Pseudogout, also referred to as “chondrocalcinosis” or “calcium pyrophosphate dehydrate (CPPD) crystal deposition disease”, is mainly considered a common disease affecting the knee joint of elderly people, but is rare in animals. Horses and dogs may have pseudogout, particularly in locations subject to increased mechanical use, most often in the carpal region [3, 5, 6]. There are 3 reports of this disease in reptiles, but no clinical diagnosis, procedures, or prevalence are presented [2, 14]. Here we report a case of pseudogout surgically removed from a painted turtle (Chrysemys picta).

A 5-year-old, male painted turtle (Chrysemys picta) was referred to an animal clinic, with a complaint of a nodular enlargement of the right femorotibial joint (Fig. 1). The affected area exhibited multinodular calcification in radiography (Fig. 2). It gradually grew larger during the following month, and made it impossible to bend the leg into the carapace. Enlargement of the other hind limb was also detected at the time. Accordingly, the right leg was amputated at the coxofemoral joint for diagnostic therapy, and a formalin-fixed sample was examined pathologically. The surgical incision healed favorably, and body weight increased slightly. Later, reflection of the left foot was lost, and the tortoise died, two months after the procedure. We were not able to perform a necropsy.

The nodule was 1cm in diameter, and felt gritty and frail when cut with a knife. The cut surface was yellowish white in color, and colloidal to chalky in gross appearance (Fig. 1). The sample was cut longitudinally at the femorotibial joint after decalcification, and conventional paraffin embedded sections were made.

Microscopically, multinodular lesions could be seen in the periphery of the articular cavity (Fig. 3). Each nodule was bounded by a thin layer of connective tissue, which showed partial cartilaginous metaplasia. Within the nodule, many deposits of variable size and shape were observed. These deposits were basophilic, and were positive for von Kossa’s reaction in undecalcified tissue section. Tissue sections stained with neutral alcoholic eosin staining [12] revealed numerous rod- and pleomorphic-shaped crystals with weakly positive birefringence under the polarizing microscope (Fig. 4). Macrophages and a few heterophils surrounded the deposits, and giant cells of foreign-body type and ghost cells were observed in some regions. Adjacent skeletal muscles showed atrophy, accompanied by mononuclear cell infiltration. Microorganisms were not observed anywhere in the sections.

Many deposits of the same kind were also observed within the articular cavity, characterized by notable formation of a foreign-body granuloma originating from the articular capsule. The surface of the articular cartilage was severely contoured by erosion and cracking (Fig. 4). Alcian blue staining revealed a decrease in proteoglycan content of the articular cartilage. Focal necrosis and reactive proliferation of chondrocytes was prominent in the afflicted area. Fibrosis occurred around the epiphyses.

The above findings demonstrated that the constituent of the deposits was calcium pyrophosphate [12]. In general, two primary types of crystal arthritides are known, gout caused by deposition of urates, and pseudogout caused by deposition of calcium pyrophosphate. Gout is a common affliction in some reptiles, since they lack uricase, which degrades uric acid [9]. However, urate crystals, characterized by their needlelike shape, were not detected in this case. In addition, histological findings revealed that the articular cartilage was mainly involved in the pathological onset of the disease, and therefore the case was diagnosed as tophaceous pseudogout.

It is considered that CPPD deposition is related to levels
of inorganic pyrophosphate (PPI) [1, 7]. Therefore, CPPD deposition is initiated around the chondrocytes, and eventually emigrates from the cartilage lacuna to the articular cavity, where the inflammatory response is initiated [8, 11]. Prostaglandin E2 and NO, produced along with inflammatory response, are known to suppress cartilage proteoglycan synthesis, resulting in cartilage disruption [4, 13]. In this case, a large amount CPPD accreted, forming multi-nodular foreign-body granuloma around the right femorotibial joint.

The scale of the lesion showed that the sequence of pathological alteration occurred over a long period of time, causing severe injury to the articular cartilage. In humans, pseudogout is divided into idiopathic, hereditary, and secondary types. Various disorders, including previous joint damage, hyperparathyroidism, hemochromatosis, hypomagnesemia, hypothyroidism, ochronosis, diabetes, and Wilson’s disease are associated with the secondary type. Though excessive feeding of shrimp, which contain large...
amounts of calcium, or a diet for mammals, which contains excessive amounts of vitamin D, is inferred to be causative in some reports, the etiology of the disorder in reptiles is not clear [10].

This case may have had chronic renal disease or imbalance of nutrition which induced metastatic calcinosis, because it had plural analogous lesions. However, the presence of such factors was not confirmed.

The fact that reptiles share the same pathological characteristic of pseudogout with humans is quite intriguing from a comparative medical point of view.

REFERENCES