



First report of subcutaneous *Taenia crassiceps* cysticercosis in a dog in Poland

Dawid Jańczak^{1,2,A-D} , Jakub Kędziorek^{2,B} , Karolina Ściubisz^{3,B} , Karolina Tomasz^{4,B} , Joanna Taborska^{4,C} , Kamil Kowalczyk^{4,C} , Maksymilian Lewicki^{5,C} , Olga Szaluś-Jordanow^{6,D-F}

¹ Department of Infectious and Invasive Diseases and Veterinary Administration, Institute of Veterinary Medicine, Faculty of Biological and Veterinary Sciences, Nicolaus Copernicus University, Toruń, Poland

² Department of Parasitology and Molecular Biology, Animallab Veterinary Laboratory, Poland

³ Canine and Feline Nutrition Specialist, Pupil Veterinary Clinic, Karolina Ściubisz, DVM, Gliwice, Poland

⁴ Veterinary Clinic, AniCura, Gliwice, Poland

⁵ Department of Internal Diseases with Clinic for Horses, Dogs and Cats, Faculty of Veterinary Medicine, University of Environmental and Life Sciences, Wrocław, Poland

⁶ Department of Small Animal Diseases with Clinic, Institute of Veterinary Medicine, Warsaw University of Life Sciences-SGGW, Warsaw, Poland

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Abstract

A 3-year-old male German Shepherd with Addison's disease, hematological abnormalities, and chronic glucocorticoid therapy developed subcutaneous nodules containing abundant cestode larvae. Microscopy, PCR, and mtCO1 sequencing confirmed *Taenia crassiceps* metacestodes (GenBank accession no. PX219651). This unusual case illustrates a domestic dog as an aberrant intermediate host with both subcutaneous and peritoneal cysticercosis, likely favored by long-term immunosuppression. The findings align with previous reports in immunocompromised hosts and emphasize clinical awareness of atypical parasitic infections. Cytology and molecular assays proved valuable for diagnosis. Given the zoonotic potential of *T. crassiceps* and its broad host range, early recognition in immunocompromised animals is critical for intervention, case management, and prevention. A One Health approach integrating veterinary and medical perspectives is essential to reduce its impact as a parasitic threat.

Key words

Poland, dog, immunosuppression, cysticercosis, COX-1, *Cysticercus longicollis*

INTRODUCTION

Taenia crassiceps (Zeder, 1880) (Cyclophyllidae, Taeniidae) is a cestode species with a complex and heteroxenous life cycle. In most cases, rodents and lagomorphs serve as intermediate hosts in which the larval stage develops as a metacestode (*cysticercus longicollis*) [1, 2]. The larval stage develops within the body cavities of the intermediate host, where it undergoes continuous asexual proliferation, producing numerous scolices [3].

The adult tapeworm of this species develops in the small intestine of domestic dogs, various wild canids, and in rare cases, in cats. The species is widely distributed across the Northern Hemisphere, where its transmission cycles are maintained in both sylvatic and domestic environments [4, 5, 6].

Foxes constitute a key reservoir as definitive hosts, and the prevalence of this tapeworm has been reported at 2.7% in Italy [7]. Moreover, *T. crassiceps* DNA was detected in stool samples collected from two privately owned dogs in the Sub-Carpathian Province of south-east Poland [8], and six domestic dogs in Switzerland [9].

✉ Address for correspondence: Dawid Jańczak, Department of Infectious and Invasive Diseases and Veterinary Administration, Faculty of Biological and Veterinary Sciences, Institute of Veterinary Medicine, Nicolaus Copernicus University, Lwowska 1, 87-100 Toruń, Poland
E-mail: parazytologia.vet@gmail.com

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While several studies have detected *T. crassiceps* DNA in environmental samples, evidence for the presence of viable eggs and, consequently, for a real risk of infection through environmental exposure, such as contaminated soil, water, fruits or vegetables, remains limited [10, 11].

The documented cases of *T. crassiceps* infections in humans indicate that this parasite should be regarded as a genuine zoonotic threat, particularly in immuno-compromised individuals. Molecular diagnostic methods enabled the unambiguous identification of tissue-invasive *Taenia* larvae in human specimens, confirming that *T. crassiceps* is capable of crossing species barriers and establishing infection in atypical hosts [12]. Moreover, although human infections remain relatively rare, the accumulation of multiple well-documented cases of *T. crassiceps* cysticercosis, particularly under conditions of immune suppression or immuno-modulation strongly supports the view that zoonotic transmission is both biologically plausible and clinically significant [11].

In rare cases, animals that usually function as definitive hosts in the life cycle of Taeniidae may instead become accidental intermediate hosts. This aberrant development is typically associated with establishing larval stages within atypical tissues, often resulting in progressive and debilitating lesions. Such infections are generally identified incidentally, for instance, during necropsy or advanced imaging, and are usually diagnosed at a stage too advanced for effective therapeutic management [13].

Cysticercosis caused by *T. crassiceps* has previously been reported in dogs in North America [1, 14], with additional cases described in Europe [15]. In Poland, *T. crassiceps* was identified in a captive ring-tailed lemur (*Lemur catta*) in Opole, south-west Poland [16], and in a common vole (*Microtus arvalis*) from the Masuria region in north-east Poland [2].

The case report presents a fatal case of subcutaneous *T. crassiceps* cysticercosis in a pet dog, which, to the best of our knowledge, represents the first such case documented in Poland.

CASE REPORT

The clinical history concerns a 3-year-old male German Shepherd dog, first presented in 2022, residing in the Silesian Province in south-west Poland, with no history of international travel. At that time, the owner reported anorexia and exercise intolerance during walks, and the results of a blood test submitted for consultation indicated chronic anaemia. Suspecting autoimmune haemolytic anaemia (AIHA), treatment with prednisone was initiated at a dose of 2 mg/kg body weight (Encorton 20 mg tablets, Adamed Pharma SA), which improved haematological parameters. However, during attempts to taper and subsequently discontinue the steroid therapy, the dog developed fever and dyspnea. Cardiologist examination revealed a reduction of left ventricular Lung ultrasound examination confirmed pneumonia. Metronidazole was introduced into the treatment protocol (Metronidazole 0.5% Polpharma, 5 mg/mL – solution for injection and infusion, Zakłady Farmaceutyczne POLPHARMA SA).

At the same time, erythrocytic parameters again deteriorated. Despite the reduced levels of red blood cells, haematocrit and haemoglobin, the patient's condition remained stable following treatment for pneumonia. Subsequent diagnostic tests confirmed Addison's disease, and prednisone therapy was reinstated. Under this treatment, no further episodes of fever, dyspnea, or anorexia were observed.

The patient later underwent castration with removal of a cryptorchid testis, after which an Addisonian crisis occurred, which was managed by administration of a primary dose of prednisone (2 mg/kg body weight). The patient's condition subsequently stabilized, and the prednisone dose was gradually tapered over time.

The owner became concerned about the appearance of subcutaneous lesions in the perianal region. Cytological examination revealed that these changes had developed as a result of calcium deposits.

In June 2025, the patient again showed a decline in erythrocytic parameters and became apathetic. The prednisone dose was increased from 0.1 mg/kg BW to 1 mg/kg BW; however, this adjustment did not fully improve the haematological values. In addition, an inflammatory process associated with the perianal subcutaneous lesions was observed. The dog developed a doughy oedema in the distal parts of the pelvic limbs, as well as decubital ulcers.

A bacteriological swab was collected from one of the wounds, yielding *Pseudomonas aeruginosa*, *Serratia marcescens*, *Providencia rettgeri*, *Streptococcus haemolyticus* group G, and *Enterococcus faecalis*. All isolates were susceptible to ciprofloxacin, which was therefore introduced

as treatment at a dose 20 mg/kg BW once a day (Cipronex 500 mg, tab., Zakłady Farmaceutyczne POLPHARMA SA), and subsequently replaced after several days with marbofloxacin at dose 2.5 mg/kg body weight (Marfloxin 80 mg tab., KRKA, Slovenia).

In July 2025, an abdominal ultrasound was performed, revealing splenomegaly with a four-fold enlargement of the spleen, along with multiple inflammatory foci within the hepatic and pancreatic parenchyma.

In early August 2025, the dog developed episodes of dyspnea, accompanied by the appearance of a mass in the region of the right forelimb. By mid-August, marked leukocytosis with neutrophilia was observed: WBC 45.6×10^3 [reference interval (RI): $6.0\text{--}12.0 \times 10^3$], NEU 33.28×10^3 (RI: $3.6\text{--}12.7 \times 10^3$), together with a concurrent decrease in erythrocytic parameters: RBC 4.1×10^6 (RI: $5.5\text{--}8.0 \times 10^6$), HGB 6.7 mmol/L (RI: 7.45–11.17 mmol/L), PCV 31% (RI: 37–55%). Serum biochemistry revealed particularly elevated hepatic enzyme activities: AST 64 U/L (RI: 3.0–45.0 U/L), ALT 234 U/L (RI: 5.0–60.0 U/L), ALP 821 U/L (RI: 5.0–155.0 U/L), and GGT 70 U/L (RI: 5.0–25.0 U/L).

One week later, the patient was referred to a partner clinic for an ACTH stimulation test, which again confirmed hypoadrenocorticism. During this visit, multiple subcutaneous lesions were noted over the presternum, the right axillary region, the right side of the neck, both inguinal regions, and extending from the hip and thigh to the tarsal joint of the left hind limb (Fig. 1). On palpation, these lesions were fluctuant, markedly oedematous, and painful, with overlying skin showing erythema and early signs of necrosis.

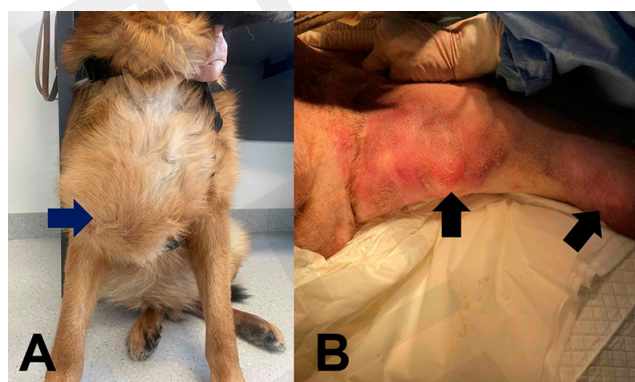


Figure 1. A large, fluctuant subcutaneous mass located in the pre-sternal region, extending cranially above the right thoracic limb – blue arrow (A); subcutaneous masses containing cystic material, are visible on the pelvic limb extending from the thigh to the tarsal joint – black arrows (B)

Material was collected from the mass located near the sternum for laboratory analysis. The contents were fluid and contained whitish oval structures measuring approximately 2–5 mm (Fig. 2). Despite intensive therapy and continuous veterinary care, the patient's condition continued to deteriorate. A surgical procedure was undertaken to evacuate the fluid accumulated within the subcutaneous tissue. Samples were collected and submitted to a commercial veterinary laboratory in Warsaw for microscopic and molecular examinations.

In addition, faecal samples were also submitted to the laboratory for parasitological examination. The faecal analysis did not reveal the presence of protozoan stages, nor any helminth eggs or larvae.

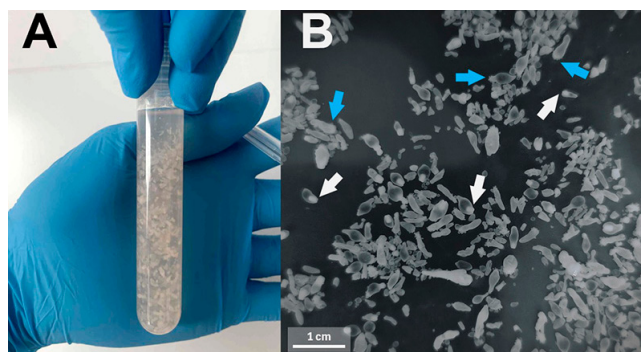


Figure 2. (A) contents aspirated from subcutaneous cysts from the pre-sternal region cysticerci. (B) larval stages of the cestode, with visible invaginations containing scolices (white arrows) and proliferative forms (blue arrows)

The patient did not survive to receive the results of the parasitological examination and initiation of targeted treatment.

Results of parasitological examination. Microscopic examination of the whitish structures revealed the presence of a cestode scolex containing two rows of hooks. The hooks were documented photographically and subjected to morphometric measurements (Fig. 3). Measurements of 10 large and 10 small hooks were performed. The total length of the small hooks ranged from 115.32 μm – 147.37 μm , with a mean length of 127.91 μm . In contrast, the large hooks measured between 163.19 μm – 177.13 μm , with a mean length of 169.34 μm .

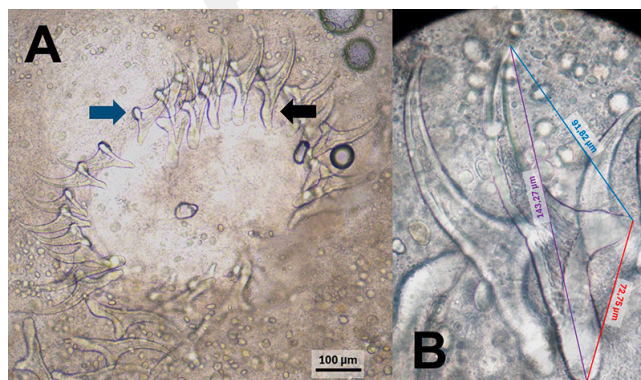


Figure 3. (A) a scolex of *T. crassiceps* cysticercus with two rows of hooks: small hooks (blue arrow) and large hooks (black arrow). (B) high-magnification of the small hooks: showing blade length (blue line), handle length (red line), and total hook length (violet line)

Numerous structures in the fluid were identified as larval stages of cestodes (cysticerci). Molecular analyses were performed to determine the species. Genomic DNA was extracted using the AX Sherlock kit (A&A Biotechnology, Gdańsk, Poland) according to the manufacturer's instructions. For amplification of an mt-CO1 gene fragment, primers JB3 (5'-TTTTTTGGGCATCCTGAGGTTTAT-3') and JB4.5 (5'-TAAAGAAAGAACATAATGAAAATG-3') were employed [17].

PCR conditions were as follows: initial denaturation at 95 °C for 3 min; 35 cycles of denaturation at 95 °C for 1 min, annealing at 45 °C for 1 min, and extension at 72 °C for 1 min; followed by a final extension at 72 °C for 10 min. Amplifications were performed in a MultiGene optiMAX thermocycler (Labnet International, Taoyuan, Taiwan). The

PCR products were separated on a 1.5% agarose gel, stained with Midori Green Advance DNA Stain (Nippon Genetics Europe GmbH, Dürren, Germany), and visualized using a UV transilluminator. A 446 bp fragment of the mt-CO1 gene from one cysticercus sample was sequenced. An external commercial provider (DNA Sequencing and Synthesis Laboratory – Oligo IBB, Warsaw, Poland) performed bi-directional PCR product sequencing using the Sanger dideoxy method. Sequence identity was assessed with BLAST algorithms against the databases of the National Centre for Biotechnology Information (<http://www.ncbi.nlm.nih.gov/>).

The nucleotide sequence showed 100% identity with *Taenia crassiceps* (Fig. 4). The sequence was deposited in GenBank® under Accession No. PX219651 (accessed on 25 August 2025).

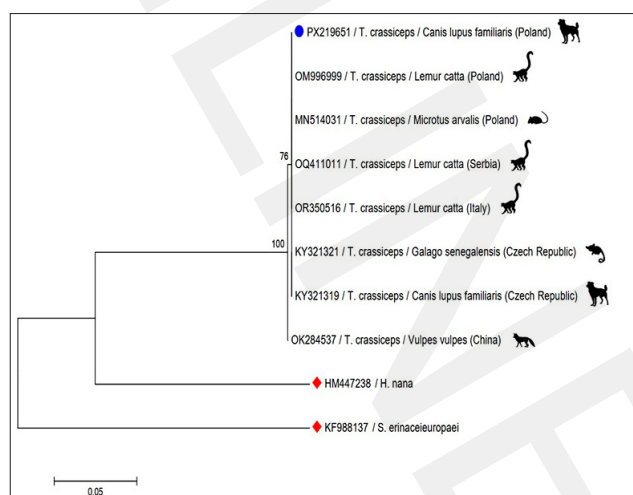


Figure 4. Phylogenetic relationships of *Taenia crassiceps* isolated from animals, generated using MEGA version 7 software. Analysis was performed using the neighbour-joining method based on nucleotide sequences of the mt-CO1 gene fragment. The evolutionary distances were computed using the Kimura 2-parameter method, and expressed as the number of base substitutions per site. All positions containing gaps and missing data were eliminated. Host names and corresponding GenBank accession numbers are provided. The sequences of *H. nana* and *S. erinaceieuropaei* were used as an outgroup

DISCUSSION

Taenia crassiceps is a cosmopolitan cestode, distributed throughout the Northern Hemisphere, with its occurrence frequently reported in both Europe and North America [1, 2, 5, 8, 16, 18]. Domestic dogs and wild canids, including red foxes (*Vulpes vulpes*) and wolves (*Canis lupus*), serve as the primary definitive hosts. By shedding infective eggs in their faeces, these hosts contribute to environmental contamination and thus maintain transmission cycles, providing a source of infection for intermediate and paratenic hosts [7, 8, 9].

The ring-tailed lemur (*Lemur catta*) is among the most susceptible non-human primate species. Subcutaneous infections with *T. crassiceps* have been documented in ring-tailed lemurs in several zoological gardens across Europe, including Croatia, Serbia, Italy, and Poland [16, 19, 20], as well as pleural infection reported in Italy and Bosnia and Herzegovina [18, 21]. Although the underlying immune mechanisms in lemurs remain poorly characterized, it has been suggested that their immune response may be less effective in restricting the rapid and proliferative growth

of cysticerci compared to natural rodent hosts. Additional research is warranted to better elucidate the pathogenic potential of this parasite and its highly invasive nature in lemurs [18].

T. crassiceps exhibit a remarkably broad range of intermediate hosts, including atypical ones like humans. This is supported by the report of a case of neurocysticercosis in a 71-year-old immunocompetent man who was presented with stroke-like symptoms [22].

In the current case, the dog becomes an atypical intermediate host, with *T. crassiceps* metacestodes proliferating within the subcutaneous tissue. Comparable cases of subcutaneous cysticercosis in dogs have been previously documented in the USA [1, 23], Germany, and the Czech Republic [15, 24]. Additionally, a case of pleural and peritoneal cysticercosis was described in a six-month-old female dog in the USA [14], as well as a pleural infection in an 18-month-old male Yorkshire Terrier in the Czech Republic [24].

The association between canine cysticercosis and underlying endocrine or immuno-suppressive conditions is particularly interesting. Two previously reported cases involved dogs with Cushing's syndrome [15], and one case with bilateral exophthalmos treated with long-term immuno-suppressive therapy using prednisolone and azathioprine [14].

Recent studies have demonstrated that *T. crassiceps* metacestodes can synthesize steroid hormones, including testosterone, from steroid precursors, and that androgens may play a role in parasite growth and proliferation. Moreover, experimental models have shown that *T. crassiceps* infection can alter the host's endocrine balance by modulating serum steroid hormone concentrations and reproductive behaviour in male mice [25]. These findings suggest that host-parasite interactions in *Taenia* infections are, at least in part, hormonally mediated. In this context, administering exogenous glucocorticoids, such as prednisone or prednisolone, may further influence the course of infection. Glucocorticoids exert strong immunosuppressive effects, which can impair the host's ability to control parasitic proliferation. At the same time, as steroid compounds, they may provide additional substrates or create favorable endocrine conditions that support parasite survival and development [26]. In the current case, the dog had been receiving long-term prednisolone therapy due to Addison's disease, which may have predisposed the patient to the establishment and progression of *T. crassiceps* cysticercosis. This observation is consistent with previously reported canine cases in which endocrine disorders or immuno-suppressive therapy were implicated as contributing factors.

No effective treatment protocol has been established in animals. Studies conducted in a murine model demonstrated that combined therapy with albendazole and ivermectin was the most effective in inducing *T. solium* cysticercus degeneration and suppressing the host inflammatory response [27].

CONCLUSIONS

Taenia crassiceps infections can affect domestic dogs, wild canids, rodents, and a broad and expanding range of atypical and accidental hosts, including humans. This highlights the need for coordinated efforts between veterinary and medical professionals to prevent potential zoonotic transmission and to raise awareness of this emerging parasitic threat.

Early detection is critical because infections are relatively frequent among animals in Europe, including Poland. Cytological, histopathological, or imaging examinations of easily accessible subcutaneous lesions, should be considered, particularly in animals receiving immuno-suppressive therapy or with underlying endocrine disorders. Such proactive diagnostic approaches may allow timely intervention, reduce parasite proliferation, and mitigate the risk of atypical or severe infections.

Adopting a One Health perspective, which integrates animal and human health considerations, is especially relevant in the context of *T. crassiceps*. Enhanced surveillance, systematic reporting of unusual cases, and preventive measures in both domestic and wildlife populations are essential to limit the spread of this zoonotic cestode and to safeguard public health.

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