

Bones of the Boy-King: The Mysterious Ailment of the Pharaoh Siptah

Matthew D. Turner^{1*} and Michael Lawson²

¹U.S. Army, Emergency Medicine Program, Penn State Hershey S. Milton Medical Center, 500 University Drive, Hershey, PA, 17033, USA

²U.S. Army, Dermatology Program, Brooke Army Medical Center, 3551 Roger Brooke Dr, Fort Sam Houston, TX 78234, USA

Abstract

Ever since the mummy of Pharaoh Siptah was examined in the early twentieth century, the remains of Siptah, a young pharaoh of ancient Egypt, have been an object of curiosity. The etiology of the pharaoh's unusually malformed left leg has been theorized to have been the result of congenital clubfoot, poliomyelitis, or cerebral palsy. We conclude that Siptah's deformity was likely due to congenital clubfoot.

Keywords: Congenital clubfoot; Foot; Historical medicine; Pharaoh Siptah; Ancient Egypt

Introduction

The Nineteenth Dynasty was a dark time for ancient Egypt. 30 years after the dynasty had finally slid into obscurity and oblivion, Ramesses II wrote of that period: "The land of Egypt was cast adrift, every man a law unto himself ..." [1]. Despite Ramesses' bold declaration that the dynasty before his own had consisted of only "empty years" [1], a discovery was made in the early twentieth century that cast an interesting new light on this previously obscure period in Egyptian history. In 1905, while examining the embalmed mummy of Siptah, penultimate pharaoh of Egypt's Nineteenth Dynasty, archaeologists discovered that the pharaoh possessed an unusual deformity: that of a malformed left foot, with a corresponding shortening of his left lower extremity [2]. Since then, there has been a significant debate in the literature regarding the true etiology of Siptah's unusual presentation, with three major theories emerging over the decades [3].

Description

The puppet pharaoh

In the later years of ancient Egypt's declining Nineteenth Dynasty, a sickly teenager rose to the throne of Egypt. By some accounts, Siptah was not even the son of the previous Pharaoh, Seti II, and was actually the offspring of one of Seti's greatest political rivals [4]. The weak child-king won the throne largely due to the machinations of the chancellor named Bay, who controlled Siptah and the kingdom through a regency council for the next several years [4]. During his lifetime, Siptah does not appear to have had much political power or influence. His claim to the throne was "slender" and later generations ignored him as a usurper [5]. Chancellor Bay presented himself as the kingmaker for the young Pharaoh, proudly proclaiming that the king was little more than his puppet [4] and that he had "fixed [Siptah's] eye on him alone" [5]. In ancient Egypt-where "king-making was theoretically a job for the gods", such arrogance was regarded as close to blasphemy. This was not helped by the massive tomb of "royal proportions" that the chancellor had built for himself in the Valley of the Kings [6].

It was perhaps this arrogance that sealed Bay's fate. 5 years into the regency, Queen Tawosret, widow to the previous pharaoh Seti II, moved to silence the troublesome chancellor for good. Bay's "fall from grace was swift and absolute"; he lost all power at court, was executed for treason, and his name was "officially proscribed, so denying him eternal life" [4]. Within a year of Bay's death, Siptah mysteriously died as well. The exact means of his death remain unclear, but researchers have suggested that "something sinister lay behind the young man's

death" [6]. Wilkinson notes that the death was certainly "convenient" for Bay's old political enemies [4]. Tawosret seized the crown, and swiftly launched a "full-scale persecution of the puppet king's memory" [4]. Even the images of the king in his own tomb were replaced by images of her late husband, Seti II [6]. Even death was not the end of the king's troubles. Later kings of the Twentieth Dynasty refused to regard him as a legitimate ruler, and what little legacy that remained from his 6-year reign suffered [5]. To add insult to injury, some 150 years after his death, Siptah's tomb was plundered by a later regime seeking treasure to maintain its status [4]. When Siptah's mummy was examined in 1905, it was found that plunderers had broken off his right hand and that it had had to be clumsily splinted into place when the priests rewrapped the body [2]. Ultimately, Siptah would likely have been little more than a footnote in history, a puppet-king ruled by an unscrupulous regency council and quickly disposed of, had it not been for the discovery of his mummy in 1898 and its subsequent autopsy in 1905 [2]. During his investigation of Siptah's remains, the archaeologist Grafton Elliot Smith found that the pharaoh-a "young man... with a thick crop of short, reddish brown, curly hair"-had a significant deformity in his left foot, "distorted in the manner known as talipes equino-varus" [2]. Later studies confirmed that Siptah's mummy has a severely deformed left foot, as well as significant shortening in his left leg [7]. While this deformity was originally believed to have been a case of congenital clubfoot [8], a number of theories to explain it have developed over the years. In this paper we evaluate the various theories and suggest our conclusion-that the young pharaoh suffered from an undiagnosed case of spastic hemiplegic cerebral palsy (Figures 1 and 2).

Talipes Equinovarus

Smith's initial 1905 autopsy concluded that Siptah likely suffered from Talipes equinovarus [2], also known as clubfoot. A frequent congenital deformity that is twice as common in males, the precise etiology of Congenital Talipes Equinovarus (CTEV) remains unknown

***Corresponding author:** Matthew D. Turner, U.S. Army, Emergency Medicine Program, Penn State Hershey S. Milton Medical Center, 500 University Drive, Hershey, PA, 17033, USA

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Figure 1: Siptah's mummified legs. Note the deformity of the left foot. Obtained from the University of Chicago Libraries. This picture is in the Public Domain [9].

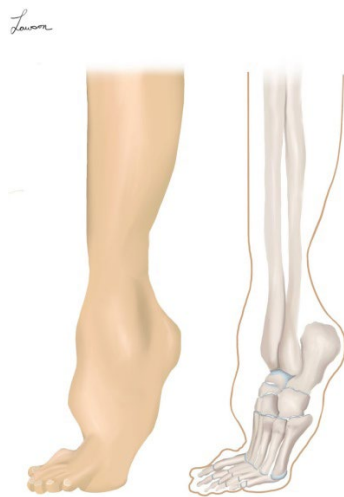


Figure 2: A depiction of Siptah's foot, drawn by Dr. Michael Lawson, one of the authors of this paper.

[9,10]. CTEV may manifest with “malposition of the tarsal bones, atrophy of the calf muscle, and shortness of the foot” [10], but may also present with leg length discrepancy [11]. In idiopathic CTEV, the upper limbs of the patient remain unaffected [12]. CTEV appears to have been a known syndrome in ancient Egypt. Several depictions of inward-turned feet “which correspond very closely to talipes equinovarus as seen today” have been found in ancient papyri [8]. The relative frequency of CTEV-estimated at 1-2 per 1000 births [13] likely made it well-known in the greater ancient world. Hippocrates described the syndrome several centuries after Siptah's death [13]. During some periods of ancient Egyptian history, “attendants with club-feet or humps... seem to have been appreciated by the Old and Middle Kingdom elite” in an ambivalent cultural mixture of “respect, veneration, and rejection” [14]. Although Siptah's Nineteenth Dynasty arose hundreds of years after the Old and Middle Kingdoms [6], this demonstrates that CTEV occurred on a regular basis in the land of the Nile, and further supports Siptah's diagnosis of CTEV [15]. In recent years, Siptah's diagnosis of CTEV has come into doubt. A 1973 publication noted that the shortening of

Siptah's left leg, as well as the “atrophy of the soft tissues indicate[s] the presence of a neuro-muscular disease in childhood” [1]. However, leg muscle atrophy in CTEV can develop and worsen with age [16], possibly explaining the soft tissue atrophy noted by the study [1]. However, a 1986 assessment of Siptah's foot concluded that he suffered from a case of “extreme equinus deformity, possibly from poliomyelitis or cerebral palsy rather than congenital clubfoot” [3]. A further difficulty with the CTEV hypothesis is that approximately 49% of CTEV patients present with bilateral CTEV; only 22% have solely the left foot affected, a proportion that is “very similar in all populations” [11]. With this statistic in mind, it is important to assess other, more likely causes of the pharaoh's abnormality.

Poliomyelitis

The same 1973 publication that observed the significant atrophy of the soft tissues in Siptah's left leg also proposed that the pharaoh's abnormality may have been due to a childhood neuromuscular disease, specifically poliomyelitis [2]. This is possible; poliovirus attacks the Central Nervous System (CNS) and spreads throughout the afferent nerve pathway, causing particular destruction to the anterior horn cells of the spinal cord [17]. This may lead to widespread muscular atrophy, leading to flaccid paralysis. However, in the pediatric form, this may cause an asymmetrical paralysis of the limbs [17]. In Siptah's case, this asymmetrical paralysis of his left leg could explain the “left foot compensat[ing] the shorter leg by dislocation of the tarsal and metatarsal bones, tendon and muscles” [18]. However, clear evidence for the presence of poliomyelitis in ancient Egypt is extraordinarily rare and controversial [19]. During either the Eighteenth or Nineteenth Dynasty-nearly contemporary with Siptah-a doorkeeper named Roma was depicted on his funerary stela “with a grossly wasted and shortened leg accompanied by an equinus deformity of the foot” [8]. Although it has been tentatively proposed that Roma may have suffered from poliomyelitis contracted in childhood, the evidence remains unclear [8] it may be that Roma suffered from CTEV [20]. Aside from these cases, and an isolated shortened femur dating approximately 500 years later, there is no other evidence for the existence of poliomyelitis in ancient Egypt [20].

While polio almost certainly existed before the modern era, child paralysis was extremely uncommon. Before the development of modern sanitation, “enteric infections were so common that babies, exposed to human waste and poliovirus early in life, would have been protected by maternal antibodies [from breastfeeding]... in these instances, the invasion of poliovirus into the central nervous system and the resulting paralysis was avoided” [19]. Most infant infections are typically subclinical; when paired with a high infant mortality “paralytic poliomyelitis could have remained undetected for centuries or even millennia” [19]. In ancient Egypt, where approximately 20% of infants did not survive past 1 month of age [21], it likely would have been extremely difficult for paralytic poliomyelitis to develop in the population. It was only with the development of the medicine and infrastructure of recent centuries that delayed the infection of children until later in life, after the completion of weaning, that caused the outbreaks of “hemiplegic child paralysis” that poliomyelitis is now known for [19]. In addition to this, Egyptian infants were typically breastfed for the first three years of life [21] this may have provided protection against early enteric infections of poliovirus, due to the presence of maternal antibodies [19]. Given this, it appears unlikely that Siptah suffered from a childhood form of poliomyelitis. The evidence for the diseases' presence in this period is extremely scarce [20]. Even if it had been present, the lack of modern sanitation, combined with early exposure during the breastfeeding stage, would have made the

modern presentation of childhood hemiplegic paralysis unlikely in such conditions [19].

Spastic hemiplegic cerebral palsy

Cerebral palsy has also been proposed as a possible explanation for Siptah's deformity [3,18,22]. A neuromotor disorder that is "characterized by abnormal tone, posture and movement", cerebral palsy is even more common than CTEV, occurring at approximately 2-3 out of 1000 live births [23]. Of these, 25% of children present with spastic hemiplegia, affecting one side of the body, which is associated with "normal cognitive abilities" and a "high level of functional abilities" [23]. Equinus deformity is the most common musculoskeletal abnormality associated with cerebral palsy, eventually developing into a fixed deformity over time due to muscle shortening and worsening joint abnormalities, with a typical process of "unrelenting progression" [24]. Approximately 75% of patients with cerebral palsy will develop some form of equinus [25]. In cases of spastic hemiplegic cerebral palsy, this may even progress into leg length discrepancy [26], as demonstrated in Siptah's mummy [2]. Further supporting this hypothesis is Siptah's young age—approximately 20—at the time of his death, which has been noted to be the average lifespan for cerebral palsy patients [22]. While Siptah's death was convenient for his successors and may have been the direct result of Tawosret's political machinations [4], his mummy displays no evidence of foul play [2]. With her place on the regency council following chancellor Bay's execution [4], we theorize that there may be no need for Tawosret to have her puppet assassinated—it could be that her rise to the throne was simply taking advantage of Siptah's natural death due to complications from cerebral palsy. While it is impossible to fully determine, we suggest that Siptah's equinus deformity, as well as his associated leg shortening, was likely due to a neuromuscular etiology [20]. Given the lack of evidence for the existence of poliomyelitis in the ancient world [20], and given the relatively high incidence of cerebral palsy and the pharaoh's skeletal resemblance to this disease, we suggest that cerebral palsy, possibly of the spastic hemiplegic variant, is responsible for Siptah's symptoms, as well as his early death.

Conclusion

Of the three main theories produced over the last several decades regarding Pharaoh Siptah's skeletal abnormalities, we suggest that an undiagnosed case of cerebral palsy is the most likely. This disease process is more common than CTEV, is consistent with the young king's anatomy and early death, and appears more likely than poliomyelitis. However, until more evidence comes to light, it is likely a definitive diagnosis of the king will remain unknown.

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