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ABNORMAL MORPHOLOGY OF THE SUPERFICIAL TEMPORAL ARTERIES IN THE NOBEL LAUREATE THEODOR MOMMSEN (1817–1903): GIANT CELL ARTERITIS (HORTON'S DISEASE) OR ARTERIOSCLEROSIS? A PALAEOPATHOLOGICAL REASSESSMENT

ABSTRACT: Christian Matthias Theodor Mommsen (1817–1903), brilliant German historian of ancient Roman history and Nobel Prize Laureate in 1902, had excellent health in his youth and maturity but developed serious health problems in his elderly years that greatly limited his work and social activities. Prominent tortuous temporal arteries can be clearly appreciated in Mommsen's portraits and photographs. Additionally, he had recurrent small strokes becoming blind in his final years. He finally died of a stroke in 1903. Autopsy of his brain did not include a reference to the superficial temporal arteries, was inconclusive regarding Mommsen's underlying neurological disease. Giant cell arteritis (GCA, Horton's disease), first reported in 1934, affects elderly people and can contribute to unilateral or bilateral blindness and brain strokes fitting well Mommsen's symptoms. Unluckily the lack of temporal biopsy findings leaves the differential diagnosis of Mommsen's disease, GCA versus brain arteriosclerosis open to debate.

KEY WORDS: Atherosclerosis – Brain autopsy – History of medicine – Horton's disease – Germany – Palaeopathology – Palaeoneurology

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INTRODUCTION AND AIM

Christian Matthias Theodor Mommsen (1817–1903), (*Figure 1A*) the son of a Lutheran minister, was one of the most accomplished German classicists (The Nobel Prize 2023). He became famous for his life-long commitment to research and worked until the very last moments of his existence (Farnell 1934). In 1902, he was awarded the Nobel Prize for Literature for his outstanding contribution to the field, defeating giants of world literature such as the Russian Leo Tolstoy (1828–1910). The committee's reasons for giving him the award was because he was "the greatest living master of the art of historical writing, with special reference to his monumental work, *A History of Rome*." (*Römische Geschichte*, The Nobel Prize 2023). His research embraced different aspects of Ancient Roman history and civilisation. Notably he edited the first volumes of the *Corpus Inscriptionum Latinarum* ("Body of Latin Inscriptions") which continues to this day, and, together with jurist Paul Krüger (1840–1926), he edited an influential modern edition of the Justinianic *Corpus Iuris Civilis* ("Body of Civil Law") (Getzler 2020). Besides his scientific interests and work, Mommsen was a liberal and heavily involved in the politics of his day. He lived throughout the phases of German unification, being remembered as a staunch opponent of its champion Otto von Bismarck (1815–1898). The feud between the two Prussians reached an apogee when *der Eiserne Kanzler* sued the historian for slander, an allegation from which Mommsen would later be acquitted (Stunkel 2012).

While Mommsen's life, political activity, and, above all, academic output has been intensely investigated over the years, not much has been revealed about his diseases and the ultimate cause of his demise. This paper, combining data from contemporaneous historical documents and pictorial representations, including photographs, attempts to shed light on his cause of death and the intrinsic difficulties in reaching a definitive conclusion.

MATERIALS AND METHODS

In order to assess Mommsen's pathology, biographies and newspapers mentioning his life and events in order to collect data of pathological relevance were perused. In addition, a series of photographs and images of the renowned historian at various stages of his life in order to assess his evolving cutaneous morphology was examined. The data were interpreted following the palaeopathographical approach, a sub-branch of classical palaeopathology that focuses on palaeosemiology from indirect sources of evidence built on the foundations laid out by the founders of the Paleopathology Association, the Cockburns, in the 1970s (Galassi 2022, Rühli *et al.* 2016, Bianucci *et al.* 2022a, Bianucci *et al.* 2022b). Intrinsic limitations as to the possibility of an aetiological framing of Mommsen's cause of death were taken into account (Galassi, Gelsi 2015, Rühli *et al.* 2016).

RESULTS

A – Clinical presentation from the literary sources

Mommsen enjoyed a long life without any major illnesses. He was uninjured from a fire that completely destroyed his library in Berlin on the 12th of July 1880 (Rebenich 2002). For many years he was a heavy wine drinker. From 1894, he had several episodes of "melancholy", whether linked to a depressive condition secondary to alcoholism, burnout, or underlying auto-immune pathology, it recurred in a severe form in the summer of 1901 (Rebenich 2002). Nevertheless, on his 85th birthday (11th November 1902), he was still described as a vivid, healthy and active man (Rebenich 2002).

The year 1903 marked a decline in his health. His wife Marie died of a stroke in January 1903. A month later (21st February 1903), he was struck by a horse-tram (*Das interessante Blatt* 1902). Even though he was not significantly injured, his health worsened. In autumn 1903 he became blind in one eye (the side is not reported) and, gradually, lost the sight in his other eye (Rebenich 2002). A photograph dated to 1903 (*Figure 1D*) shows haemorrhage of his right sclera.

Although he experienced "several small stroke attacks" (Hansemann 1907), he was intellectually completely active until the very final days of his life (Neue Freie Presse 1903) [9]. On the 30th of October 1903, he had another stroke and died in Berlin the following day (1st November 1903) aged 86 (Rebenich 2002).

B – Mommsen's autopsy findings

Mommsen corpse underwent autopsy on the 3rd of November 1903 (Rebenich 2002); the report stated:

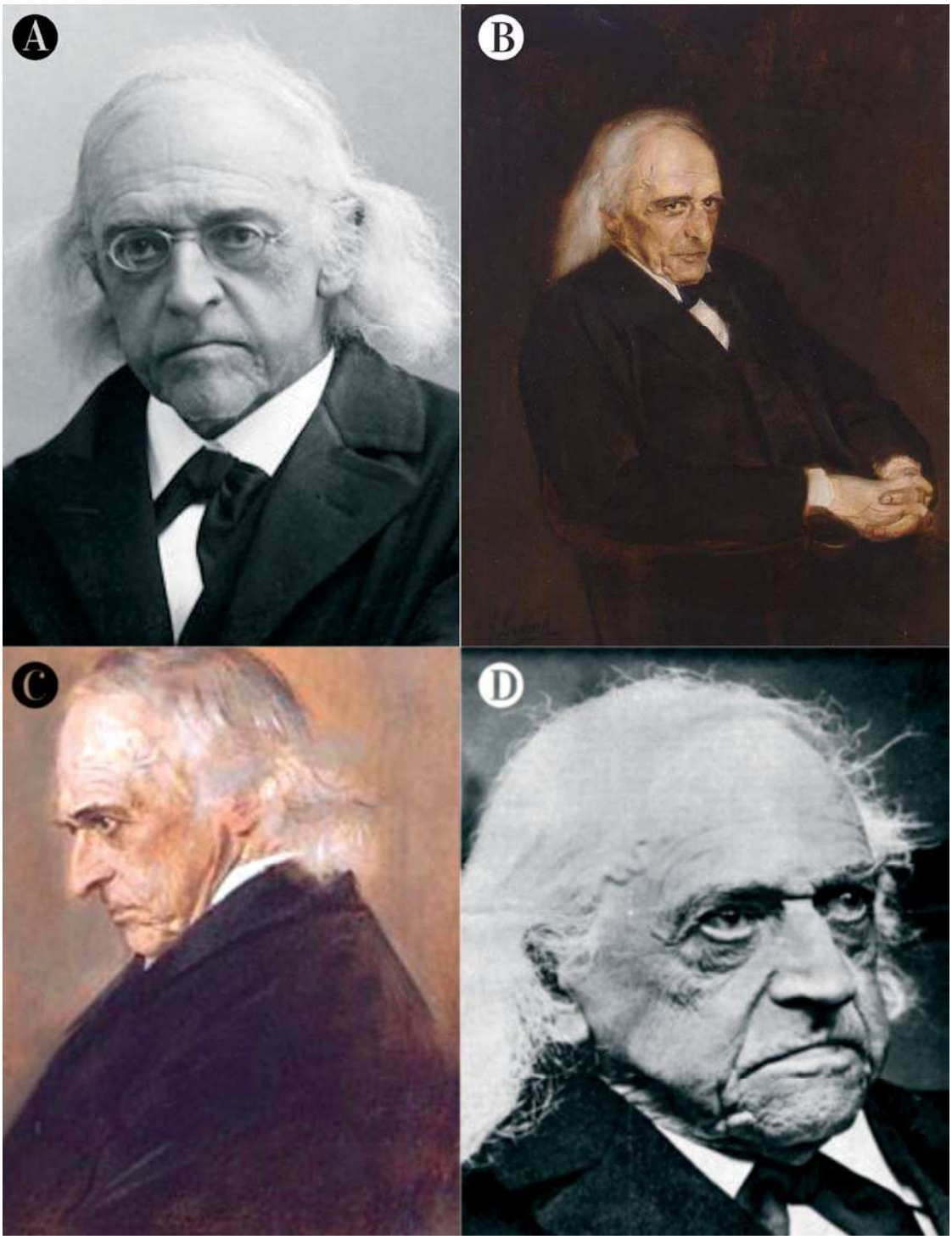

FIGURE 1: A – Theodor Mommsen's picture from the Nobel Foundation archive. Reproduced with permission. B – Mommsen by Franz von Lenbach (1836–1904) in 1897. Source: dpa/Archiv ANZEIGE). Reproduced with permission. C – Mommsen by Franz von Lenbach (1836–1904) in 1903; Source: dpa/Archiv ANZEIGE). Reproduced with permission. D – Mommsen in 1903 displays haemorrhage of the right sclera and a mismatch of the sight axes between both eyes as seen by the light reflection – indicating that one eye might not be functional. Reproduced with permission.

... the brain of Theodor Mommsen, whose autopsy has been thankfully wished by his family, first reveals obviously an age-associated atrophy which considerably influenced not only the weight, but also the form and width of individual gyri of the brain. Secondly, the right hemisphere contains numerous superficial liquefactive foci of recent date, most obviously resulting from the last few days before death. Since it was, for the proper assessment of the changes, necessary to remove the pia mater before fixation, these changes caused considerable damage to the surface. ... therefore, the right hemisphere was evaluable only for major changes... The largest circumference of the head above the skin was 56 cm, the skull circumference 54 ½ cm, the skin is therefore quite thin. The skull bone is considerably thickened, in its posterior region 6 mm, heavy and sclerosed. The external surface does not show atrophy. The dura mater is firmly attached to the inner surface, there are no exostoses. The venous blood vessels are intact. On removal of the brain, a lot of clear fluid discharges. The blood vessels at the base are heavily sclerosed, patchy thickened and narrowed. The right half of the cerebellum is almost completely liquified. The liquefaction is a white one, and does not show any transition into a yellow one. It doubtlessly occurred only shortly before death. Additionally, there exist numerous superficial liquefaction foci in the right hemisphere, only few in the left hemisphere. The largest is located in the upper gyri close to the midline and measuring c. 3 cm in diameter. This closely attaches the precentral gyrus, but does not affect it. Additionally, in the right posterior lobe, there exists an older liquefaction focus of 1 ½ cm with yellow colour. All further liquefaction foci are small, of white colour, and only visible after removal of the pia mater. The latter one is strongly oedematous and clouded above the frontal lobes. The gyri are remarkably small, the sulci are deep and wide. The cerebellum and pons weigh 220 g, the cerebrum 1205 g, so in total 1425 g, which corresponds to the mean weight of an adult male. However, if one takes the atrophy into account, it is feasible that the brain previously had a much higher weight. It remains, however, undetermined whether final oedema contributed to the final brain weight.

(Hansemann 1907 – translation by coauthor A. G. Nerlich) (*Figure 2A, B*).

C – Morphology of his temporal arteries

In his later years, his temporal arteries became visibly swollen and tortuous (*Figure 1B, C*), a condition observable in different photographs and portraits dated between 1897 and 1903.

DISCUSSION

Sudden loss of vision can be exaplained by several aetiologies, mainly neoplasia, inflammation, ischaemia and infection to the optic nerve, optic tract chiasm, lateral geniculate, parietal and temporal optic radiations, and the occipital region (Graves, Galetta 2012, Bagheri, Mehta 2015). However, association with a prominent tortuous temporal artery suggests either a giant cell arteritis (GCA) or an arterial, age-related disease. Ocular ischaemic disorders secondary to arteriosclerosis are assumed to be secondary to embolism or haemodynamic disorders from atherosclerotic plaques or marked stenosis/thrombosis in the regional arteries (Hayreh 1999). Arteriosclerosis typically affects middle-aged and elderly people and can cause gradual or sudden loss of vision (Fishbein, Fishbein 2009).

GCA, also named temporal arteritis or Horton's arteritis, is a vasculitis characterised by inflammation of the medium-and large-sized arteries involving one or more branches of the carotid artery, especially the temporal artery. GCA occurs more frequently in individuals older than 50 years old, and its frequency increases with age. GCA patients present with headaches, malaise, fever, fatigue, anorexia, weight loss and anaemia. Scalp pain and claudication of the jaw may be also present. GCA may cause an ischaemic optic neuropathy leading to severe visual problems including sudden blindness; treatment is therefore an emergency. Claudication of the extremities, strokes, myocardial and visceral organ infarctions have also been noted in GCA patients. Almost half of GCA patients have polymyalgia rheumatica manifesting as limb girdle muscle weakness. Patients with GCA may have tender, thickened, nodular temporal arteries on physical examination. Temporal artery biopsy is essential for diagnosing GCA, a vasculitis that responds quickly and successfully to glucocorticoid therapy (Sneller *et al.* 2005, Hunder 2006). GCA may affect the central retinal artery leading to unilateral or bilateral blindness.

From a historical perspective, GCA was probably already present in antiquity (possibly in ancient Egypt in the 14th century BC) and should not be regarded as a contemporary rheumatological disease, although all subsequent evidence is based on literary and artistic sources instead of biological ones. The most famous and likely historical presentations of the diseases include

Canon van der Paele (15th century AD) and the French surgeon Ambroise Paré (16th century AD). They are worthy of mention since they involve a match of literary and artistic evidence, plus, in the latter's case, the description of a surgical intervention, the severing of his temporal artery, performed by himself in order to relieve the pain caused by the associated migraine (Meige 1924, Galassi, Rühli 2016, Galassi, Galassi 2016).

The eye symptoms, along with swollen temporal arteries clearly visible in Mommsen's photographs and portraits, plus a history of strokes, suggest that Mommsen may have had GCA. It should be noted that there was no mention of having *polymyalgia rheumatica* in the available sources. In addition, GCA is known to be more frequently unilateral rather than bilateral (Coors, Simon 2002).

However, more common aetiologies should be considered. The brain autopsy findings are more compatible with chronic ischaemic vascular disease with prominent atherosclerosis. The whole-body autopsy report is lacking; the Rudolf-Virchow-Krankenhaus in Berlin ones are from 1906 onwards. The autopsy of the brain reveals very significant atrophy of the right cerebral hemisphere with numerous "fresh" infarction areas on both sides, most probably due to a recent stroke secondary to very significant arteriosclerosis of the intracranial arteries showing an uneven thickening pattern of the vessels. This would be much more in favour of a "simple" (and possibly systemic) arteriosclerosis and not to a localised arterial inflammation as in GCA and the likely aetiology of the loss of vision. However, arteriosclerosis might not explain Mommsen's bilateral blindness. Sudden loss of vision implying retinal artery occlusion is an unusual complication of atherosclerotic cerebrovascular disease (Hayreh 1999). Takayasu's arteritis, an inflammation

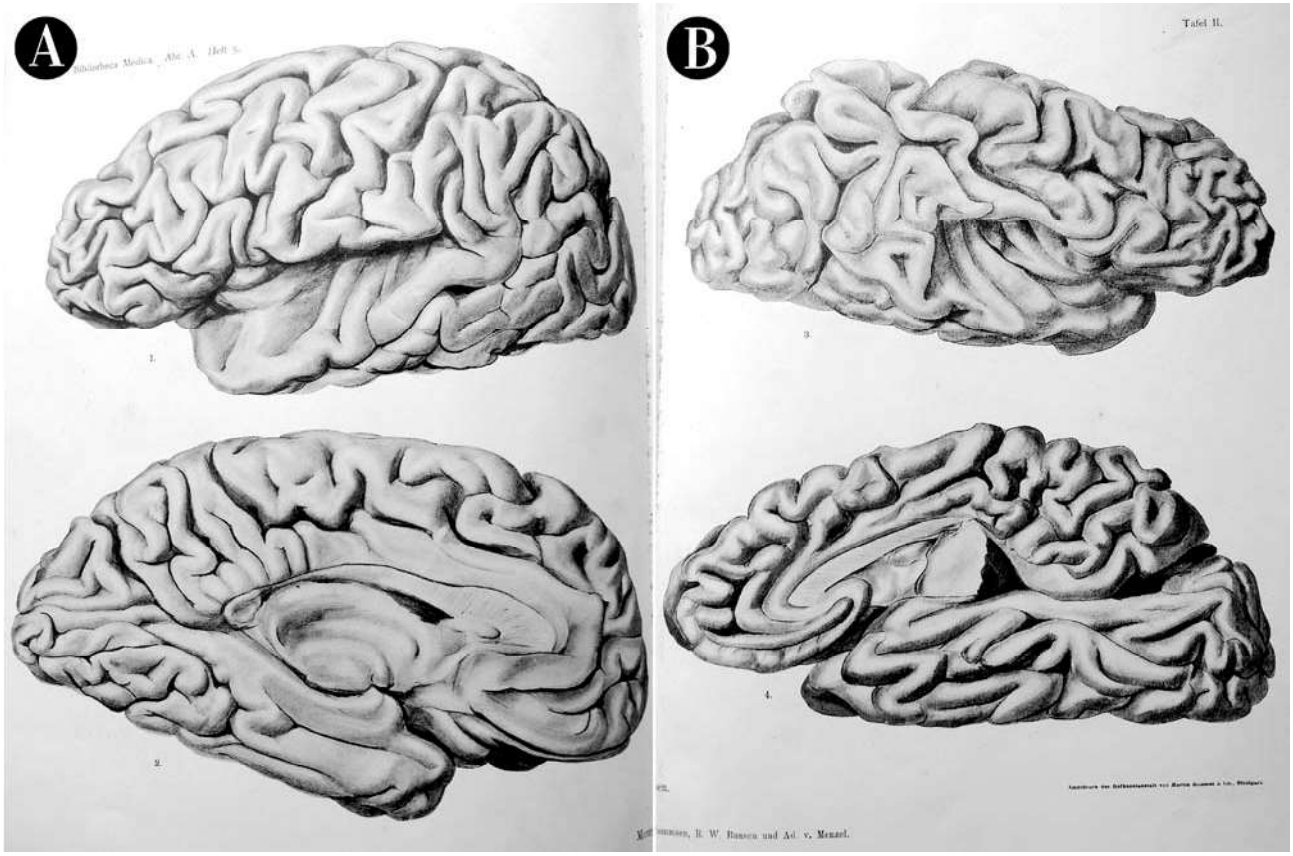

FIGURE 2: A, B – Plate with two views of the left and the right hemisphere of Mommsen's brain. Global age-associated atrophy of the brain lobes, more prominent on the right hemisphere, with widening of the sulci can be observed. Reproduced with permission.

of medium-and large-sized arteries, especially of the aortic arch and its branches, might also produce visual changes if the common carotid is affected, although blindness is unusual. This arteritis is most prevalent in young Asian women (Kerr *et al.* 1994).

Moreover, GCA autopsy findings could also have been missed. Indeed, GCA is a vascular inflammatory entity, discovered in 1934 by Horton, Magath and Brown, therefore, it was unknown to German pathologists in 1903, when Mommsen's autopsy was performed (Hunder 2006, Boes 2007).

CONCLUSIONS

Although the exact nature of Mommsen's *causa mortis* and underlying pathology remains unclear, additional and previously unnoticed data have been furnished in this paper that link some of his known symptomatology to degenerative morphological changes of his superficial temporal arteries, which had previously gone unnoticed. In the absence of a palaeopathological study on his bodily remains at the Kreuzberg cemetery in Berlin (should the soft vascular tissues have preserved at all or significantly) giant cell arteritis and/or arteriosclerosis are the most likely conditions that could have affected the famous historian and led to his demise, a finding which is consistent with previously reported definitive diagnostic uncertainty in retrospective palaeopathological studies of these two diseases (Galassi *et al.* 2017). Notwithstanding, the natural history of these diseases fit best with the documentary and pictorial evidence currently available.

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