Histopathological findings of apical fracture of the proximal sesamoid bones in young Thoroughbred foals

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Although radiographic findings at the apical portion of the proximal sesamoid bone (PSB) are often observed in young Thoroughbred foals, conflicting findings, either fractures or apparent secondary ossifications centers, have been reported. Three cases (aged 2, 5, and 7 weeks) were identified in 30 necropsied foals (0–31 weeks old). Histopathologically, the subchondral trabecular woven bone was fractured and exhibited focal necrosis of woven bone, fibrin exudate, and/or fibrosis within the foci. In the 7-week-old case, proliferations of chondrocytes were also observed. These findings suggest that the radiographic findings represented a healing process of the apical PSB fractures associated with the mechanically damaged subchondral trabeculae. Developmental PSB injuries should be taken into consideration during the management of young Thoroughbred foals.

Key words: foal, proximal sesamoid bone fracture, subchondral lesion

The equine proximal sesamoid bones (PSBs) are small, paired, almond-shaped bones present on the palmar and plantar surfaces of the fetlock joints. They form an important part of the suspensory apparatus of the fetlock joints with the suspensory and sesamoidean ligaments that exert tensile forces during strenuous exercise [11]. Apical PSB fracture of the forelimb is the most common injury in racehorses. This is because it is caused by a combination of fetlock joint hyperextension and suspensory tensile forces [11, 13]. Conversely, the PSB is poorly developed in young foals because ossification of it begins at the end of the gestation period (290–330 days) [2, 7]. Poor PSB development in young foals likely makes it more vulnerable to injury. The PSBs within the suspensory apparatus may sometimes be subjected to tensile forces when the young foals attempt to keep pace with the mares sprinting across an open field [7]. This may lead to a small, isolated, nonunion but non-displaced bone occurring within the apical portion of the PSB; the nonunion is radiographically apparent with a radiolucent line in young foals aged less than 2 months [1–3, 7, 10, 13]. Because of the absence of clinical signs, these small nonunion may represent a controversial condition: either a non-displaced fragment of a PSB fracture or an apparent secondary ossification center of the PSB [1–3, 6–8, 10, 12–14]. Bipartite PSBs are presumed to be of congenital and not traumatic origin and are present at the age of 2–8 weeks; they fuse at the age of 4 months to form a prominent apical enthesophyte [12, 14]. This assumption also applies to the non-displaced fragments of PSB fractures. Several studies have described PSB fractures in young foals as being of traumatic origin [1–3, 6, 10, 11, 13]. In most cases, the clinical signs are subtle, with slight or no lameness, and the prognosis is good [2, 6]. However, the histopathological features of a small, isolated bone within the apical portion of PSB are still unclear. The purpose of the present study was to examine the histopathologic characteristics of the radiographic findings of the PSB in young Thoroughbred foals.

The subjects were 30 Thoroughbred foals aged less than 7 months (age range: 0–31 weeks) that had either died or
been euthanized with diseases other than fracture of the PSB in dissection rooms. All four limbs of each foal were checked for the presence of radiographic findings within the apical portion of the PSBs using a digital radiography system (AeroDR1012, Konica Minolta Inc., Tokyo, Japan). The limbs that were confirmed to exhibit radiographic findings within the apical portion of the PSBs were subjected to additional radiographic examinations to determine the side (medial or lateral) of the PSB on which they occurred. A total of 3 foals (aged 2, 5, and 7 weeks) exhibited radiographic findings within the apical portion of PSBs, and necropsy and histopathological investigations of the PSBs were performed. General information and lateromedial radiograph images of the cases are shown in Table 1 and Fig. 1, respectively.

In Case 1, macroscopically a micro-focal liner lesion within the apical portion of the medial PSB of the left forelimb was detected (Fig. 2A). The palmar fibrocartilage and dorsal hyaline cartilage were intact. Histopathologically, the focus was detected within the apical part of the PSB, corresponding to the macroscopic observation (Fig. 2B). The focus consisted of centrally located necrotized eosinophilic trabecular bones and fibrin (Fig. 2C and 2D). Although fibrosis was not observed within the necrotizing area, mild proliferation of fibroblasts was seen in the surrounding trabecular lumen around the necrotic focus. Furthermore, although no radiographic findings were observed within the apical portion of the medial and lateral PSBs of both hindlimbs, a liner cleft within the apical portion of the lateral PSBs of both hindlimbs was observed macroscopically. Histopathologically, the cleft was observed at the boundary subchondral region between the cartilage and the bone body at the apical portion of the PSB, and it was filled with fibrin (data not shown). It would be revealed that subtle injury had also occurred in radiographically normal PSBs.

In Case 2, the macroscopic liner lesions in both lateral PSBs of the left and right forelimbs were more obvious than Case 1 (Fig. 2E and 2I). Histopathologically, a liner to zonal focus within the trabecular bone of the lateral PSB was observed (Fig. 2F and 2J). In particular, the focus of the left PSB was elongated and reached the cartilage layer (Fig. 2F). The affected cartilage was necrotized: the cartilage layer and chondrocytes within the focus lost their stainability due to necrosis (Fig. 2G). At the liner focus of the trabecular bone, a thin fibrinous layer and micro-focal necrotized trabecular bone were surrounded by sparsely proliferated immature fibroblasts and connective tissue (Fig. 2G and 2H). Multiple micro-focal hemorrhages were also seen in the connective tissue (Fig. 2G). In the liner focus of the right PSB, densely proliferated immature fibroblasts and connective tissue were observed (Fig. 2K and 2L). In addition, osteoclasts increased in number around the focus (Fig. 2L).

In Case 3, cicatricial lines within the apical portion of the medial and lateral PSBs of the left forelimb were observed macroscopically (Fig. 2M and 2Q). The focus of the medial PSB was filled with loose connective tissue and matured fibroblasts that were arrayed along the vertical axis (Fig. 2N, 2O and 2P). In the focus of the lateral PSB, the densities of the proliferated connective tissue and fibroblasts were higher than that of those of the medial PSB, and proliferation of chondrocytes was also observed within the fibroblast tissue (Fig. 2R, 2S and 2T).
The present study included 3 Thoroughbred foals aged less than 7 weeks that exhibited radiographic finding within the apical portion of their PSBs. Although the severity of the lesions varied from mild (only a slight line) to moderate (damaged around cartilage) in each case, proliferation of fibroblast tissue in the dehiscence of the trabecular bone was commonly observed in all cases. Furthermore, the trabecular lesion observed in the 7-week-old foal was filled with connective tissues with cartilaginous tissue formations, that is to say, a soft callus (Fig. 2S and 2T) [4]. Although it is not certain when the trabecular lesion occurred in this case, it is presumed that an appreciable time had already passed. These findings suggest that the radiographic findings represented a repair process of the apical PSB fractures associated with the mechanically damaged subchondral trabeculae. To date, there are no histopathological reports that focus on the radiographic findings within the apical portion of the PSB in young Thoroughbred foals. Therefore, the proximal one-third of the nonunion bone is often mistaken to be a bipartite bone originating from a second ossification center of the PSB [3, 12, 14]. However, there was no evidence of the presence of any secondary ossification centers in the equine PSBs in the present study.

The PSB is an organ comprising cancellous trabeculae. It has been hypothesized that cancellous bone fracture healing follows a pathological process comprising bleeding, cell proliferation, woven bone formation, lamellar bone formation, and bone remodeling stages [8]. In comparison with diaphyseal cortical bone healing, cancellous bone fractures exhibit less bone necrosis, barely any hematoma formation, limited inflammatory events, and no external callus formation [8, 9]. PSBs histologically exhibit poor blood supply and are encased within ligaments and cartilages. In the present 3 cases, there was very few hemorrhage in the lesions. The lack of an obvious hemorrhage might be due to poor blood supply of PSB.

The equine PSB begins as a cartilaginous nodule that undergoes endochondral ossification during late pregnancy and is not fully mineralized until 3 months of age [3]. In particular, the apical region of the PSB is a thriving site for cartilage cell division. In the process of endochondral ossification, the grown cartilage cells undergo apoptosis and calcification of the matrix, and the continuous ossified zone forms an open mesh structure comprising an immature trabecular bone called woven bone. This woven bone, distributed under the growing cartilage tissue, forms a site that is weak with respect to physical stress. Tension on ligaments may cause damage as well, provoking avulsion fractures of epiphyseal or metaphyseal ossifying bone, which are classified as juvenile osteochondral conditions [5]. The biomechanical insults may also eventually result in osteochondral fragmentation. In Case 1, although the lateral PSB of the left hindlimb had no radiographic findings within the apical portion, a cleft at the boundary region between the cartilage and the trabecula bone was observed histopathologically. In young foals, the chondral tissue of the apical portion of the PSB is an actively growing part. The abovementioned cleft would heal via proliferation of fibroblast tissue, and at the same time, the avulsed osteochondral tissue might grow bigger itself via endochondral ossification. These are thought to be mechanisms by which the avulsed osteochondral tissue changes into an isolated bone at the apical portion of the PSB with a radiolucent line.
in its healing process.

There are few reports on the onset of an apical fracture of the proximal sesamoid bones in young foals. Kroll et al. reported that isolated opacities proximal to the ossified parts of the PSBs were observed on the radiographs of 10.7% of foals (14/130 foals) [12]. In the present study, the radiographic finding within the apical portion of the PSBs were observed on the radiographs of 10.0% of foals (3/30 foals). The incidence rate was similar to that in the previous report. However, the foals targeted in this study included foals that died without sufficient exercise due to infectious disease, injuries, or congenital abnormalities, and the incidence rate in healthy foals may be greater. Further investigation is needed to clarify the incidence of apical PSB fracture in healthy young foals.

The radiographic findings within the apical portion of the PSBs in Thoroughbred foals are often nonclinical and discovered incidentally during radiographic examinations, and they generally heal without incident [2]. However, the healing process may be delayed in severe cases exhibiting damage of the surrounding cartilage tissue. Excessive physical stress may be a risk factor for this type of injury. Therefore, it is important to reexamine the pasturing methods used for young Thoroughbred foals to prevent the occurrence of severe apical PSB fractures. Future studies should focus on the progress and treatment of apical PSB fractures and influence of the racing period on the breeding of Thoroughbred racehorses.

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