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HISTORICAL NOTE

Wallenberg's syndrome

In his time, Adolf Wallenberg (1862–1949) was renowned for his careful history taking and neurological examination, and for his insistence on clinicopathological confirmation. With Edinger and Gordon Holmes, he described the avian brain. He was responsible for relating the olfactory system to recognition and taste for food; he documented the anatomy of the trigeminal lemniscus in 1900; and provided a clinical masterpiece in his depiction of lateral medullary infarction, remembered as Wallenberg's syndrome.[1]

The earliest known description of lateral medullary infarction was given in 1810 not by Wallenberg, but by Gaspard Vieusseux of Geneva,[2] at the Medical and Chirurgical Society of London:

> "Vertigo, unilateral facial numbness, loss of pain and temperature appreciation in the opposite limbs, dysphasia[sic] and hoarseness, minor tongue involvement, hiccups (cured by taking up the habit of a morning cigarette) and a drooped eyelid."

However, Wallenberg's case report[3] in 1895 amplified the clinical signs with accurate localisation of the lesion, which he later proved at postmortem.

Wallenberg published four papers on the syndrome. The 1895 paper provided the clinical findings in his first patient. Based on the anatomical work of Duret, Wallenberg thought that the lesion was in the lateral medulla supplied by the posterior inferior cerebellar artery (PICA). The second paper (1901) described the postmortem medullary infarction and a stenosis at the origin of the PICA with total occlusion 2 cm distally.[4] The third paper reported another single case[5] and by 1922, he had reported his 15th patient with clinicopathological correlations.[5]

The first patient was:

> "A 38-year-old man, with poor vision caused by a pre-existing ocular condition (cataract on the left side, corneal scarring and anterior synechia on the right side) suffered an attack of vertigo without loss of consciousness. At the same time he developed pain and hyperaesthesia on the left side of the face and body, hypoaesthesiae of the right half of the face, and loss of pain and temperature sensitivity in the right extremities and the right half of the torso, with retention of the sense of touch. There was paralysis of swallowing; impaired sensation on the mucosa of the mouth, throat and palate; disturbed motility of the soft palate (on the first day bilateral, later left-sided); total paralysis of the left recurrent laryngeal nerve, and paresis of the left hypoglossal muscle, with no disturbance in the innervation of the facial muscles. He also had ataxia of the left extremities without impairment of gross strength, and he fell to the left side. The pulse became slower...During the ensuing days the sensitivity of the right half of the face returned to normal. The hyperaesthesia of the left half of the body disappeared, and that of the left trigeminal region changed to anaesthesia predominantly for pain and temperature (less for proprioceptive and electrocutaneous sensations), with suppression of the corneal and conjunctival reflexes. The pulse quickened again, but the other disturbances remained. On the eighth day an herpetic eruption appeared on some of the analgesic areas: the left face (including nasal mucosa; the sensitivity of the mouth and throat had returned), right shoulder, and right inguinal region."[6]

There followed a detailed account of persisting clinical features 2 to 3 months after the attack:

> "In the following weeks, the difficulty swallowing, the falling to the left, and the ataxia gradually disappeared. The other phenomena. . . remained unchanged." A Horner's syndrome was probably missed because the patient had a cataract in one eye and corneal scar in the other.

The arterial lesions of the LMS are principally atherothrombotic vertebral and basilar arteries supplying the PICA, with smaller numbers of dissections, and embolism.[7] Sacco *et al* collected data from all the large reported series. MRI abnormalities[8 9] are evident in more than 90% of patients and vary in size. The prognosis for the majority is good, with a large measure of functional independence.

Wallenberg painted a distinctive picture, which has not been bettered after 100 years. Max Nonne wrote about him: "As a human being, Wallenberg was characterised by a rare modesty and he was unusually warm-hearted and helpful".

> Victor Von Weizsäcker noted:
> " . . .such mastery is only conferred on him who works with endless patience and renouncement, with a proud conscience . . ."

Wallenberg,[10] the son of the physician Samuel Wallenberg, was born on 10 November 1862 in Stargard (near Danzig, then in Western-Prussia). His grandfather was a Rabbi. Samuel died when Adolf was six. As a boy, Adolph learned to play the violin. With his brother Georg a cellist, who became a mathematician, and Theodor a pianist, who became an ophthalmologist, he formed a trio. Wallenberg studied medicine, under Erb in Heidelberg, and in Leipzig, under Strümpell and Weigert. He wrote a thesis on poliomyelitis. He returned to Danzig, as a physician at the city hospital. When the horse from his carriage bolted, in 1891, he suffered from a skull base fracture causing diplopia and anosmia; he later believed it caused his personality to become more compulsive. He refused offers of several chairs, preferring to stay in his homeland. In 1907, he was appointed chief physician to the city hospital. During the first world war, he was advisor to the 17th army. He received the Erb medal in 1929, for his "merits in the field of anatomy, physiology, and pathology of the nervous system". A prolific researcher and writer, he published 47 papers before retiring. In 1938, he had to end his career, under intolerable pressure from the Nazis. Only at the relentless insistence of his wife did they flee through Holland to Oxford, where he worked with LeGros Clark. In 1943, he emigrated to the USA, 50 miles from Chicago.

Wallenberg died from ischaemic heart disease in 1949.

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