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AN EARLY CASE OF SYPHILIS FROM KABERLA, NORTH ESTONIA

This article presents a case study of congenital syphilis identified from Kaberla cemetery in North Estonia. As far as the author is aware, this is the earliest reported case identified from skeletal evidence in Estonia, and is thus particularly interesting. This paper will present some general background information about the disease and its development, as well as presenting the specific lesions noted on the Kaberla skeleton that led to the diagnosis of syphilis.

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Site archaeology

Skeleton CI was discovered while analysing the skulls from Kaberla cemetery for dental diseases relating to diet. Kaberla is located about 30 km east of Tallinn and archaeological excavations have taken place there over several years: in 1955 by Aita Kustin, 1956 by Silvia Laul, 1958, 1962, 1964 by Jüri Selirand and in 1966 by Mare Aun. The cemetery was used over several centuries, beginning in the late 12th and ending at the end of the 17th century (Selirand & Tõnisson 1984, 162). The cemetery has produced 242 skeletons, but some graves have been destroyed by gravel quarrying, and perhaps as many as 300 skeletons were interred there.

The skeleton in question (CI) appears to belong to the later period of the cemetery as indicated by the presence of a coin made in 1667, located near to the body. The body lay supine with its head pointed to the west and its arms crossed over the waist. Several iron nails were located next to the body and represent remnants of the child's coffin (Selirand (1962) for a detailed description of Kaberla cemetery, and Mark (1962) for a presentation of anthropological data).

Background notes about syphilis

Of the treponomal diseases, none has received more attention or moral judgement than venereal syphilis, due to its relationship to sexual activity. The

disease has had many names historically and has also been known as Mal de Naples, Arboyne pimple, Scottish sibbens, Swedish salfluss, Morbus gallicus and Spanish pox, to name a few (Roberts & Manchester 1995, 153). Venereal syphilis belongs to a group of diseases that are caused by the bacteria *Treponema* sp. The other diseases are yaws, endemic syphilis and pinta. The basic bone changes of the four diseases are virtually identical, except for pinta, which only seems to affect the skin. Yaws, endemic syphilis and pinta can be transmitted through basic bodily contact, while venereal syphilis is a sexually transmitted disease. Also differentiating venereal syphilis is its ability to be passed from the infected mother to her unborn child across the placenta (congenital syphilis).

Before the advent of effective antibiotic treatment, one could note a distinct geographical distribution of the treponomal diseases. Pinta occurred in a Central American position only; yaws was found in equatorial regions; endemic syphilis in arid areas north and south of the yaws territory; while venereal syphilis was more widespread and it seems to surface in crowded urban centres (Roberts & Manchester 1995, 156).

Most scholars accept that syphilis, or some precursor, originated in Africa, and followed man in his travels, arriving in America when man crossed the Bering land bridge. The disease then, for reasons unknown, disappeared in Eurasia, for the most part. Many scholars believe that syphilis was brought back to Europe from America by Columbus. Currently there is only a handful of cases of venereal syphilis in Eurasia dating before Columbus's return, while the Americas have many undeniable cases of pre-Columbian syphilis.

The disease reached near epidemic status around 1500 in Europe, according to documentary records, about a decade after the return of Columbus (Roberts & Manchester 1995, 157). This has led to questions concerning the Columbus theory, since this is insufficient time to unleash a European epidemic. It has been proposed (Hackett 1967) that venereal syphilis in Europe prior to the Columbian expeditions was not identified specifically from several other diseases, including leprosy. Possible support of this idea is noted in the medieval misconception that leprosy was transmitted sexually. However, skeletal evidence from medieval leprosy cemeteries does not support this theory. It seems apparent that for further enlightenment about the origins and spread of venereal syphilis, precise dating of skeletons and accurate diagnosis is necessary for determining the pre- or post Columbian origin of the disease.

Symptoms of venereal syphilis

Primary syphilis. Primary syphilis begins two to six weeks after sexual contact and results in painless red sores, usually on the genitals. Without treatment, the sores heal after four to six weeks, leaving a thin scar.

Secondary syphilis. This stage usually begins two to ten weeks after the initial sores heal. Syphilis bacteria enter the blood and spread through the body causing different symptoms, such as a general skin rash, fever, headache, loss of appetite, weight loss, sore throat, muscle aches, joint pain, swollen lymph nodes. In this stage, syphilis may attack the liver, kidneys, and eyes.

Late syphilis. After the secondary stage passes, some people progress to a 'latent stage' where they have no more symptoms. After up to several years of latency, symptoms develop that affect the eyes, large blood vessels, nerves, and brain. At this stage, the victim develops memory loss, psychological problems, difficulty walking, loss of balance, loss of feeling (especially in the legs), vision problems and heart disease. The most advanced stage is often termed 'paralysis of the insane' due to faltering gait and fits of irrational madness. Death is a welcomed relief at this point and usually results from a ruptured artery.

It is in the late stages where the palaeopathologist can recognise the disease, since it is here that the bones are affected. It should be noted though that this stage is reached in only about 10% of cases, indicating that the prevalence of syphilis could be underestimated by up to 90% by palaeopathologists.

The bone changes are of osteomyelitis, resulting in gross bone destruction called a gumma; this is accompanied by new bone growth that leads to changes in the bone shape. The tibiae are the most affected long bones, but there is usually multiple bone involvement throughout the skeleton. Similar changes arise in yaws and endemic syphilis, but are not as severe and destructive as in venereal syphilis. In venereal syphilis the skull is also frequently affected, leaving a characteristic 'worm-eaten' appearance known as caries sicca. The frontal and parietal bones are the most commonly affected areas, and crater-like lesions are formed that show a mixture of healing and destruction. Changes in the maxilla and nasal area are also common, but not usually as severe as in yaws or endemic syphilis.

As mentioned earlier, venereal syphilis has the ability to pass from infected mother across the placenta to her fetus, leading to what is termed congenital syphilis. The clinical features of congenital syphilis are similar to those of acquired syphilis. Congenital syphilis is rare in the palaeopathological context, mainly since 50% of infected babies die either before or around birth.

Osteology of skeleton CI

Bone preservation was excellent and surface erosion did not hinder identification of surface changes to the bone; however, several bones, particularly the axial skeleton, are absent. Table 1 indicates the presence or absence of skeletal parts.

Table 1. Skeletal representation of skeleton CI

Bone element	Present
Cranium	+
Mandible	+
Left clavicle, right clavicle	–, –
Left scapula, right scapula	–, –
Sternum	–
Ribs	–
Vertebrae	–
Left humerus, right humerus	+, +
Left ulna, right ulna	+, +
Left radius, right radius	+, +
Hand bones	–
Pelvis	–
Left femur, right femur	+, +
Left patella, right patella	–, –
Left tibia, right tibia	+, +
Left fibula, right fibula	–, –
Foot bones	–

Skeleton CI belongs to a child as indicated by unfused epiphyses of the long bones, and a mixture of permanent and deciduous teeth. Dental development indicates an age at death of about 8–9 years. However, long bone lengths give conflicting results, and suggest an average age of about 5.5–6.5 years of age. Since long bone length, however, is to a greater extent under environmental control (i.e. disease and nutrition can affect growth), while dental development is mostly under genetic control, it is most likely that the age attained by the dentition is the accurate reflection of the true age at death. The stunted growth is likely a result of disease and/or malnutrition. The sex of the skeleton cannot be determined since the distinguishing features between males and females do not appear before puberty.

Palaeopathology

Enamel hypoplasia. The presence of grooved lines across the crowns of the anterior teeth indicate the presence of recurrent stresses during childhood. Only the crowns of the anterior teeth of $_{1}C$, C^{1}, and ^{2}I are present, and all three teeth display two hypoplasias per tooth. Their position on the crown indicates that the periods of stress occurred about a year apart between 3.5 and 4.5 years of age.

Cribra orbitalia. Both orbits show pitting, indicating iron deficiency anemia (Fig. 1). Although possibly diet-related, studies have shown that the body makes itself iron-deficient in times of disease stress, since the pathogens require iron to survive. Perhaps, then, the anemia is related to the syphilis.

Fig. 1. Cribra orbitalia on the roof of the right orbit. Photos by Jaanus Heinla.
Joon. 1. *Cribra orbitalia* parema silmakoopa laes. Fotod Jaanus Heinla.

Caries Sicca. As described before, caries sicca presents itself as crater like lesions on the skull vault and are a telling sign of syphilis. Skeleton CI has two such lesions (Fig. 2). The first is located approximately 40 mm superior to the medial half of the left orbit and has a transverse diameter of about 17 mm, a superior-inferior diameter of 11 mm, and a depth of several millimetres (Fig. 3). The lesion is surrounded by pinprick sized holes. The lesion displays signs of healing and has produced a scar like the appearance on the inner part of lesion. The second lesion is not as severe in appearance and is located on the frontal bone, lateral to the left orbit, at the articulation point to the left zygomatic bone (Fig. 4). The lesion has a height of 13 mm and a width of about 9 mm.

Osteomyelitis. Osteomyelitis is brought about by the spread of infection into the medullary cavity of the bones. It results in bone destruction and pus formation, and simultaneous bone repair, leading to an enlarged appearance with cavity formation extending from the interior to the external part of the bone. The tibiae (shinbones) display the largest lesions, and the mixture of gross bone destruction and remodelling have caused the bones to appear grossly misshapen (Fig. 5). There are several abscesses on each bone that are as large as 15 mm in diameter, and new bone formation covers the entire shafts of the bones. The distal 1/3 of the right radius (lateral bone of the forearm) is also affected and the diameter of the bone is ensheathed in a callous-like structure which is pitted with multiple cavities (Fig. 6), one of which extends completely through the bone. Finally both humeri show lesions at their distal ends (Fig. 6) in the form of cavities located on the posterior surface just proximal to the olecranon fossa. The left humerus also displays a cavity on the distal anterior surface and the lesion is surrounded by plaques of new-bone formation.

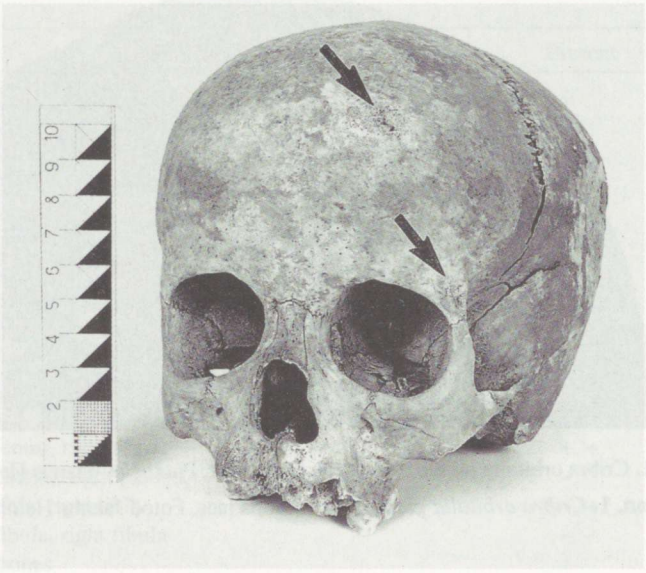

Fig. 2. Cranium showing the position of caries sicca.

Joon. 2. *Caries sicca* asukoht koljul.

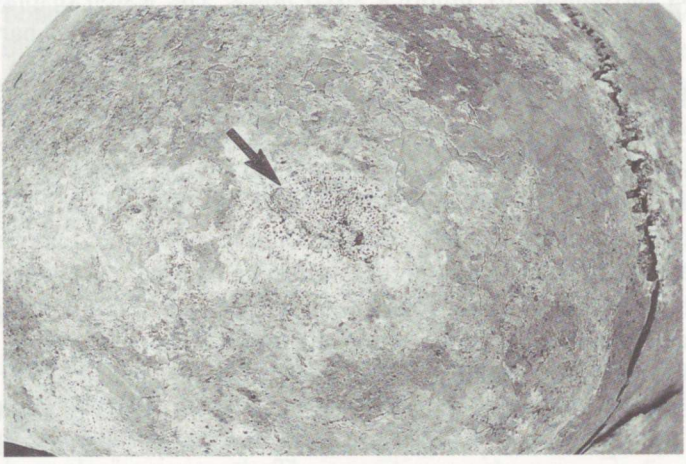

Fig. 3. Caries sicca on frontal bone.

Joon. 3. *Caries sicca* kolju esiosas.

Fig. 4. Caries sicca near the left zygomatic suture.

Joon. 4. *Caries sicca* vasaku silmakoopa vasakus ääres.

Fig. 5. Osteomyelitis of the tibiae.

Joon. 5. Osteomüeliit sääreluul.

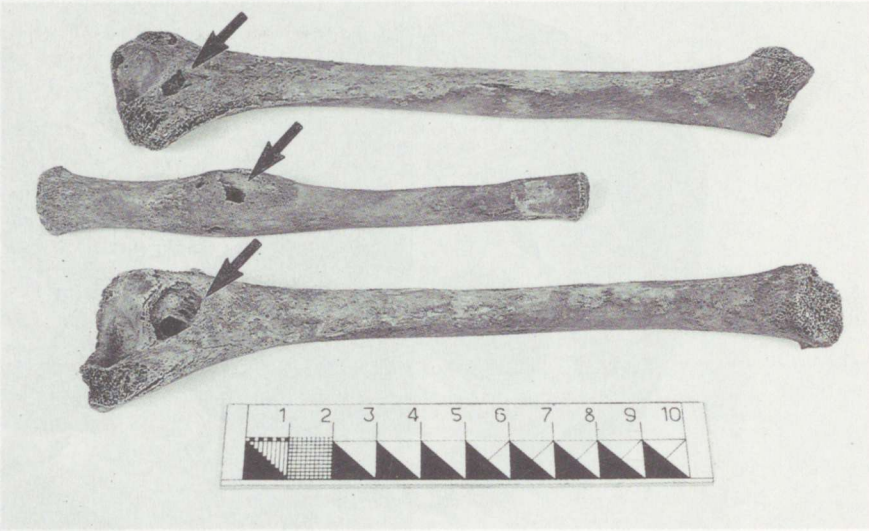

Fig. 6. Osteomyelitis of right humerus, right radius, and left humerus (from top to bottom).
Joon. 6. Parema õlavarreluu, parema kodarluu ja vasaku õlavarreluu osteomüeliit (ülevalt alla).

Other. Additional signs of pathology are present on the maxilla which is covered in a multitude of pinprick size holes, and evidence of bone resorption and remodelling immediately superior to the upper incisors and below the nasal cavity.

Discussion

The combination of gross bone destruction and remodelling of the long bones, caries sicca on the cranium, and bone changes in the maxilla and nasal areas are all cardinal signs of syphilis. The fact that the victim is a child suggests that this is a case of congenital syphilis. Upon identification of the disease, the question arose as to the whereabouts of the child's mother. However, no other skeletons at Kaberla displayed evidence of the disease. As mentioned before, though, only about 10% of syphilis victims progress to the stage where bone changes are obvious.

Written sources tell us that syphilis reached epidemic proportions in Estonia at the end of the 17th century, fitting the same time frame of this identified case. The spread of the disease was undoubtedly facilitated in the past by urbanisation, prostitution, and military and religious pilgrimage.

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VARANE SÜÜFILISE JUHTUM KABERLAST

Resümee

On kirjeldatud kaasasündinud süüfilise juhtumit, mis pärineb Kaberla kalmistust Põhja-Eestis. Antropoloogilise materjali põhjal on see varaseim teadaolev süüfilise juhtum Eestis. Kalmistut kasutati alates 12. sajandi lõpust kuni 17. sajandi lõpuni. Luustik nr. CI asus kalmistu hilisemas osas. Dateerimisel oli abiks hauast leitud 1667. aasta münt.

Paleopatoloogid võivad süüfilise ära tunda üksnes viimases staadiumis, kui see on jõudnud luudeni. Ainult 10% haigusjuhtudest jõuab sellesse staadiumi. Nii võivad paleopatoloogid süüfilise levikut alahinnata kuni 90% ulatuses. Veneerilisel süüfilisel on võime kanduda emalt platsenta kaudu üle lootele, kes haigestub. Paleopatoloogilises kontekstis on kaasasündinud süüfilis harv nähtus, sest 50% nakatunud beebidest sureb kas enne sündi või vahetult pärast seda.

Siin käsitletava luustiku luude säilimisaste on väga hea ja pinna kuluvus ei takistanud luu pinna identifitseerimist. Samas puuduvad mitmed luud, eriti aksiaalne skelett (tab. 1).

Luustik CI kuulub lapsele, nagu võib järeldada pikkade luude poolikust liitumisest ning jääv- ja piimahammaste olemasolust. Hammaste arengu järgi võib oletada, et laps oli surmahetkel 8–9-aastane. Samas osutab pikkade luude pikkus sellele, et laps võis olla u. 5,5–6,5-aastane. Et aga pikad luud on üldjuhul rohkem keskkonna kujundada (haigus ja toitumine võivad kasvu mõjutada) ja hammaste areng enamasti geneetiliselt kontrollitav, siis tõenäoliselt on indiviidi tegelik vanus surmahetkel täpsemini määratletav hammaste kaudu. Vähene kasv on tõenäoliselt haiguse või halva toitumise tulemus. Skeleti sugu ei ole määratav, sest meeste ja naiste vahelised erinevused ei ilmne enne puberteediiga.

Skeleti patoloogia on järgmine.

Hüpoplaasia. Vaotaolised jooned, mis jooksevad üle esihammaste kroonide, viitavad korduvale stressile lapsepõlves. Olemas on ainult $_1C$, C^1 ja ^2I esihamba

kroonid ning neil kõigil on kaks joont hamba kohta. Joonte asukoht hambakroonil näitab, et stressiperioodide vahel oli umbes aasta ja need avaldusid siis, kui laps oli 3,5–4,5 aastat vana.

Cribra orbitalia. Mõlemas silmakoopas on näha mitu nõelatorke suurust auku (joon. 1), mis on omased raua puudulikkusest tekkinud kehvveresusele. Kuigi see võib olla seotud toitumise eripäraga, näitavad uurimused, et organism muudab ennast haigusega seotud stressi ajal rauapuudulikuks, sest patogeenidel on eluks rauda vaja. Ehk on aneemia sel juhul süüfilisega seotud.

Caries sicca. Kraatrisarnased vigastused koljul viitavad süüfilisele. Luustiku CI koljul on kaks seesugust vigastust (joon. 2). Esimene on u. 40 mm vasaku silmakoopa sisemisest poolest kõrgemal. Selle diameeter on ligi 17 mm, superioorne-inferioorne diameeter 11 mm ning sügavus mitu millimeetrit. Vigastuse ümber on nõelatorke suurused augud (joon. 3). Vigastusel on paranemismärke, mille sisepind on armitaoline. Teine vigastus ei ole nii raske ja külgneb vasaku silmakoopaga (joon. 4). See on 13 mm pikk ja u. 9 mm lai.

Osteomüeliit. Luuüdipõletik tuleneb nakkuse levimisest luude medullaarõõnsusse. See põhjustab luu purunemist, mäda teket ja samaaegselt luu paranemist, mis suurendab luud. Seetõttu moodustub õõnsus luu sisepinnalt välispinnani. Sääreluudel on suurimad vigastused, s.t. märgid suuremahulisest luu purunemisest ja taastumisest. Need luud on väga muundunud kujuga (joon. 5). Igal luul on kuni 15 mm diameetriga mädanikud ja uus luu katab terve olemasoleva luu pinna. Nakatunud on ka parema kodarluu distaalne kolmandik, kus luu välispind on krobeline ja pikitud mitmete vigastustega (joon. 6), millest üks ulatub täielikult läbi luu. Mõlemal õlavarreluul on distaalses otsas vigastused (joon. 6). Need vigastused asuvad tagumisel pinnal *olecranon fossae* kõrval. Vasakul õlavarreluul on vigastus samuti distaalsel esipinnal ning seda ümbritseb uus luu.

Muud patoloogia märgid on ülemisel lõualuul, mis on kaetud mitme nõelatorke suuruse auguga. Ülemisel lõualuul on märke luu imendumisest ja taastumisest täpselt ülemiste lõikehammaste kohal ja ninaõõne all.

Luu purunemise ja pikkade luude taastumise kombinatsioon, kraatrisarnased vigastused koljul ning luu moondumine ülemisel lõualuul ja ninaõõne alal on põhilised süüfilise tundemärgid. See, et ohver on laps, viitab kaasasündinud süüfilisele. Pärast haiguse tuvastamist tekkis küsimus, kus asub kalmes lapse ema. Ühelgi teisel Kaberlast leitud skeletil seesuguse haiguse märke aga ei leitud.

Kirjalikud allikad viitavad sellele, et süüfilis jõudis Eestis epideemiliste mõõtmeteni 17. sajandi lõpul. See langeb kokku siin käsitletud juhtumi dateeringuga. Haiguse levikut hoogustasid kindlasti linnastumine, prostitutsioon ning sõjalised ja usulised rännakud.

ÜLEVAATED

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Aliise ja Harri Moora.

2. märtsil 2000 toimus Eesti Teaduste Akadeemias teaduskonverents, mis oli pühendatud akadeemik Harri Moora ja ajalookandidaat Aliise Moora 100. sünniaastapäevale. Avasõnad lausus Eesti TA president akadeemik Jüri Engelbrecht ning seejärel peeti neli ettekannet, mis on siin avaldatud lühendatult.