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Elongated odontoid process in late Holocene skeletal remains from B6 archaeological site, Mendoza, Argentina

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ABSTRACT

The odontoid process of the axis can be affected by congenital or acquired pathologies. While abnormalities such as os odontoideum, agenesis, and fractures are reported in archaeological remains, the abnormality of an elongated length of the odontoid process has not been described in the paleopathological literature. The aim of this paper is to evaluate two individuals with elongated odontoid processes from a skeletal assemblage from the B6 archaeological site (Mendoza, Argentina), and to discuss the possible etiologies of the condition, with particular attention given to the relation to trauma and Crowned Dens Syndrome (CDS), a condition characterized by the ossification of ligaments of the odontoid process of the second cervical vertebra.

1. Introduction

The odontoid process (the dens) is an anatomical feature of the second cervical vertebra (C2), and is the central pillar of the craniovertebral junction. Defects of the odontoid process can be produced by congenital or acquired conditions. Congenital conditions, reported in the clinical and archaeological literature, include os odontoideum, odontoid aplasia and persistent ossiculum terminale (Curate, 2008; Hensinger et al., 1978; Jain et al., 2016). Acquired defects may be produced by trauma or degenerative, infectious or metabolic diseases (Jain et al., 2016). Defects of the odontoid process are seldomly reported in archaeological remains, but a few cases of os odontoideum, in which the dens is separated from the vertebral body (e.g. Curate, 2008; Mann et al., 2013), agenesis (e.g. Barnes, 2012), and fractures (e.g. Weber et al., 2003) appear in the literature. On the contrary, as far as we know, the elongated length of the odontoid process, recorded in clinical research as evidence of trauma or inflammatory diseases, has not been described in the paleopathological literature.

The aim of this paper is to evaluate and report the presence of two cases of elongated odontoid processes found on skeletal remains from a late Holocene multiple burial (site B6) in Mendoza (Argentina).The recognition of this trait in skeletal remains could be helpful for paleopathologists seeking to complete differential diagnoses of the hitherto rarely reported pathological condition in archaeoalogical remains.

2. Material and methods

2.1. Archaeological background

The archaeological locality of Barrancas is located at the eastern lowlands of Maipú Department, Mendoza, Argentina (Fig. 1). The largest multiple burial of Barrancas is the B6 site (Fig. 1), a cemetery encompassing 8 m2 found in 2009 (Novellino et al., 2013). The skeletal assemblage from this site consists of 34 individuals (16 non-adults and 18 adults; 6 females, 11 males and 17 of indeterminate sex). Twenty-nine of individuals, including the two skeletons reported here, were buried in a semicircle (Fig. 2) (Barberena et al., 2017; Novellino et al., 2013). The only cultural artifacts are two projectile points, one lodged in a dorso-ventral direction in the sternum of Skeleton 19, and the other between two lumbar vertebrae of Skeleton 30, both adult males aged > 45 years old. The skeletons from the B6 site are currently housed at the Museum of Natural and Anthropological Sciences "J. Cornelio Moyano", Mendoza, Argentina.

Three radiocarbon dates were obtained from human bone samples from this site, resulting in non-calibrated dates of 2260 ± 80 years BP -LP-2387 (Novellino et al., 2013), 2450 ± 60 years BP- LP-3110 and 2251 ± 49 years BP-AA98707. A foraging subsistence was suggested for human groups living during the middle-late Holocene, based on technological, zooarchaeological, and stable isotopic data (Barberena

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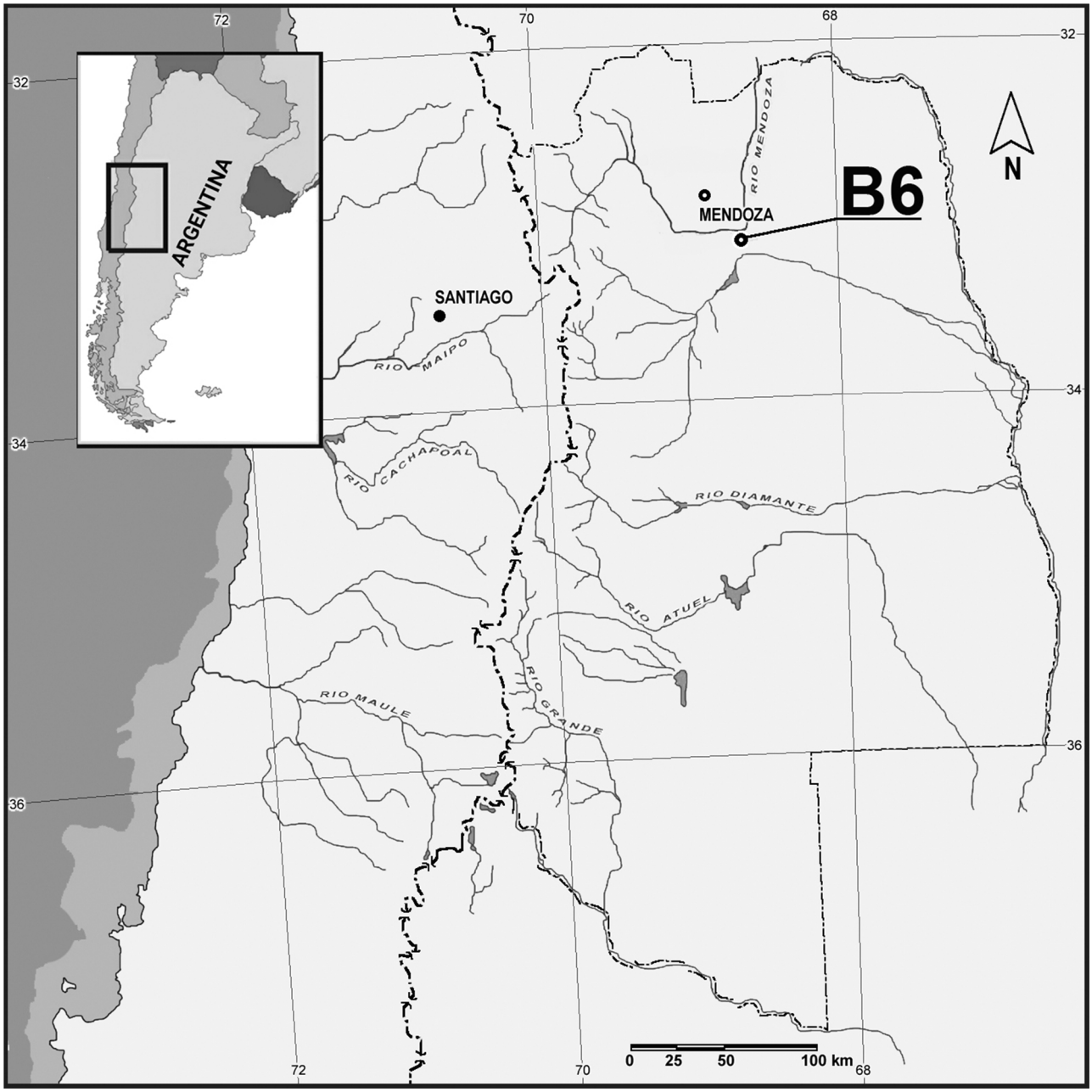

Fig. 1. Location of the B6 archaeological site in Mendoza Province, Argentina.

Fig. 2. Multiple burials found in the B6 archaeological site, Mendoza Province, Argentina.

et al., 2017), with diets predominantly based on animal protein (Gil et al., 2014a; Novellino et al., 2013). Infrequent migration between lowland and highland regions was also proposed from strontium and oxygen stable isotope data (Barberena et al., 2017; Gil et al., 2014b).

2.2. The individuals

Skeletons 2 and 8 (Sk 2 and Sk 8) from the B6 site are well preserved and almost complete. They were determined to be males based on the presence of rugose features of the crania, and morphological features of the os coxa, including narrow sciatic notches, acute subpubic angles, and lack of ventral arcs (Buikstra and Ubelaker, 1994). Based on the morphological changes of the auricular surface (Lovejoy et al., 1985) and the pubic symphysis (Brooks and Suchey, 1990; Todd, 1921a,b), the age-at-death of Sk 2 was determined to be 35–50 years old, while Sk 8 was 50+ years old.

2.3. Methods

All C2 vertebrae of the skeletons recovered from the B6 site were macroscopically examined with the aid of a 10X magnifying glass. The form, texture and location of all lesions observed on the odontoid process were recorded and described. The length and thickness of new bone formation was measured using a digital vernier slide caliper. X-ray images were obtained in order to detect the presence of bone fractures. Other heterotopic ossification, defined as the formation of lamellar bone inside soft-tissue structures (Bossche and Vanderstraeten, 2005), was also recorded.

Since it has been suggested that osteoarthritis (OA) could be associated with diseases in which ligament ossification of the dens is involved (Rosenthal and Ryan, 2016), OA was diagnosed in all available joints when eburnation was present, or when two or more of the following joint changes were observed: 1) marginal osteophytes; 2) new bone formation on joint surface; 3) pitting on the joint; 4) alteration in joint shape (Rogers and Waldron, 1995). The degree of severity was classified as slight, moderate, or severe (Jurmain, 1990; Rojas-Sepúlveda et al., 2008).

3. Results

Elongated odontoid processes were identified in the C2 vertebrae of Sk 2 and Sk 8 from the B6 site. No other individuals displayed these pathological lesions. Therefore, 2 out of 14 (14.3%) adult skeletons with preserved C2 vertebrae display this trait.

Skeleton 2 displays a curved ossification over the antero-superior end of the odontoid process, 5.55 mm long and 1.77 mm thick, with a smooth surface and ridged edge (Fig. 3). No evidence of fracture was detected in this vertebra, neither from macroscopic inspection nor from x-ray images (Fig. 4a and b). No lesions were recorded on the occipital bone, and no other heterotopic ossification was found in this individual. Degenerative joint disease compatible with OA was found only on the vertebrae of this individual. Severe OA affected the C3 inferior endplate, the C4 superior and inferior endplates, and the C5 superior endplate, with marginal osteophytes and pitting on the joint surfaces. Slight OA was also observed on T9-T12 and L2-L5. Moreover, a Schmorl´s node was present on C5 on the superior aspect of the body. Anterior compression of the vertebral body of C5 (anterior height: 16.2 mm; posterior height: 19.9 mm) was noted, as well as on T11 (anterior height: 17.3 mm; posterior height: 23.3 mm) and T12 vertebrae (anterior height: 16.3 mm; posterior height: 26.2 mm).

Skeleton 8 displays curved and smooth new bone formation on the antero-superior aspect of the dens, 4.40 mm in length and 2.95 mm thick, with a prominent superior ridge (Fig. 5). No signs of fracture were observed in x-rays images (Fig. 4c and d), and no lesions were observed on the occipital bone. Moreover, no other heterotopic ossification was found. Moderate OA, with osteophytes and pitting on joint surfaces, was only identified in the superior and inferior lateral articular facets of C3. Slight OA was noted on C6, T8, T11 and L5 vertebrae.

4. Differential diagnosis

Ligament ossification around the odontoid process of C2, similar in morphology to the ones identified in the skeletons from the B6 site, have been recently described in clinical literature associated with metabolic diseases, classified as pseudogout (Sekijima et al., 2010; Soo Lee et al., 2014), and associated with trauma, neoplasm, and infectious or degenerative diseases (Godfrin-Valnet et al., 2013). Metastatic or primary bone cancer may affect the odontoid process, though cervical tumors are more commonly associated with lytic lesions and chord complications than with new bone formation on the odontoid process (Godfrin-Valnet et al., 2013; Jain et al., 2016). Degenerative joint disease is frequently noted on this vertebra, but osteoarthritis rarely affects the odontoid process (Weber et al., 2003). Finally, some infectious diseases, such as TB, could affect the dens (Jain et al., 2016), but neither skeleton from the B6 site displayed evidence of neoplastic lesions or new bone formation compatible with infectious disease on any anatomical element of the body, nor was degenerative changes of atlantoaxial joints observed.

There are numerous examples of localized trauma to C2 contributing to lesions of the odontoid process (e.g. Korres et al., 2017; Weber et al., 2003). In particular, Type1fractures affecting the superior aspect of the odontoid process can lead to defects in this portion of the vertebra (Anderson and D'Alonzo, 1974). Moreover, cervical vertebrae trauma, without fracture, has been documented as related to the ossification of the ligaments around the dens (e.g. Che Mohamed and Abd Aziz, 2009; Sim and Park, 2006), with soft tissue trauma responsible for

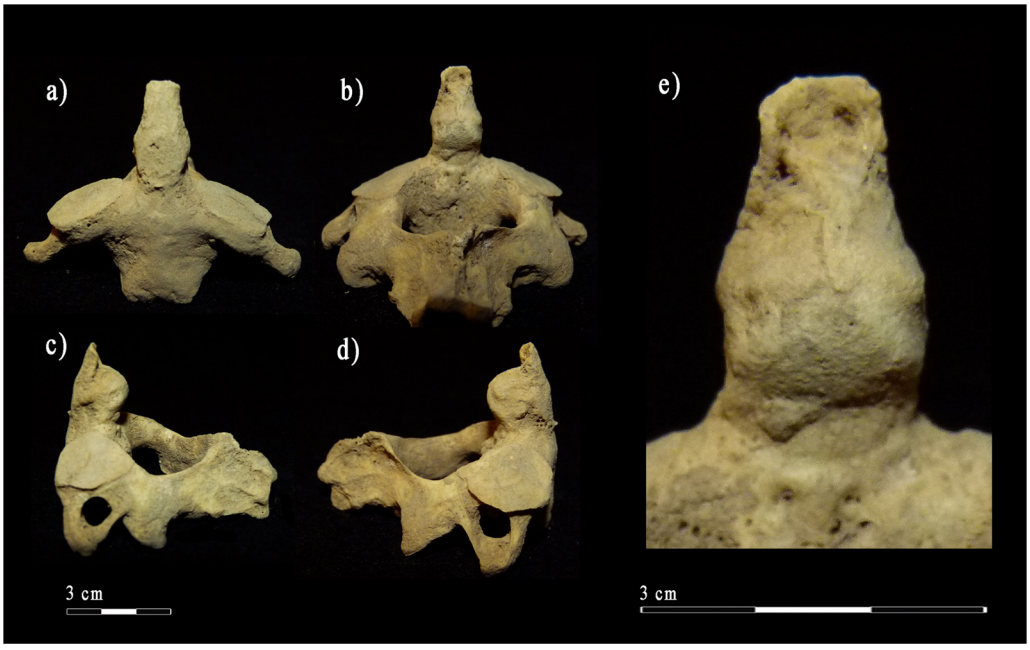

Fig. 3. C2 of adult male (Sk 2) of the B6 site. a) anterior view; b) posterior view; c) left view; d) right view; e) close up of odontoid process in posterior view.

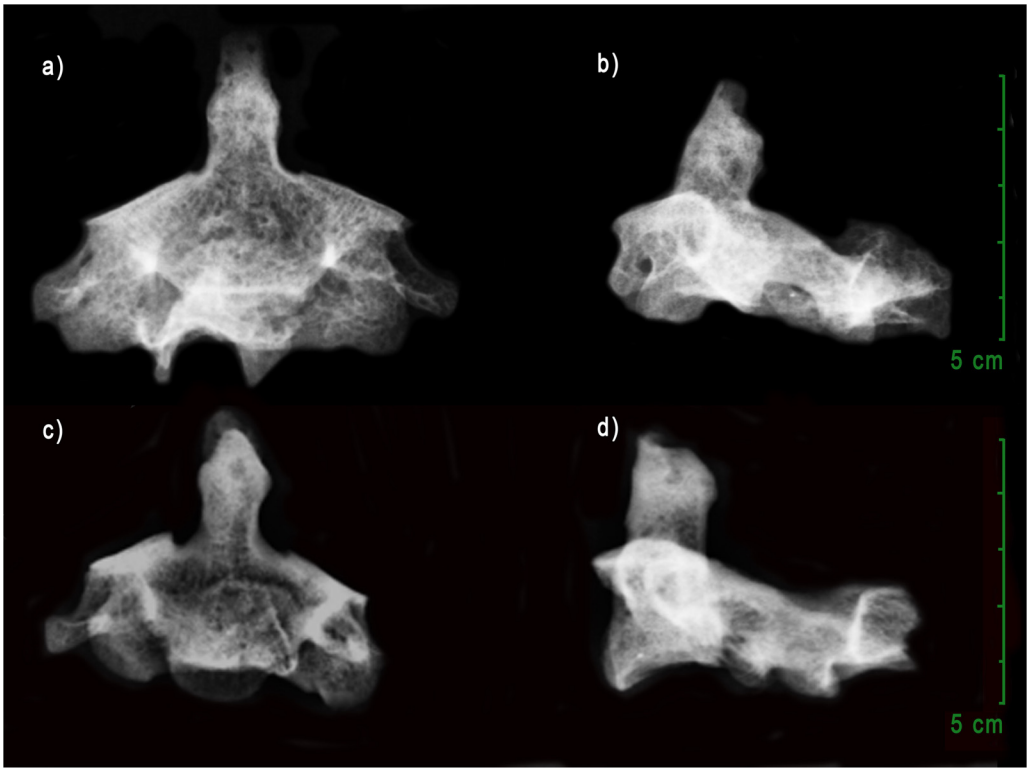

Fig. 4. X-ray images from Sk 2 (a: frontal view; b: lateral view) and Sk 8 (c: frontal view; d: lateral view) from the B6 archaeological site.

Fig. 5. C2 of adult male (Sk 8) from the B6 site. a) posterior view; b) right view.

heterotopic ossification (Bossche and Vanderstraeten, 2005; McCarthy and Sundaram, 2005).

Subsequently, although Sk 2 and Sk 8 from the B6 site did not show signs of fracture to the C2 vertebrae, cervical trauma cannot be rejected as the cause of the apical ligament ossification, since the vertebral body compression, observed in Sk 2, may be related to spinal compressive trauma. Occupational trauma could lead to ossification of the ligaments of the dens, but as yet there is no evidence to support this hypothesis.

Alongside trauma, ligament ossification of the odontoid process has been associated with Crowned Dens Syndrome (CDS), a condition characterized by pyrophosphate or hydroxyapatite crystal deposition around the dens (Bouvet et al., 1985; Dirheimer and Wackenheim, 1974; Inoue et al., 2017). Calcification affects the transverse or alar ligament in most of the cases (Godfrin-Valnet et al., 2013; Goto et al., 2007; Koyfman and Yaffe, 2014), but could also affect the apical ligament (Sekijima et al., 2010; Soo Lee et al., 2014). Despite the fact that CDS has gained considerable attention during the last decade, the only case of CDS diagnosed in skeletal remains was described in a modern skeleton displaying apical ligament ossification (Prathap Kumar et al., 2014). Although initially considered a rare condition, CDS appears to be more common than first suspected, with a prevalence ranging between 2% (Goto et al., 2007) and 15.9% (Sano et al., 2017) in modern populations, and more frequently observed in women over 60 years old (Godfrin-Valnet et al., 2013; Sano et al., 2017).

The pathogenesis of CDS is not fully understood, and for that reason its differential diagnosis is problematic even in clinical cases (Sano et al., 2017). Considering that ossification is produced by the deposition of pyrophosphate or hydroxyapatite crystals, it has been suggested that CDS could be produced by Calcium Pyrophosphate Deposition Disease (CPPD) and Calcium Hydroxyapatite Deposition Disease (CHDD), but it has also been identified in cases of Diffuse Idiopathic Skeletal Hyperostosis (DISH) and ankylosing spondylitis (Ali et al., 2011; Baysal et al., 2000; Resnik et al., 1978). In DISH, the anterior or right longitudinal ligaments of the thoracic spine are ossified, with fusion of three or four vertebrae (Arlet and Maziéres, 1985; Holgate and Steyn, 2016; Resnick and Niwayama, 1976; Utsinger, 1985). However, bilateral calcified entheses in calcanii, patellae, ulnae, and the os coxae, alongside ossification of the apical ligament of the axis could be present in less advanced cases (Arlet and Maziéres, 1985; Holgate and Steyn, 2016). All these spinal and extraspinal lesions, with the exception of the ligament ossification around the dens, are absent in the two skeletons from the B6 site. For that reason, the findings described here appear to be incompatible with DISH, although the presence of initial stages of the disease cannot be completely rejected. Similarly, ankylosing spondylitis typically manifests as erosive lesions and/or ankylosis of the sacroiliac joint, which is frequently the first place affected by the disease and considered critical to its diagnosis (Aufderheide and Rodríguez Martín, 1998; Holgate and Steyn, 2016; Ortner, 2003). None of these changes are evident in the skeletons from the B6 site.

Calcification of the ligaments of the dens due to CDS is more frequently suggested to be a consequence of CPPD, and in some occasions is due to CHDD. Both disorders are characterized by crystal deposits in multiple appendicular and vertebral joints. Calcium Hydroxyapatite Deposition Disease is an idiopathic condition, appearing secondary to other metabolic diseases such as Vitamin D disorders or collagen vascular diseases, but is also linked to hereditary factors (Hayes and Conway, 1990; Schneider and Hirsch, 2017). Multiple deposition sites are common, including the cervical vertebrae, but the shoulders, wrist and fingers are the most frequently affected joints (Hayes and Conway, 1990). Deposition of hydroxyapatite crystals in tendon insertions might cause articular destruction and chronic periarticular bursitis and tendinitis at these anatomical locations (Kerl-Skurka et al., 2015; Schneider and Hirsch, 2017).

On the other hand, CPPD is a group of conditions in which calcium deposits appear in ligaments, bursae, articular cartilage, synovium, and capsules of multiple joints, including knees, hips and wrists (Lee et al., 2014; Richette et al., 2009; Rosenthal and Ryan, 2016).

Ligament calcification in the periodontoid joint is also frequently present (Abhishek, 2016; Baysal et al., 2000), and appears in higher prevalence than initially suspected (Chang et al., 2013). CPPD is also commonly related to OA, due to the fact that joint degeneration may be produced by crystal deposition, although the co-occurrence in aged individuals has suggested a possible explanation of this co-morbidity (Richette et al., 2009; Rosenthal and Ryan, 2016).

Taking into account that extraspinal heterotopic ossifications were not noted in the skeletons from the B6 site, CHDD seems to be an incompatible etiology for the ligament ossification in these skeletons. However, Transmission Electron Microscopy (TEM), a technique successfully employed in the identification of urate crystals in gouty individuals (Limbrey et al., 2011), could help clarify if hydroxyapatite crystals were deposited on the dens of the B6 skeletons. As TEM is a destructive method, it has not been carried out in these remains so far.

The absence of peripheral ossifications also suggests that CPPD did not affect these individuals. The recorded OA observed in Sk 2 and Sk 8 might not be related to CPPD, particularly because typical appendicular joints affected by CPPD, such as knees, wrists and hips, were not affected by OA in these individuals. However, the presence of CPPD cannot be completely rejected considering that crystal deposits could be present in some joint structures without bony changes. Diagnosis of CPPD deposition is often based on the presence of crystals in synovial fluid (Richette et al., 2009), which manifests as chondrocalcinosis, where fibro or hyaline cartilage of peripheral joints become calcified (Abhishek, 2016; Richette et al., 2009; Rosenthal and Ryan, 2016). In acute cases crystal deposits can be detected in joints that have never been inflamed (Richette et al., 2009). Consequently, acute cases of CPPD might not appear in archaeological skeletons if crystal deposits only affect soft tissues such as cartilage, synovium or joint capsules, and without calcifications of enthesis and tendons. Therefore, although no evidence related to CPPD was noted, other than the periodontoid ligament calcification, the initial stage of this disease cannot be completely excluded as a cause of CDS in the skeletons from the B6 site.

The age-at-death of both individuals is consistent with the possible development of CPPD, as periodontoid calcification rarely occurs in individuals under 40 years old (Abhishek, 2016). Another aspect to consider is the possible genetic pathogenesis of CPPD. More than one member of a family could be affected, particularly noted when it occurs before 60 years old, as a genetic mutation may result in a gain of function of the ANKH protein that regulates inorganic pyrophosphate levels (Williams et al., 2003). Since CPPD affected the two skeletons studied here, they could have been genetically related. Further phylogenetic analyses are needed to explore this aspect.

Clinical manifestations of CDS are acute or chronic neck pain, headache (usually occipital), and neck stiffness with restricted motion of the head associated with rotation (Aouba et al., 2004; Godfrin-Valnet et al., 2013; Salaffi et al., 2008; Wu et al., 2005). Thus, if Sk 2 and Sk 8 suffered CDS, these clinical features might have been present during their lives. Although acute cases of CPPD are usually asymptomatic, warmth, erythema, swelling and pain can occur in joints with crystal deposits.

5. Conclusions

Based clinical reports, and taking into account that no lesions indicative of neoplasia, OA, DISH, or ankylosing spondylitis were found in the two skeletons from the B6 site, soft tissue traumatic injuries without bone fracture appears to be the most probable cause for the elongated odontoid processes. However, the presence of Crowned Dens Syndrome produced by CPPD could not be completely rejected. To our knowledge, ligament ossification of the dens has not been described in the paleopathological literature. We hope this short communication will encourage others to pay attention to periodontoid ligament ossification and its possible association with trauma, usually diagnosed alongside the presence of fractures. Obviously, CDS needs to be considered a viable option in the differential diagnosis of morphological changes to the odontoid process in archaeological skeletal remains, as this will offer new data about this disease and its presence in individuals from ancient populations.

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References

Abhishek, A., 2016. Calcium pyrophosphate deposition disease: a review of epidemiologic findings. Curr. Opin. Rheumatol. 28, 133–139.
Ali, S., Hoch, M., Dadhania, V., Khurana, J.S., 2011. CPPD Crowned dens syndrome with

clivus destruction: a case report. J. Radiol. Case Rep. 5 (8), 30–37.
Anderson, L.D., D'Alonzo, R., 1974. Fractures of the odontoid process of the axis. J. Bone Jt. Surg. Am. 56, 1663–1674.
Aouba, A., Vuillemin-Bodaghi, V., Mutschler, C., De Bandt, M., 2004. Crowned dens syndrome misdiagnosed as polymyalgia rheumatica, giant cell arteritis, meningitis or spondylitis: an analysis of eight cases. Rheumatology 43, 1508–1512.
Arlet, J., Maziéres, B., 1985. La maladie hyperostosique. Rev. Med. Interne 6, 553–564.
Aufderheide, A.C., Rodríguez Martín, C., 1998. The Cambridge Encyclopedia of Human Paleopathology. Cambridge University Press, New York.
Barberena, R., Duran, V., Novellino, P., Winocur, D., Benítez, A., Tessone, A., Quiroga, M.N., Marsh, E.J., Gasco, A., Cortegoso, V., Lucero, G., Llano, C., Knudson, K.J., 2017. Scale of human mobility in the southern Andes (Argentina and Chile): a new framework based on strontium isotopes. Am. J. Phys. Anthropol. 164 (2), 305–320.
Barnes, E., 2012. Atlas of Developmental Field Anomalies of the Human skeleton. A Paleopathological Perspective. John Wiley & Sons, New Jersey.
Baysal, T., Baysal, O., Kutlu, R., Karaman, I., Mizrak, B., 2000. The crowned dens syndrome: a rare form of calcium pyrophosphate deposition disease. Eur. Radiol. 10, 1003–1005.
Bossche, L.V., Vanderstraeten, G., 2005. Heterotopic ossification: a review. J. Rehabil. Med. 37, 129–136.
Bouvet, J.P., Le Parc, J.M., Michalski, B., Benlahrache, C., Anquier, L., 1985. Acute neck pain due to calcifications surrounding the odontoid process: the crowned dens syndrome. Arthritis Rheum. 28, 1417–1420.
Brooks, S.T., Suchey, J.M., 1990. Skeletal age determinations based on the os pubis: a comparasion of the Acsadi-Nemeskeri and Suchey-Brooks methods. Hum. Evol. 5 227e238.
Buikstra, J.E., Ubelaker, D.H., 1994. Standard for Data Collection from Human Skeletal Remains. Arkansas Archaeological Survey Research Series N°44. Arkansas. .
Chang, E.Y., Lim, W.Y., Wolfson, T., Gamst, A.C., Chung, C.B., Bae, W.C., Resnick, D.L., 2013. Frequency of atlantoaxial calcium pyrophosphate dihydrate deposition at CT. Radiology 269 (2), 519–524.
Che Mohamed, S.K., Abd Aziz, A., 2009. Calcification of the alar ligament mimics fracture of the craniovertebral junction (CVJ): an incidental finding from computerized tomography of the cervical spine following trauma. Malays. J. Med. Sci. 16 (4), 69–72.
Curate, F., 2008. A case of *Os odontoideum* in the palaeopathological record. Int. J. Osteoarchaeol. 18, 100–105.
Dirheimer, Y., Wackenheim, A., 1974. Crowned odontoid apophysis. Apropos of the development mechanism of certain images of axis manifestations. Sem. Hop. 50, 2077–2080.
Gil, A.F., Villalba, R., Ugan, A., Cortegoso, V., Neme, G., Michieli, C.T., Duran, V., 2014a. Isotopic evidence on human bone for declining maize consumption during the little ice age in central western Argentina. J. Archaeol. Sci. 49, 213–227.
Gil, A.F., Neme, G.A., Ugan, A., Tykot, R.H., 2014b. Oxygen isotopes and human residential mobility in Central Western Argentina. Int. J. Osteoarchaeol. 24, 31–41.
Godfrin-Valnet, M., Godfrin, G., Godard, J., Prati, C., Toussirot, E., Michel, F., Wendling, D., 2013. Eighteen cases of crowned dens syndrome: presentation and diagnosis. Neurochirurgie 59, 115–120.
Goto, S., Umehara, J., Aizawa, T., Kokubun, S., 2007. Crowned dens syndrome. J. Bone Jt. Surg. Am. 89 (12), 2732–2736.
Hayes, C.W., Conway, W.F., 1990. Calcium hydroxyapatite deposition disease. Radiographics 10, 1031–1048.
Hensinger, R.N., Fielding, J.W., Hawkins, R.J., 1978. Congenital anomalies of the odontoid process. Orthop. Clin. North Am. 9, 901–912.
Holgate, R.L.V., Steyn, M., 2016. Diffuse idiopathic skeletal hyperostosis: diagnostic, clinical, and paleopathological considerations. Clin. Anat. 29 (7), 870–877.
Inoue, A., Kanehisa, K., Satoko, N., Hitomi, T., Shinji, I., Shiro, O., Kenji, K., Kensho, O., Shinya, F., Haruhisa, I., Shinji, O., Saya, O., Bungo, O., 2017. Usefulness of cervical computed tomography and magnetic resonance imaging for rapid diagnosis of crowned dens syndrome: a case report and review of the literature. Int. J. Surg. Case Rep. 30, 50–54.
Jain, N., Verma, R., Garga, U.C., Baruah, B.P., Jain, S.K., Bhaskar, S.N., 2016. CT and MR imaging of odontoid abnormalities: a pictorial review. Indian J. Radiol. Imag. 26, 108–119.
Jurmain, R., 1990. Paleoepidemiology of a central California prehistoric population from CA- ALA-329: II. Degenerative disease. Am. J. Phys. Anthropol. 83, 83–89.
Kerl-Skurka, A., Flueckiger, B., Ali, S., Kilgus, M., Theiler, R., 2015. Severe bursitis in shoulder arthritis – a manifestation of hydroxyapatite crystal deposition disease. Case Rep. Clin. Pathol. 2 (2), 7–11.
Korres, D.S., Chytas, D.G., Markatos, K.N., Efstathopoulos, N.E., Nikolaou, V.S., 2017. The "challenging" fractures of the odontoid process: a review of the classification schemes. Eur. J. Orthop. Surg. Traumatol. 27 (4), 469–475.
Koyfman, A., Yaffe, D., 2014. Crowned dens syndrome. A case report. Neuroradiol. J. 27, 495–497.
Lee, G.S., Kim, R.S., Park, H.K., Chang, J.C., 2014. Crowned dens syndrome: a case report and review of the literature. Korean J. Spine 11 (1), 15–17.
Limbrey, S., Brickley, M., Marques, C., Swinson, D., 2011. Identification of urate crystals in gouty individuals. J. Archaeol. Sci. 38, 2497–2501.
Lovejoy, C., Meindl, R., Mensforth, R., Barton, T., 1985. Multifactorial determination of skeletal age at death: a method and blind tests of its accuracy. Am. J. Phys. Anthropol. 68 1e14.
Mann, R.W., Tuamsuk, P., Hefner, J.T., 2013. Aplasia of the os odontoideum: skeletal manifestation from Thailand. Int. J. Paleopat. 3 (4), 302–306.
McCarthy, E.F., Sundaram, M., 2005. Heterotopic ossification: a review. Skeletal Radiol. 34 (10), 609–619.
Novellino, P., Brachetta Aporta, N., D'Addona, L.A., Estrella, D., Bernal, V., Devincenzi, S., Cortegoso, V., Durán, V., 2013. Sitios de entierro de la localidad arqueológica Barrancas, Maipú (Mendoza, Argentina). Intersecciones en Antropología 14, 271–277.
Ortner, D.J., 2003. Identification of Pathological Conditions in Human Skeletal Remains, 2nd ed. Elsevier Science/Academic Press, New York.
Prathap Kumar, J., Anupama, K., Radhika, P.M., Komala, N., 2014. Elongated odontoid process of axis vertebra. Int. J. Anat. Res. 2 (3), 594–596.
Resnick, D., Niwayama, G., 1976. Radiographic and pathologic features of spinal involvement in diffuse idiopathic skeletal hyperostosis (DISH). Radiology 119, 559–568.
Resnik, D., Guerra, J., Robinson, C.A., 1978. Association of diffuse idiopathetic skeletal hyperostosis (DISH) and calcification and ossification of the posterior longitudinal ligament. AJR Am. J. Roentegenol. 131 (6), 1049–1053.
Richette, P., Bardin, T., Doherty, M., 2009. An update on the epidemiology of calcium pyrophosphate dihydrate crystal deposition disease. Rheumatol. (Oxf. 48 (7), 711–715.
Rogers, J., Waldron, T., 1995. A Field Guide to Joint Disease in Archaeology. Wiley, Chichester.
Rojas-Sepúlveda, C., Ardagna, Y., Dutour, O., 2008. Paleoepidemiology of vertebral degenerative disease in a Pre-Columbian muisca series from Colombia. Am. J. Phys. Anthropol. 135 (4), 416–430.
Rosenthal, A.K., Ryan, L.M., 2016. Calcium pyrophosphate deposition disease. N. Engl. J. Med. 374 (26), 2575–2584.
Salaffi, F., Carotti, M., Guglielmi, G., Passarini, G., Grassi, W., 2008. The crowned dens syndrome as a cause of neck pain: clinical and computed tomography study in patients with calcium pyrophosphate dihydrate deposition disease. Clin. Exp. Rheumatol. 26, 1040–1046.
Sano, M., Yamashita, S., Aiba, T., 2017. The prevalence of calcification around odontoid process and the incidence of crowned dens syndrome in the neurosurgical ward: a single institution's analysis. Mod. Rheumatol. 28 (1), 182–187. http://dx.doi.org/10.1080/14397595.2017.1316461.
Schneider, D., Hirsch, M., 2017. Acute calcific tendonitis of dorsal interosseous muscles of the hand: uncommon site of a frequent disease. Reumatismo 69 (1), 43–46.
Sekijima, Y., Yoshida, T., Ikeda, S., 2010. CPPD crystal deposition disease of the cervical spine: a common cause of acute neck pain encountered in the neurology department. J. Neurol. Sci. 296 (1–2), 79–82.
Sim, K.B., Park, J.K., 2006. A nodular calcification of the alar ligament simulating a fracture in the craniovertebral junction. Am. J. Neuroradiol. 27 (9), 1962–1963.
Soo Lee, G., Kim, R.S., Park, H.K., Chang, J.C., 2014. Crowned dens syndrome: a case report and review of the literature. Korean J. Spine 11 (March (1)), 15–17 2014.
Todd, T., 1921a. Age changes in the pubic bone. I: the male white pubis. Am. J. Phys. Anthropol. 3 285e334.
Todd, T., 1921b. Age changes in the pubic bone. III: the pubis of the white female. IV: the pubis of the female white-negro hybrid. Am. J. Phys. Anthropol. 4 1e70.
Utsinger, P.D., 1985. Diffuse idiopathic skeletal hyperostosis. Clin. Rheum. Dis. 11, 325–351.
Weber, J., Czarnetzki, A., Spring, A., 2003. Paleopathological features of the cervical spine in the Early Middle Ages: natural history of degenerative diseases. Neurosurgery 53 (6), 1418–1424.
Williams, C.J., Pendleton, A., Bonavita, G., Reginato, A.J., Hughes, A.E., Pearlso, S., Doherty, M., McCarty, D.J., Ryan, L.M., 2003. Mutations in the amino terminus of ANKH in two US families with calcium pyrophosphate dihydrate crystal deposition disease. Arthritis Rheum. 48, 2627–2631.
Wu, D.W., Reginato, A.J., Torriani, M., Robinson, D.R., Reginato, A.M., 2005. The crowned dens syndrome as a cause of neck pain: report of two new cases and review of the literature. Arthritis Rheum. 53 (1), 133–137.