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Case Report

An autopsy case of internal jugular vein thrombophlebitis involving sepsis following blunt neck injury

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Abstract

We report an unusual case of delayed death due to sepsis following closed blunt injury to the neck. The victim was a 71-year-old male with a clinical history of hypertension, diabetes and gout. He was found dead about three weeks after being assaulted. He had not consulted a hospital after the assault. Forensic autopsy demonstrated a large liquefied subcutaneous hematoma on the right side of the neck, peri- and thrombophlebitis of the right internal jugular vein. Otherwise, there was no evidence of trauma. Histological examination showed dermal vesicles in the skin covering the hematoma, accompanied by marked inflammatory cell infiltration phagocytosing gram-positive streptococci, subcutaneous edema, panphlebitis with partially organized thrombi and bacterial colonies, pulmonary edema and multiple pulmonary microthrombi involving bacterial aggregates. Postmortem serum C-reactive protein and neopterin levels were markedly elevated. These findings suggest sepsis as the cause of death, induced by infected internal jugular vein thrombophlebitis following blunt neck injury involving impaired skin barrier.

Keywords: Blunt injury; Neck; Thrombophlebitis; Sepsis; Neopterin

1. Introduction

Blunt injury to organs in the neck is uncommon,[1,2] due to the anatomic structures of the mandible protecting from these organs major impact trauma,[3] as well as the mobility and compressibility of the neck itself.[4,5] Previous reports have shown that blunt injury to the neck may incidentally cause various complications including syringomyelia, laryngeal fracture, tracheal rupture and carotid artery occlusion.[6,2,7,8] However, secondary infection through the intact skin affecting the cervical vessels appears very unusual. Although vascular inflammation in the neck is infrequent, internal jugular vein thrombophlebitis associated with acute pharyngitis, tonsillitis and parotitis may be accompanied by fatal sepsis (Lemierre's syndrome).[9–14] These complications are closely related to the complicated anatomic structure of the neck organs.

In the present report, we describe an unusual case of delayed death due to internal jugular vein thrombophlebitis involving sepsis following closed blunt neck injury.

2. Case reports

Case history: The victim was a 71-year-old male, who lived alone. The subject had a clinical history of hypertension, diabetes (188 mg/dL), and gout (uric acid 7.7 mg/dL). In October, he told his friend that he had been assaulted by an unknown robber and sustained a blow to the neck. When the friend visited him 14 days later, the subject was lying down at home, but was still alive. Thereafter, he was found dead on his bed on the 20th day. He had not consulted a hospital.

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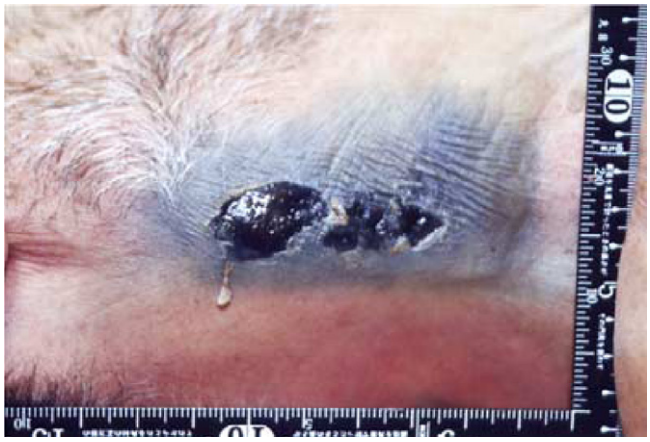

Fig. 1. Right lateral views of the neck, showing dark reddish discoloration and erosions due to vesicle formation.

Autopsy findings: The body was slightly emaciated; 170 cm in height, weighing 51.7 kg. Dark purplish livor was moderately observed on the back, and postmortem rigidity was weak (3–4 days postmortem). The palpebral conjunctivae were pale, and a few petechiae were noted in the right eye. On the right lateral neck region, dark reddish discoloration (5×10 cm) with partial epidermal erosion due to vesicles was observed (Fig. 1). Otherwise, there was no evidence of trauma.

On internal examination, a large liquefied subcutaneous hematoma (15×10 cm) was found in the right neck triangle behind the sternocleidomastoid muscle, covering the right internal jugular vein. The partially organized inner wall of the hematoma adhered to the internal jugular vein, and there was perivascular inflammation, partially organized thrombi in the vascular lumen (Fig. 2). The other neck organs including the right carotid artery were intact. Internal viscera were generally edematous, and petechial hemorrhages were scattered. The heart (460 g) was hypertrophic with enlarged ventricles. There was no evident pathology in the coronary arteries. The lung (left, 445 g; right, 625 g) showed advanced edema. The brain (1375 g) was edematous without any other pathology.

Histological observations: There were dermal vesicles containing gram-positive streptococci in the skin of the neck covering the hematoma, accompanied by marked inflammatory cell infiltration and subcutaneous edema (Fig. 3a). The right internal jugular vein showed partial disruption, dissection and necrosis of the tunica media with bleeding and inflammatory cell infiltration (Fig. 3b), partially organized mixed thrombi involving numerous bacterial aggregates, in which gram-positive streptococci phagocytosed by macrophages were detected. On Azan staining, collagen fiber proliferation in the vascular wall and intravenous thrombi were noted (Fig. 4). The lungs showed edema and multiple microthrombi involving bacterial aggregates (Fig. 5). The pancreas did not show evident fatty infiltration or fibrosis. In the kidney, there was no pathology except for mild thickening of the walls of small arteries. The adrenal glands showed inflammatory cell infiltration.

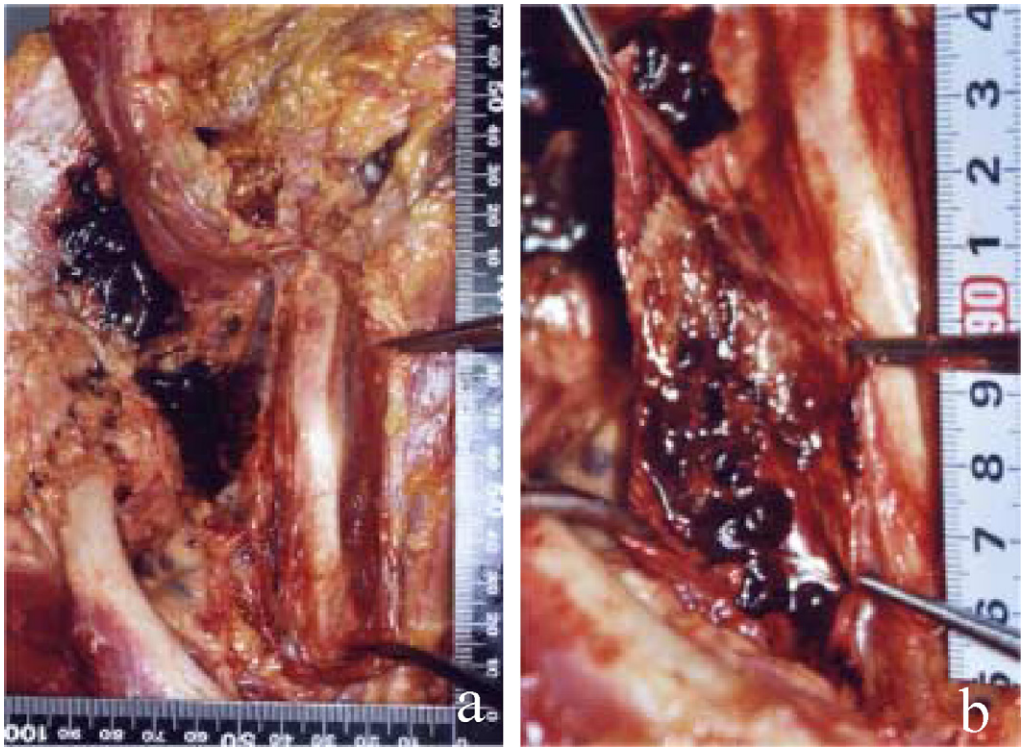

Fig. 2. (a) Internal view of the neck, showing a liquefied hematoma behind the right sternocleidomastoid muscle. (b) Periphlebitis of the right internal jugular vein and partially organized thrombi.