Medical Mycology Case Reports 10 (2015) 29–33

Contents lists available at ScienceDirect

Medical Mycology Case Reports

journal homepage: www.elsevier.com/locate/mmcr

Disseminated *Chrysosporium* infection in a German shepherd dog

Emily Cook, Erika Meler*, Katrina Garrett, Hanna Long, King Mak, Carol Stephens, Ann Thompson

The University of Queensland. School of Veterinary Sciences, Veterinary Medical Centre, Gatton campus, Gatton 4343, QLD, Australia

ARTICLE INFO

Article history:
Received 21 January 2016
Accepted 22 January 2016
Available online 23 January 2016

Keywords:
Canine
Chrysosporium
Disseminated fungal infection
German shepherd dog

ABSTRACT

Disseminated *Chrysosporium* spp. infection was diagnosed in a German shepherd dog based on a positive fungal culture and cytological findings of intralesional fungi associated with granulomatous splenitis and neutrophilic lymphadenitis. The clinical presentation that could mimic a multicentric lymphoma, including markedly enlarged lymph nodes and a very abnormal splenic appearance on ultrasound makes this case even more atypical. The patient showed rapid clinical improvement on oral posaconazole and remains clinically stable ten months after diagnosis.

1. Introduction

German shepherd dogs (GSD), particularly young to middle-aged females, are well-known for their predisposition to disseminated *Aspergillus* spp. infection [1,2]. The familial tendency is further supported in this breed with the development of disseminated fungal disease in close relatives [3]. A genetic predisposition leading to a deficient immune response to fungi, possibly related to immunoglobulin A (IgA) deficiency and/or dysfunction has been reported in this breed [4,5]. However, the aetiology is likely multifactorial, involving dysfunction of both humoral and cell mediated immunity against *Aspergillus* spp. infection [3,5,6]. Literature tends to suggest that GSD are also prone to other disseminated fungal infections caused by species such as *Penicillium* spp., *Paecilomyces* spp., *Pseudallescheria boydii, Scytalidium* spp., *Scedosporium prolificans, and Chrysosporium* spp., most of which would be classified as opportunistic [1,7,8].

Infection with *Chrysosporium* spp. is uncommon in both human and veterinary literature, being mostly described in reptiles. Information on clinical management is then quite variable and depends on the species involved, the extent of the disease (focal vs systemic involvement), and drug availability. Infection of a dog by *Chrysosporium* spp. has only be reported three times to date.

The difference in clinical presentation, ultrasound findings and long term treatment with posaconazole in the case described herein adds valuable information about the diversity of signs that *Chrysosporium* spp. infection can cause in dog and brings a new light on possible treatment. Indeed, to the author's knowledge, the successful use of posaconazole for *Chrysosporium* spp. infection has not yet been reported in humans or animals, making the description of this case unique.

2. Case

A 3 year old, female speyed GSD, weighing 28 kg, presented for assessment of an acute history of lethargy, anorexia, pyrexia, and pelvic limb ataxia. The dog had been reported to be clinically well five days prior to referral, then rapidly deteriorated.

On initial presentation to the referring veterinarian (day 0), the dog was lethargic, hyperthermic (rectal temperature of 40.5 °C) and ataxic in both pelvic limbs. The dog was painful on palpation of the thoracolumbar region and caudal ventral abdomen, and conscious proprioception was delayed in both pelvic limbs. In-house haematology and biochemistry were performed and main abnormalities included a mild neutrophilic leucocytosis, hyperglobulinaemia, and hypoalbuminaemia (Table 1). Spinal radiographs were unremarkable. The dog was treated with intravenous replacement crystalloid fluid (Hartman's®) and administered methadone. The following morning the dog had developed profuse watery diarrhoea, pigmenturia, and remained hyperthermic (40.4 °C). Worsening paresis of the pelvic limbs was noted. Referral to a specialty practice for further evaluation was recommended.

On initial examination at the University of Queensland Veterinary Medical Centre (day 1), the dog was found to be in poor body condition (score of 3/9). Upon physical examination, lethargy, tachypnoea and persistent hyperthermia were noticed (40.1 °C). Cardiac and pulmonary auscultations were normal. Popliteal and

* Corresponding author.
E-mail address: e.meler@uq.edu.au (E. Meler).

http://dx.doi.org/10.1016/j.mmcr.2016.01.002

Table 1
Summary of major haematology, serum biochemistry and urinalysis findings.

Haematology	Units	Reference interval	DAY 0	DAY 1	DAY 14	DAY 24	DAY 36
PCV/HCT	%	37–55	41	40	27	36	39
Reticulocytes	$X10^9$/L	< 60	N/A	N/A	132.33	N/A	N/A
White cell count	$X10^9$/L	6.0–17.0	21.9	18.1	35.6	N/A	26.1
Granulocytes	$X10^9$/L	3.0–12.0	17.6	15.3	N/A	N/A	N/A
Neutrophils	$X10^9$/L	3.0–11.5	N/A	N/A	21.7	N/A	18.3
Eosinophils	$X10^9$/L	0.1–1.2	N/A	N/A	2.5	N/A	0.8
Monocytes	$X10^9$/L	0.2–1.5	1.3	0.6	0.7	N/A	1.3
Lymphocytes	$X10^9$/L	1.0–4.8	2.9	2.2	10.7	N/A	5.7
Serum biochemistry							
Albumin	g/L	25–44	22	19	19	23	23
Globulin	g/L	23–52	64	56	34	42	46
ALP	U/L	20–150	191	N/A	187	190	207
ALT	U/L	10–118	63	N/A	51	47	37
AST	U/L	13–37	N/A	127	85	42	40
Urinalysis							
Collection method			**Voided**	**Cystocentesis**	**Cystocentesis**	**N/A**	**Cystocentesis**
USG		1.001–1.065	> 1.060	1.032	1.028	N/A	1.028
pH	pH	5.5–8.5	7.0	7.5	6.5	N/A	6.0
Sediment:							
-Casts (granular)	/hpf	None	none	1	1–2	N/A	none
-RBC	/hpf	< 5	5–20	1–2	negative	N/A	40-50
-WBC	/hpf	< 5	< 5	< 1	< 1	N/A	< 1
-Fungal hyphae	/hpf	None	N/A	none	none	N/A	none
UPC		< 0.5	N/A	3.0	1.4	N/A	0.3

inguinal lymph nodes were severely enlarged whereas submandibular and pre-scapular lymph nodes remained of normal size. A 3-cm mobile, firm, subcutaneous mass was palpated over the right lateral mid-thorax. Abdominal palpation was painful and confirmed the presence of significant caudal abdominal organomegaly. A T3–L3 spinal lesion was suspected based on the absent proprioception and paresis of the pelvic limbs and severe pain on direct palpation of T2–L2. Mentation was appropriate, cranial nerve examination was normal and fundic examination was unremarkable.

Thoracic radiographs showed no significant abnormalities. Abdominal ultrasound demonstrated severe enlargement of medial iliac lymph nodes. The spleen was diffusely mottled in echotexture with multiple faint hypoechoic nodules up to 13 mm in size throughout the parenchyma. Fine needle aspirates of the spleen, and both medial iliac lymph nodes were performed and Wright's Giemsa stained direct smears were examined by light microscopy. A cytological diagnosis of granulomatous splenitis and neutrophilic lymphadenitis associated with the presence of macrophages containing branching, septate fungal hyphae was made, consistent with a multi-organ fungal infection (Fig. 1). Urinalysis showed mild haematuria, few granular casts and severe proteinuria (Table 1). Fungal hyphae were not identified on urine sediment examination.

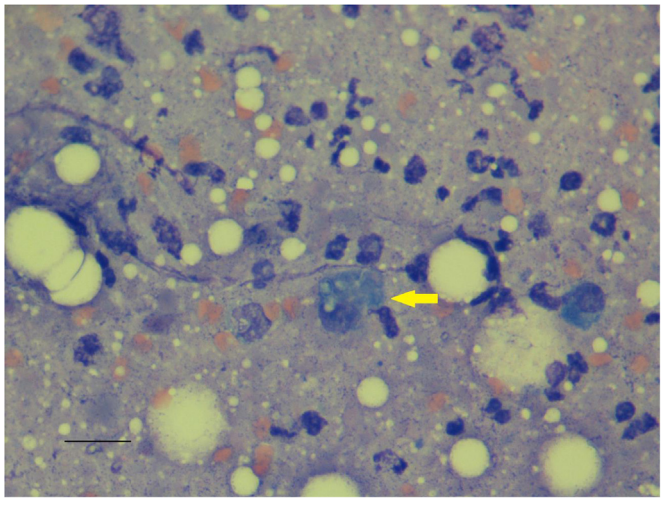

Fig. 1. Microscopic image of lymph node aspirate. Arrow: macrophage containing intracytoplasmic, septate fungal hyphae. Scale bar = 50 μm.

The dog was admitted to hospital, maintained on intravenous crystalloids (Hartman's®) and administered multimodal analgesia based on a combination of methadone, gabapentin and fentanyl. The following day (day 2), the dog appeared more comfortable but neurological examination was unchanged, and rectal temperatures remained persistently greater than 40.0 °C.

A tissue biopsy of the right subcutaneous thoracic mass was performed using a punch biopsy (6 mm) and submitted for fungal culture. The sample was cultured on Sabouraud's dextrose agar (Thermo Fisher, United Kingdom) and incubated at 30 °C. Fungal growth was detected after five days incubation. After six days incubation, thallus was flat, white and cottony in texture. The reverse was cream in colour. Microscopic examination revealed hyaline, septate hyphae. Single celled conidia were borne on the hyphae and in short chains at the ends of hyphae. Conidia were hyaline, smooth-walled and pyriform, with a broad base. Conidia were broader than the hyphae and attached by short pedicels. Those conidia which had become detached from the hyphae carried a distinct, annular scar, the remnant of the hyphal wall. No macroconidia were observed. No growth was obtained during subsequent incubation on Sabouraud's dextrose agar at 37 °C. On the basis of the above colonial and microscopic features, the isolate was identified as *Chrysosporium* spp. Speciation of the isolate and antifungal susceptibility testing unfortunately could not be performed due to organism death in the laboratory.

Based on cytology of lymph nodes and spleen, on the culture of the subcutaneous mass, and in association with compatible clinical signs, disseminated fungal infection was diagnosed. Discospondylitis was suspected as a cause of the neurological signs but could not be identified on plain radiography. Advanced imaging, including magnetic resonance imaging (MRI) and computed tomography (CT) were offered to investigate for the possibility of a fungal granuloma, but declined by the owners for financial reasons. The dog was administered one dose of dexamethasone (0.075 mg/kg IV) to control inflammation and posaconazole

(10 mg/kg PO every 24 h) was commenced concurrently.

On day 3, the dog developed severe oedema over the ventral abdomen, and pelvic limbs. Hypotheses considered included decreased oncotic pressure, systemic infection related vasculitis, lymphatic/venous obstruction from severe lymphadenomegaly, or a combination of all. Given that serial plasma albumin concentrations remained at all-times greater than 21 g/L (reference interval: 23–40 g/L), and due to the reported severe enlargement of iliac medial lymph nodes, it was felt that the lymphadenomegaly played a larger role in oedema development.

The dog was hospitalised until day 9 and showed overall a gradual improvement in demeanour, neurologic deficits and feces consistency; however, body temperature continued to wax and wane between 38.8 °C and 40.6 °C and oedema remained prominent. At discharge (day 9), the following medications were prescribed: posaconazole (10 mg/kg PO every 24 h), gabapentin (10.7 mg/kg PO every 8 h), omeprazole (0.66 mg/kg PO every 24 h), metronidazole (10 mg/kg PO every 12 h), and a fentanyl patch (100 µg/h patch).

The patient returned for follow up at day 14, 24 and 36 with continued improvement in demeanour and neurological deficits at each visit. On day 14, the dose of posaconazole was decreased from 10 mg/kg PO every 24 h to 8.5 mg/kg PO every 24 h due to financial constraints expressed by the owner. On day 36, the dog was normothermic (38.8 °C). Popliteal and inguinal lymph node size had improved dramatically but remained significantly larger than normal. Right thoracic mass had also decreased in size (approximately 1 cm in diameter). Haematology showed further improvement in haematocrit and resolution of the marked leukocytosis. Serum biochemistry showed improvement in hypoalbuminaemia (Table 1). There was resolution of proteinuria, hematuria and absence of fungal hyphae or urinary casts on sediment examination (Table 1). Spinal radiographs showed no evidence of discospondylitis. However, development of a lesion (approximately 3 × 5 cm) on the wing of the ileum of the right pelvis was identified. It appeared as moth-eaten lysis with a small 3 cm length minimally displaced pathological fracture in the proximal tip of the ileal wing was noted. On abdominal ultrasound, splenic parenchyma was significantly different from initial examination with a diffusely hypoechoic parenchyma and multiple pinpoint hyperechoic foci giving a "snowstorm appearance" leading to better demarcation of the hypoechoic nodules previously described (Fig. 2).

At this stage the owners expressed concerns regarding the ongoing cost of posaconazole, and this medication was changed to itraconazole (7.4 mg/kg PO every 24 h) for long-term treatment.

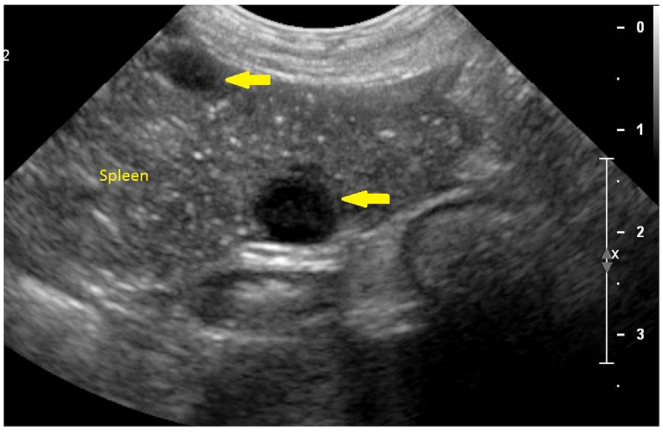

Fig. 2. Ultrasound image of the spleen on day 36. Arrows: well-demarcated hypoechoic splenic nodules underlined by the multiple hyperechoic foci within the splenic parenchyma.

At the time of writing, approximately ten months after presentation, the dog remains clinically stable, with minimal lymph node enlargement, complete resolution of peripheral oedema and neurological signs.

3. Discussion

To the authors' knowledge, this case is only the second reported in the veterinary literature of disseminated *Chrysosporium* spp. infection in a dog. Interestingly, the first published case was reported as part of a case series on disseminated fungal disease in dogs that also originated from Queensland, Australia [1].

The genus *Chrysosporium* is in the order Onygenales to which the organisms Blastomyces, Coccidioides, Histoplasma and Dermatophytes belong [11]. *Chrysosporium* spp. are saprobic species that live on the remains of hair and feathers in the soil and are involved in the breakdown of keratinous substrates [11]. These fungi are rarely reported as human or veterinary pathogens. In fact, *Chrysosporium* spp. would normally be encountered as a contaminant of cutaneous or respiratory specimens [12].

Infection with *Chrysosporium* spp. in the veterinary literature is most commonly documented in reptiles, including lizards, snakes, with one account in saltwater crocodiles. *Chrysosporium anamorph of Nannizziopsis vriesii* (CANV) tends to be the most common species involved and lesions vary from superficial dermatomycoses, to systemic mycoses. Many cases are diagnosed on postmortem examination [11].

Both local and invasive *Chrysosporium* infections are very rare in the human literature, with only thirteen cases reported to date, and affect almost exclusively immunocompromised people [12]. Organs affected include lungs, sinuses, brain, bone, heart valve, pericardium, cornea, and skin in order of decreasing likeliness of involvement [12–14]. The clinical course of disseminated *Chrysosporium* infection in people can be severe to fatal; however, six of eight cases of invasive infection have survived with aggressive management [12,14].

Infection with *Chrysosporium* spp. is extremely uncommon in domestic dogs with definitive identification in only three cases. One case report described keratomycosis in a dog (Pekingese cross) that had concurrent diabetes mellitus and was treated with topical prednisolone before diagnosis of fungal keratitis [10]. In the second case, the fungus was identified from a pathological skin lesion in a dog (dwarf pincher); however, the exact role of this fungus was not determined in this case [9]. The third case, and only one of disseminated *Chrysosporium* infection was also reported in a GSD [1]. This dog was diagnosed with discospondylitis, osteomyelitis of the radius and irregular bone formation on the accessory carpal bone and was treated with itraconazole (10 mg/kg/day PO for 6 weeks, then 17 mg/kg/day PO for another 5 weeks). Control over disease progression was unclear as there was some radiographic evidence of healing of the radial osteomyelitis, but simultaneous development of new vertebral lesions after 11 weeks of therapy. Antifungal treatment was discontinued at this point, and the patient lived for 12 months before it was euthanased for progressive paresis, lameness and signs of pain [1]. The long survival in this patient after discontinuing anti-fungal therapy may reflect poor pathogenicity of this species.

Disseminated mycoses in domestic animals and in people is often associated with immunosuppression, whether this be due to inherited immunodeficiency, administration of medications, or presence of concurrent illnesses that suppress the immune system [1,2,12]. Being keratinophilic, *Chrysosporium* spp. has been commonly found on the coats of healthy dogs in the absence skin lesions [15]. The true pathogenicity of this species of fungi is unknown, but appears to be quite weak [16]. Therefore, an

underlying immune deficiency is likely to explain the dissemination of the fungus in this case. GSD, in particular females, are overrepresented for developing disseminated mycoses, especially Aspergillosis [1,2]. An inherited immune deficiency relating to abnormalities in IgA regulation and function is suspected [5,6] with abnormally low IgA serum levels commonly reported in this breed [4]. However, not all GSD with systemic fungal disease have decreased IgA levels [3]; therefore, the cause appears to be more complex than an isolated IgA deficiency. Some authors have suggested the involvement of cell mediated dysfunction in these cases but this remains to be confirmed by further studies [6].

Clinical signs associated with systemic mycoses can be varied, and are dependent on the organs involved. In larger retrospective studies of dogs with systemic mycoses, initial clinical signs are often attributed to the musculoskeletal and neurologic system. Ataxia, neck and back pain, but also less specific signs such as anorexia and weight loss are commonly cited [1,2]. Watt et al. found 8/10 dogs with systemic mycoses suffered from back pain, and all had evidence of discospondylitis on plain radiographs [1]. Interestingly, in a larger case series of 30 dogs with systemic aspergillosis, only 5/30 dogs had spinal pain; however, 16/30 had radiographic evidence of discospondylitis [2]. Our patient did have neurological findings consistent with a T3-L3 spinal lesion and back pain; however, no evidence of discospondylitis was evident on initial spinal radiographs, nor on the radiographs taken five weeks after presentation. A similar presentation in a female GSD with disseminated *Scytalidium* spp. infection was described with the presence of a soft tissue mass on CT, located along the left lateral aspect of the vertebral canal, through the body of C5 and displacing the spinal cord [7]. Although advanced imaging could not be performed in our case, the resolution of the ataxia and proprioceptive deficits with antifungal treatment was more suspicious for a fungal granuloma associated with the spinal cord than discospondylitis.

Lymphadenomegaly of the popliteal, medial iliac, and inguinal lymph nodes was particularly profound in this case with associated lymphoedema. Peripheral lymphadenomegaly was not a greatly common finding in one case series, being reported in only 17% of cases [2]. In combination with the marked peripheral lymphocytosis and the pronounced splenic changes, lymphoma was an important differential diagnosis in this patient.

Presence of hypoechoic nodules and hypoechoic lacy regions in the spleen have been previously reported in dogs with systemic aspergillosis [2]. The appearance of the spleen in this case of *Chrysosporium* spp. infection has a similar unusual but non-specific sonographic appearance. Similarly, in humans, the “sonographic snowstorm” appearance of highly echogenic foci in the spleen is seen with a variety of different fungal organisms, and is thought to be due to extensive fibrosis or fibrinous exudates within the parenchyma [17].

Aggressive, destructive bony lesions have been reported in systemic aspergillosis and other disseminated fungal infections [1,2]. While discospondylitis was not a feature in this case, an aggressive destructive process was identified in the ileum of the right pelvis at last radiographic recheck, but was not associated with clinical signs. In mature dogs with discospondylitis, radiographic lesions can appear progressively worse for 3–9 weeks despite successful treatment [18]. Therefore, the development of the bony lesion in our patient would likely suggest a discrepancy between actual disease and radiographic resolution, rather than treatment failure, given the significant improvement in other organs involved over the same time period.

There is little data in both the veterinary and human literature regarding successful treatment of disseminated *Chrysosporium* spp. Interestingly, the only other canine case of invasive *Chrysosporium* spp. infection was treated with itraconazole, a first-generation triazole antifungal. Some bony lesions were reported to have improved during the treatment period; however, new ones developed over the same time frame and itraconazole was discontinued after only 11 weeks of treatment [1]. Therefore, long term control over the lesions with this particular antifungal could not be accurately assessed. Although the authors of this article suspected poor efficacy of this drug, it remains possible that itraconazole had a favourable initial impact on disease progression as the dog lived for a protracted time in the absence of antifungal treatment before relapsing. Response to itraconazole, appears to be strain-dependent in people, with most cases requiring more aggressive therapy with amphotericin B or voriconazole [12–14]. Successful treatment of *Chrysosporium* spp. infection in reptiles is limited, as most are diagnosed at necropsy; however, itraconazole, ketoconazole and voriconazole are suggested [11].

Posaconazole is a newer second generation triazole antifungal derived from itraconazole [19]. Posaconazole has not been reported to have been used before in human or canine patients with *Chrysosporium* spp. infection. In a recent case of invasive pulmonary infection with *Chrysosporium articulatum* in a person, minimum inhibitory concentration (MIC) was determined for this particular organism against posaconazole, and showed no evidence of resistance [14]. In our case, this particular anti-fungal was chosen before species identification was made based on its excellent in vitro activity against *Aspergillus* spp. [19], which was presumed to be the most likely causative agent in this case pending final fungal cultures. Clinical response was seen during the first week of administration leading to near resolution of the patient lymphadenomegaly, right thoracic mass, hematuria and neurological signs by week five. We therefore believe, in the absence of antifungal susceptibility testing, that posaconazole was an appropriate choice for this case of severe, systemic infection with *Chrysosporium* spp. The transition of this dog from posaconazole to itraconazole was based on financial rather than medical reasons.

In conclusion, we report an unusual case of disseminated *Chrysosporium* spp. species infection in a GSD. This case demonstrates that although this fungus is an uncommon pathogen, it can be associated with severe morbidity, and reminds clinicians that non-*Aspergillus* spp. fungi can also cause significant disease in GSD. This dog responded well to therapy with posaconazole and itraconazole. Although long term follow-up and associated imaging rechecks will be important for the objective evaluation of outcome with this treatment, at the time of writing, the dog is still alive and doing clinically well ten months after diagnosis.

Conflict of interest

None of authors declare conflict of interest pertaining to this case report.

References

[1] P.R. Watt, G.M. Robins, A.M. Galloway, D.A. O'Boyle, Disseminated opportunistic fungal disease in dogs: 10 cases (1982–1990), J. Am. Vet. Med. Assoc. 207 (1) (1995) 67–70.
[2] R.M. Schultz, E.G. Johnson, E.R. Wisner, N.A. Brown, B.A. Byrne, J.E. Sykes, Clinicopathologic and diagnostic imaging characteristics of systemic aspergillosis in 30 dogs, J. Vet. Int. Med. 22 (4) (2008) 851–859.
[3] M.J. Day, W.J. Penhale, C.E. Eger, S.E. Shaw, M.J. Kabay, W.F. Robinson, et al., Disseminated aspergillosis in dogs, Aust. Vet. J. 63 (2) (1986) 55–59.
[4] M. Olsson, M. Frankowiack, K. Tengvall, P. Roosje, T. Fall, E. Ivansson, et al., The dog as a genetic model for immunoglobulin A (IgA) deficiency: identification of several breeds with low serum IgA concentrations, Vet. Immunol. Immunopathol. 160 (3–4) (2014) 255–259.
[5] M.J. Day, W.J. Penhale, An immunohistochemical study of canine disseminated aspergillosis, Aust. Vet. J. 68 (12) (1991) 383–386.
[6] J. Perez, E. Mozos, F.C. de Lara, J. Paniagua, M.J. Day, Disseminated aspergillosis

in a dog: an immunohistochemical study, J. Comp. Pathol. 115 (2) (1996) 191–196.
[7] A.E. Dunlap, A.K. Swinford, K.L. Wells, Disseminated Scytalidium infection in a German shepherd dog, Med. Mycol. Case Rep. 7 (2015) 20–22.
[8] S.M. Haynes, P.J. Hodge, D. Tyrrell, L.A. Abraham, Disseminated Scedosporium prolificans infection in a German shepherd dog, Aust. Vet J. 90 (1–2) (2012) 34–38.
[9] M.V.Gd Hajsig, V. Sertic, T. Naglic, Chrysosporium evolceanui from pathologically changed dog skin, Vet. Arch. 44 (1974) 209–211.
[10] E.M. Scott, R.T. Carter, Canine keratomycosis in 11 dogs: a case series (2000–2011), J. Am. Anim. Hosp. Assoc. 50 (2) (2014) 112–118.
[11] F.J. Cabanes, D.A. Sutton, J. Guarro, Chrysosporium-related fungi and reptiles: a fatal attraction, PLoS Pathog. 10 (10) (2014) e1004367.
[12] G.M. Anstead, D.A. Sutton, J.R. Graybill, Adiaspiromycosis causing respiratory failure and a review of human infections due to Emmonsia and Chrysosporium spp, J. Clin. Microbiol. 50 (4) (2012) 1346–1354.
[13] P.M. Kamath, V. Prasad, V.S. Shenoy, A. Mukundan, S. Shenoy, Chrysosporium: an uncommon fungus in chronic rhinosinusitis, J. Clin. Diagn. Res. 9 (3) (2015) Md01–2.
[14] C. Suankratay, O. Dhissayakamol, N. Uaprasert, A. Chindamporn, Invasive pulmonary infection caused by Chrysosporium articulatum: the first case report, Mycoses 58 (1) (2015) 1–3.
[15] M.F. Simpanya, M. Baxter, Isolation of fungi from the pelage of cats and dogs using the hairbrush technique, Mycopathologia 134 (3) (1996) 129–133.
[16] D. Chabasse, L. De Gentile, J.P. Bouchara, Pathogenicity of some Chrysosporium species isolated in France, Mycopathologia 106 (3) (1989) 171–177.
[17] M.A. Keane, C. Finlayson, A.E. Joseph, A histological basis for the"sonographic snowstorm' in opportunistic infection of the liver and spleen, Clin. Radiol. 50 (4) (1995) 220–222.
[18] M.H. Shamir, N. Tavor, T. Aizenberg, Radiographic findings during recovery from discospondylitis, Vet. Radiol. Ultrasound 42 (6) (2001) 496–503.
[19] D.S. Foy, L.A. Trepanier, Antifungal treatment of small animal veterinary patients, Vet. Clin. N. Am. Small Anim. Pract. 40 (6) (2010) 1171–1188.