—NOTE—

Two Cases of Adiaspiromycosis in the Japanese Pika (*Ochotona hyperborea yesoensis* Kishida)

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ABSTRACT. Present report described the morphology of pulmonary adiaspiromycosis caused by *Chrysosporium parvum* var. *crescens* in two Japanese pikas (*Ochotona hyperborea yesoensis* Kishida).—**KEY WORDS:** adiaspiromycosis, Japanese pika.

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Adiaspiromycosis caused by *Chrysosporium* (*C. parvum* var. *crescens* and *C. parvum* var. *parvum*) is known as a pulmonary mycosis in humans and many wild small animals such as ground squirrels, pocket mice, kangaroo rats, beavers, cottontail rabbits, mink and raccoons in many countries [1, 2, 7, 11]. In Japan, we have only a few reports about adiaspiromycosis caused by *C. parvum* var. *crescens*, formerly called *Emmonsia crescens* (*Eothenomys smithii* in Aomori and *Apodemus speciosus* in Iwate and Miyagi [6], and *Apodemus argenteus* in Hokkaido [8]). This report describes two cases of the Japanese pika (*Ochotona hyperborea yesoensis* Kishida) affected with this disease.

Two animals (adult male and female pikas) were captured in the Taisetsu Range in Hokkaido, on July 1972, and have been bred at Obihiro zoological garden. In early September of that year, suddenly they grew weak, accompanied by diarrhea, leading to death after several days.

Necropsy of the dead animals revealed the numerous grayish white foci about 1 mm in diameter in all lobes of the lungs. The male pika had also an atelectatic lesion in the anterior lobe of right lung. No significant changes were found in any other organs and

tissues of them. Numerous grayish white foci in the lungs were found beneath the pulmonary pleura and deep in the pulmonary parenchyma. Some foci showed slight protrusion on the surface of the lungs. A spherical, translucent adiaspore separated easily with a needle was found on the cut surface of the focus. Tissue blocks were collected from the lungs, fixed in 10% formalin solution, and embedded in paraffin. Serial tissue sections were prepared and stained with haematoxylin and eosin (HE). Special stain was conducted using periodic acid-Schiff (PAS), azan and Grocott's methenamine silver.

Microscopically, the adiaspores were large enough in the pulmonary alveoli to press the surrounding pulmonary tissue (Fig. 1). The adiaspores in spherical shape were about 300 to 420 μm in diameter and composed of the eosinophilic round-thick wall and immanent basophilic fine granular materials in the sections stained with HE. The wall was 25 to 47 μm in thickness and composed of three layers; an outer layer was refractive, 2 to 6 μm in thickness and strongly stained with eosin, an intermediate layer of red stain was striped and 6 to 7 μm in thickness, and inner layer was homogenous, 26 to 32 μm in thickness and faintly stained with eosin (Fig. 2).

Fig. 1. Lung showing multiple foci. Azan, $\times 8$.

Fig. 2. Enlarged adiaspore in the pulmonary alveolus. Host tissue reaction is negative. HE, $\times 100$.

Fig. 3. Granuloma included the degenerative adiaspore consists of histiocytes and a few lymphocytes. HE, $\times 100$.

The wall and granular materials were stained purple by PAS, while the inner layer was most strongly stained. The adiaspores were also stained black by Grocott's methenamine silver. Budding or endosporulation of adiaspores were not observed. The reaction of host tissue to the adiaspores was generally negative or feeble in terms of infiltration of histiocytic cells (Fig. 2). Some degenerative

adiaspores were surrounded by the granulomatous tissue consisting of histiocytic cells, lymphocytes and rarely polymorphocytes (Fig. 3). Fibrosis was not observed. Extensive necrosis of pulmonary parenchyma with haemorrhage, thrombogenesis in the blood vessels and prominent infiltration of polymorphocytes was recognized in the atelectatic

Fig. 4. The necrotic lesion in the right lung of the male pika contains many hyphae of the zygomycete. HE, $\times 100$.

lesion of the right lung of the male pika. Numerous, irregularly branched hyphae (which were broad, infrequently septate and thin-walled) observed in the necrotic tissue and thrombus were characteristic of the zygomycetes (Fig. 4).

Mature adiaspores of *C. parvum* var. *crescens* in the pulmonary parenchyma reach a diameter of 200 to 400 μm and their walls range from 10 to 80 μm in thickness, while adiaspores of *C. parvum* var. *parvum* reach a diameter of 40 μm , their walls are 2 to 4 μm thick [4]. In adiaspiromycosis, the intervening pulmonary tissues are usually unaltered except for the compressive effects of adjacent adiaspores. Alveolar septa do not usually contain inflammatory cells. However, in a few cases, adjacent alveolar spaces may contain edematous fluid and macrophages [1]. The gross lesions, the histological appearance, the morphology and staining reactions of the adiaspores of our cases were identical to those of adiaspiromycosis caused by *C. parvum* var. *crescens* in humans and small wild mammals so far reported [1, 3, 6, 8, 9]. Though the adiaspiromycosis caused by *C.*

parvum var. *crescens* of the pika has been reported in Montana, U.S.A [5], and Alberta, Canada [10], this is the first report about the presence of this disease in the Japanese pika.

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要 約

ニホンナキウサギにおける *adiaspiromycosis* の2例 (短報): 谷山弘行・古岡秀文・松井高峯・小野 威 (帯広畜産大学家畜病理学教室) ——北海道大雪山系で捕獲され、帯広動物園で飼育されていたニホンナキウサギの肺に認めた *Cryosporium* による病変について病理組織学的に検索した。病変部に認めた直径 300~420 μm の *adiaspore* は、その大きさ、構造および染色性から、これまで多くの野生小型哺乳動物の肺に報味されてきた *C. parvum* var. *crescens* による *adiaspiromycosis* のそれに一致し、ニホンナキウサギにおける本病の存在が明らかになった。