA, proximal basilar artery, left posterior cerebral artery, and in an anterior cerebral artery a with longitudinal dissection (no cross sections were studied). The left PICA showed a haemorrhagic area on the atheromatous plaque (Fig. 1).The heart was 320 grams in weight with no ischaemic changes. Microscopically the cerebellar infarctions with neutrophil infiltrations were seen with marked oedema (Fig. 5 A, B, C & D). There were no autopsy findings to suggest deep vein thrombosis or pulmonary thromboembolism.

The cause of death was determined as cerebral oedema with herniation due to the left cerebellar infarction with PICA syndrome with the probable underlying cause of severe atherosclerotic lesions of the circle of Willis due to uncontrolled hypertension and diabetes mellitus.

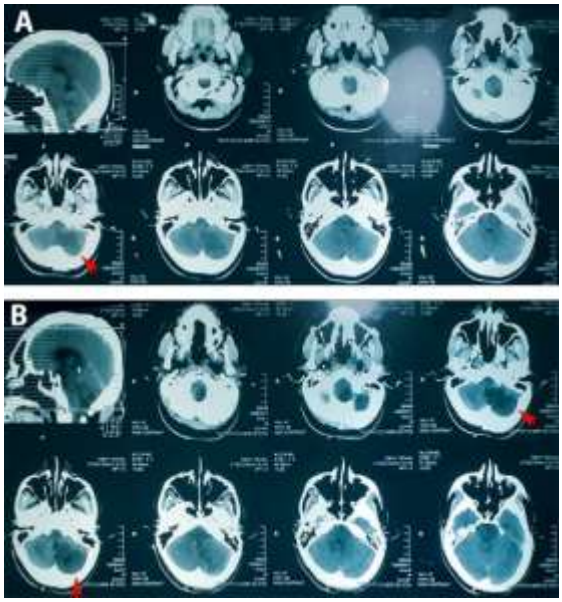

Figure 1. (A) The NCCT brain performed on the first day of admission. (B) The NCCT brain showing progressive infarctions in the day 3(indicated with arrows).

Figure 2. The photograph of the base of the brain with the oedematous and enlarged left cerebellar hemisphere (red arrow head) and the thickened arterial walls of the circle of Willis (marked with yellow arrow heads). Abnormal bilateral indentations of the basal temporal lobes caused by the upward pressure exerted by the space-occupying lesion of the posterior compartment against the falx-cerebri (white arrow heads).

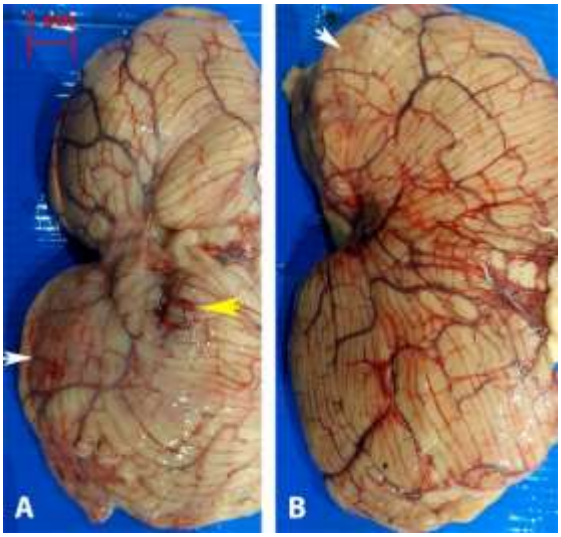

Figure 3. (A) A photograph of the infarcted left tonsils and oedematous and congested left inferior cerebellar hemisphere (arrow head). (B) The superior surface of the cerebellum with extensive left- sided necrosis (arrow head).