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Case report/Fallbericht

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Multidrug- and methicillin resistant *Staphylococcus pseudintermedius* as a cause of canine pyoderma: a case report

Methicillin- und multiresistente Staphylococcus pseudintermedius als Ursache einer caninen Pyodermie – ein Fallbericht

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Summary

A case of a dog with a long-term inflammatory skin disorder due to infection with methicillin-resistant *Staphylococcus pseudintermedius* (MRSP) is described.
After initial diagnostics of MRSP, follow-up swabs of the dog (nose, skin) were taken twice after four and seven weeks. MRSP was constantly isolated from the skin and once from the nose. Since infected humans might be a source of re-infection, the owners of the dog were screened (nasal) three times during their pet's therapy. Thereby, the male owner was found to be colonized with MRSP once in the first sampling round.
Comparative typing of all MRSP-isolates by pulsed-field gel electrophoresis (PFGE), SCC*mec* typing, multilocus sequence typing (MLST), *spa* typing, PCR-detection of the leukotoxin encoding operon (LukI) and the *Staphylococcus intermedius*-exfoliative toxin (SIET) as well as antimicrobial resistance profiling by broth microdilution revealed that all five MRSP isolates from the dog and the single isolate from the owner were indistinguishable by any of the applied methods. All isolates were assigned to a certain strain, a multidrug-resistant MRSP belonging to sequence type (ST) 71, *spa* type (t)05, harbouring SCC*mec*III as well as the genes encoding LukI and SIET.
In this case, a number of reasons might have contributed to therapy failure and re-infection, respectively (e. g. contact to other MRSP-colonized dogs, contact to MRSP-colonized humans, refusal to clip the dog`s fur). In addition, MRSP-contaminated objects or surfaces in the household, which were difficult to disinfect or simply not considered as a potential source of MRSP, might have served as a source of re-infection.
These results envision the possibility of a dog-to-human transmission of MRSP and the relevance of this aspect as a potential source of re-infection in cases of bacterial-supported long-term skin disorders in canine patients. First cases of MRSP infections in humans have been described only recently. However, the general pathogenic potential of multidrug resistant MRSP in humans is unknown so far and needs further investigation.

Keywords: methicillin-resistant *Staphylococcus pseudintermedius*, MRSP, pyoderma, dog-to-human transmission

Zusammenfassung

Es wird der Fall eines Hundes mit chronischer Hautentzündung beschrieben, die durch eine Infektion mit Methicillin-resistenten *Staphylococcus pseudintermedius* (MRSP) hervorgerufen wurde. Nach dem ersten mikrobiologischen Nachweis von MRSP in einer Hauttupferprobe wurde noch zweimal (nach vier und sieben Wochen) sowohl die Haut als auch die Hundenase mittels Abstrich untersucht. Von der Haut konnte MRSP konstant isoliert werden, während von den beiden Nasentupfern nur einer positiv war. Da unter anderem auch kolonisierte Besitzer eine mögliche Quelle für erneute Infektionen sein könnten, wurden beide Besitzer insgesamt dreimal im Laufe der Therapie ihres Tieres nasal auf MRSP

untersucht. In der ersten Tupferprobe des männlichen Besitzers konnte MRSP nachgewiesen werden.
Die MRSP-Isolate wurden durch Pulsfeld-Gelelektrophorese, SCC*mec*-Typisierung, Multilokus Sequenztypisierung, *spa*-Typisierung sowie PCR zum Nachweis der Gene für Leukotoxin (LukI) und das *Staphylococcus intermedius*-exfoliative Toxin (SIET) charakterisiert. Des Weiteren wurde für die untersuchten Isolate ein Resistenzprofil für nicht-ß-Lactam-Antiinfektiva erstellt. Mit den vorgenannten Typisierungsmethoden konnten identische Ergebnisse für alle fünf caninen sowie für das eine humane MRSP-Isolat ermittelt werden, sodass alle Isolate dem gleichen Stamm zuzuordnen waren: ein multiresistenter MRSP vom Sequenztyp (ST) 71, SCC*mec*III, *spa* Typ (t)05), der sich zudem in der PCR positiv für die LukI- und SIET-kodierenden Gene zeigte.
Grundsätzlich kommen für ein Therapieversagen bzw. für eine Reinfektion zahlreiche Ursachen in Betracht, z. B. der Kontakt zu asymptomatisch besiedelten anderen Hunden oder Menschen, unzureichend desinfizierten oder nur schwer desinfizierbaren Gegenständen oder Flächen im häuslichen Bereich sowie die nicht durchgeführte Schur des Hundes.
Dennoch deuten diese Ergebnisse auf eine grundsätzlich mögliche Übertragbarkeit von MRSP zwischen Hund und Mensch hin und lassen Raum für eine Diskussion über die Relevanz von Besitzern als potentielle Reinfektionsquelle für Tiere mit bakteriell bedingten chronischen Hautentzündungen.
Darüber hinaus wurden erst kürzlich MRSP-Infektionen beim Menschen beschrieben, jedoch ist das pathogene Potential von multiresistenten MRSP für Menschen bislang unbekannt.

Schlüsselwörter: Methicillin-resistente *Staphylococcus pseudintermedius*, MRSP, Pyodermie, Hund-zu-Mensch-Übertragung

Introduction

Canine pyoderma is the most common type of bacterial skin infection in the dog and until recently *Staphylococcus intermedius* was thought to be responsible for the majority of these infections. In 2008, sequencing of the housekeeping genes *pta* and *cpn*60 led to reclassification of the *S. intermedius*-group (SIG) into three distinct species, namely *S. intermedius*, *S. delphini* and *S. pseudintermedius* (Bannöhr et al., 2007). In addition, phenotypical differences between these individual species have been published by different authors (Devriese et al., 2005; Sasaki et al., 2007b). In summary, *S. pseudintermedius* seems to be the major pathogen of the SIG among canine pyoderma patients, rather than *S. intermedius* (Bannöhr et al., 2007).

In the last few years, increasing rates of methicillin-resistant *S. pseudintermedius* (MRSP) in small animals and horses have been reported (Loeffler et al., 2005; Bannöhr et al., 2007; Bemis et al., 2009; Black et al., 2009; Moodley et al., 2009; Ruscher et al., 2009; Ruscher et al., 2010). Furthermore, the majority of MRSP isolates were also frequently characterized by multidrug resistance against several other antimicrobial classes (Ruscher et al., 2009; Bemis et al., 2009).

As recently indicated by Cohn and Middleton, infections with methicillin-resistant *staphylococci* may be difficult to treat and predispose to increased morbidity and mortality in affected veterinary patients (Cohn and Middleton, 2010).

Although SIG have been found in medical samples occasionally (Tanner et al., 2000; Guardabassi et al., 2004a), reports about MRSP colonization or infection in humans are so far rare.

However, some cases of human infections with *S. pseudintermedius* have been reported, possibly indicating a pathogenic potential of this staphylococcal species for human hosts (Van Hoovels et al., 2006; Boost et al., 2009; Hanselman et al., 2009). Very recently, Chuang and colleagues reported a case of catheter-related bacteraemia caused by *S. pseudintermedius* in a child exposed to a dog (Chuang et al., 2010). In 2007, a person working in a veterinary university clinic in Tokyo (Japan), who was nasally colonized with the same MRSP strain which was isolated from canine patients, was identified (Sasaki et al., 2007a), indicating a possible MRSP transmission between dogs and humans in a clinical setting. Another study was applied to determine the zoonotic risk for dog owners whose animals suffer from deep pyoderma due to methicillin-resistant staphylococcal species. As a result, two MRSP colonized persons seemed to harbour MRSP strains similar to those isolated from their dogs (Frank et al., 2009). A case of postoperative sinus infection caused by methicillin-resistant SIG in a 28-year-old woman, and cultures from the patient`s dog, revealed colonization of the dog by an indistinguishable bacterial strain (Kempker et al., 2009).

The aim of this case report was to review the different aspects of deep pyoderma caused by MRSP in a canine patient with respect to a transiently colonized owner.

Case presentation

An eleven-year-old, male Airedale terrier with a seven month history of pruritus and inflammatory skin disease was referred to the Small Animal Clinic of the Freie Universität Berlin. The dog had been pre-treated with antibiotics (cefalexin), cortisone spray and enilconazole washings. On the day of admission, a clinical examination revealed a generalized pruritic dermatitis with epidermal col-

larettes and focal crusty lesions. Superficial and deep skin scrapings as well as fungal cultures were negative. Numerous skin biopsies were taken; histopathological examination revealed superficial perivascular dermatitis with infiltration of plasma cells, neutrophils and mast cells indicating a superficial pyoderma and possibly an underlying allergic component (Dermatopathology Unit, Institute of Animal Pathology, University of Bern). Except for a mild hyperproteinemia, parameters of hematology and clinical biochemistry were in the reference range. Based on endocrinological testing, hypothyroidism and hyperadrenocortcism were excluded.

Microbiological investigation of a cotton swab taken from a diseased area of the skin revealed an infection with a multidrug-resistant MRSP (Tab. 1). After this initial diagnosis of MRSP, the patient was treated with whole body washes with 2% chlorhexidine/2% miconazole shampoo every other day, selamectin (once, to exclude ectoparasites), n-3 fatty acid supplementation and antihistamines (hydroxyzine) for six weeks.

Within six weeks of treatment, the skin condition improved slightly, the dog was less pruritic. Nonetheless, a further skin swab and a nasal sample of the patient taken on day 36 turned out to be MRSP-positive. Therefore, a 2% fusidic acid topical cream applied twice daily was prescribed for the affected skin areas. Unfortunately the owners refused full-body clipping. After deterioration of clinical signs (day 48), amikacin (10 mg/kg i. v., twice daily for five days) was administered. This therapy resulted only in a slight improvement of the patient's skin condition.

Regardless of any therapeutic intervention, microbiological samples taken from the skin were proven to be MRSP-positive in the next follow up investigation (day 58), whereas the nose was MRSP-negative this time.

The dog was euthanized 3.5 months after the first presentation in the Small Animal Clinic because of its therapy-resistant skin disease and multiple arthroses.

As a presumptive dog-to-human transmission of MRSP has been described before (Guardabassi et al., 2004a; Sasaki et al., 2007a; van Duijkeren et al., 2008), additional voluntary nasal swabs of both owners (male, female) were taken during the treatment period in order to evaluate their colonization status. In these samples, which were taken three times between day 23 and day 64, MRSP was identified once in the first swab derived from the male owner (day 23).

Comparative characterization of the four canine and the single human MRSP isolate using PFGE, SCC*mec* typing, PCR detection of the genes encoding for the toxins SIET and LukI showed identical results: All isolates belonged to a certain PFGE subtype (A10; according to Ruscher et al., 2010), harboured SCC*mec*III and possessed genes encoding SIET and LukI (Fig. 1).

A representative MLST- and *spa* type analysis was conducted for one of the canine isolates (IMT-16102) and the single human isolate (IMT-15347). Sequence type (ST) 71 and *spa* type t05 was associated with both isolates.

Consequently, antimicrobial susceptibility testing of IMT-16102 and IMT-15347 showed similar results, including a broad resistance against several non-ß-lactam antibiotics (Tab. 1).

For bacterial identification and resistance determination all samples were routinely streaked onto the following media: standard nutrient agar I (Roth GmbH, Karlsruhe, Germany) charged with 5% defibrinated sheep blood, Chrom agar orientation (Mast Diagnostica, Reinfeld, Germany) and Gassner agar (Sifin, Berlin, Germany). Hemolysis and growth characteristics were evaluated after aerobic incubation at 37°C for 24 hours. Bacteriological identification of *S. pseudintermedius* was carried out as described previously (Sasaki et al., 2007b). Furthermore, sequence analysis of the housekeeping genes *pta* and *cpn*60 was carried out to approve the phenotypical species determination (Bannöhr et al., 2007).

Using the agar diffusion method (Oxacillin 5µg, breakpoint ≤ 17mm) methicillin-resistance was revealed (Bemis et al., 2009) and verified by PCR for *mec*A (Merlino et al., 2002).

Broth microdilution method including several non-ß-lactams was used for determination of antimicrobial susceptibility according to the CLSI documents (Clinical And Laboratory Standards Institute, 2008a, b). The results are shown in Table 1.

MLST and *spa* typing

Multilocus sequence typing (MLST) was performed by DNA sequence analysis of internal fragments of five genes (*cpn*60, *tuf*, *pta*, *agr*D and 16S rRNA) according to Bannöhr et al., (2007). DNA sequencing was carried out commercially by AGOWA (Berlin, Germany) and the alignment with deposited sequences for identification of sequence types (ST) was done by using the NCBI BLAST (www.ncbi.nlm.nih.gov) and Lasergene software (DNAstar, Lasergene v6, DNASTAR Inc, Madison, USA).

Spa typing of MRSP isolates was carried out as described previously (Ruscher et al., 2010). The *spa* type was assigned in accordance with previously published results (Moodley et al., 2008).

PFGE and SCC*mec* typing

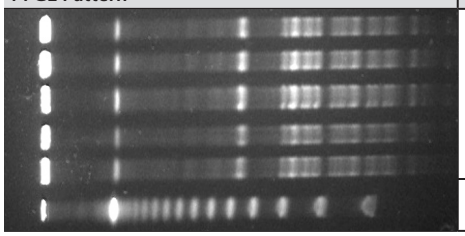
PFGE and Staphylococcal Cassette Chromosome *mec*-typing (SCC*mec*) was performed as described previously (Hanssen et al., 2004; Ito et al., 2004; Boyle-Vavra et al., 2005).

TABLE 1: *Results of MIC-determination of Non-ß-lactam agents*

Antimicrobial agent	MIC Range tested (mg/l)*	Host	
		human	dog
	strain:	IMT-15347	IMT-16102
non-ß-lactam antimicrobials			
Amikacin	≤ 4	susceptible	susceptible
Clindamycin	≤ 0,5 to >2	resistant	resistant
Ciprofloxacin	1 to > 2	resistant	resistant
Erythromycin	≤ 0,25 to > 4	resistant	resistant
Fusidic acid	≤ 4	susceptible	susceptible
Gentamicin	> 8	resistant	resistant
Levofloxacin	≤ 1 to > 4	resistant	resistant
Linezolid	≤ 1 to 4	susceptible	susceptible
Rifampin	≤ 0.5	susceptible	susceptible
Teicoplanin	≤ 1	susceptible	susceptible
Tetracyclin	≤0,5 to > 8	resistant	resistant
Tobramycin	> 8	resistant	resistant
Trimethoprim/Sulfamethoxazol	>2/38	resistant	resistant
Vancomycin	≤1 to 4	susceptible	susceptible

* Breakpoints according to Clinical And Laboratory Standards Institute (CLSI), approved standard M100–S18, M31–A3.

FIGURE1: *PFGE analysis showing indistinguishable DNA restriction pattern after digestion with SmaI of all MRSP isolates of canine (four) and human (one) origin, followed by molecular typing results (MLST, spa- and SCCmec typing in addition to PCR results for the genes encoding SIET and LukI).*

PFGE Pattern	Isolate	Host	Day of Isolation	MIC[1]	PFGE[2]	MLST	*spa* type	SCC-*mec*	SIET	LukI
	IMT-15319	dog (skin)	d 1	n. t.	A 10	n. t.	n. t.	III	pos.	pos.
	IMT-15347	human (nose)	d 23	yes	A 10	71	t05	III	pos.	pos.
	IMT-15359	dog (skin)	d 36	n. t.	A 10	n. t.	n. t.	III	pos.	pos.
	IMT-15360	dog (nose)	d 36	yes	A 10	n. t.	n. t.	III	pos.	pos.
	IMT-16102	dog (skin)	d 58	yes	A 10	71	t05	III	pos.	pos.
	Lambda ladder (PFGE)									

[1] MIC: Results of antimicrobial susceptibility testing see Table 1.

[2] PFGE subtype was denominated according to Ruscher et al., (2010).

Toxin detection

PCR-based detection was conducted for the *luk*-I-operon, which consists of luk-F and *luk*-S, and a further exotoxin (SIET) as described in previous studies (Futagawa-Saito et al., 2004; Lautz et al., 2006). Sequencing of PCR products of the SIET encoding gene, *luk*S and *luk*F was realised by AGOWA (Berlin, Germany). Results were confirmed by alignment of toxin sequences with the following published sequences: GenBank accession number AB099710 (*siet*), AB185109 (l*uk*S), and X79188 (*luk*F).

Discussion

Here we report about a long-term skin disease in a dog caused by a multidrug-resistant MRSP strain. Furthermore, recurrent nasal screenings revealed a transient nasal MRSP colonization in one of the animal owners during the study period. Finally, all topical (chlorhexidine washings, fusidic acid cream) and parenteral (amikacin) therapeutic trials failed to eradicate the pathogen from the skin of this senior dog.

MRSP infections in small animals and horses seem to be on the rise in general (Black et al., 2009; Moodley et al., 2009; Ruscher et al., 2010), and isolates resistant to five or more classes of antibiotics have been reported recently (Loeffler et al., 2005; Sasaki et al., 2007a; Ruscher et al., 2009).

The significant problem concerning MRSP skin infections and application of fusidic acid as a possible therapeutic option has already been described (Loeffler et al., 2008). However, in the case presented here, topical therapy with fusidic acid cream (2%) failed although the bacteria appeared to be susceptible to this antibiotic in vitro (Tab. 1). Furthermore, parenteral treatment with amikacin failed to improve the patient`s condition. Similar to other aminoglycosides, amikacin has a nephrotoxic and ototoxic potential, therefore a longer treatment with amikacin bears the risk of complications, especially in senior patients.

Another possible aspect of treatment failure in this case could be a re-infection with MRSP from an unknown source. The very cooperative dog owners had been instructed extensively about hygienic measures throughout therapy, including hand hygiene, washing and disinfection of floors, utensils and especially the collar. Only the proposed clipping of the fur was declined for personal reasons.

Nonetheless, re-infection through objects or surfaces in the household, which were difficult to disinfect or simply not considered as a potential source of MRSP, cannot be ruled out in this case. In addition, contact of the canine patient to other colonized animals may have also been a possibility.

Our results indicate a possible dissemination of MRSP from dog-to-human within the household by an infected and colonized dog, as described for staphylococcal species of the *intermedius* group (SIG) before: Six out of 13 (46%) owners of dogs with deep pyoderma had SIG isolated from their nasal and oral cavities. All isolates were indistinguishable by PFGE to those isolated from their individual dogs (Guardabassi et al., 2004a). In addition, close contact between household pets and humans seems to increase the risk for transmission of resistant pathogens (Guardabassi et al., 2004b). Probably, nasally colonized owners are able to re-infect their animal with MRSP. Otherwise, nasal colonization with MRSP in humans seems to be transient (Frank et al., 2009) and MRSP infection of the dog in this case continued after two negative sampling rounds of the owners.

However, transient MRSP colonization of the anterior nose in humans might be a possible source of (re-) infection for other individuals, especially in veterinary settings, like it has been demonstrated for MRSA colonized veterinary personnel (Walther et al., 2009).

In general, a pathogenic potential of SIG in humans is possible. While SIG infections seem to be related to patients who have undergone invasive procedures, healthy humans have the risk of nasal colonisation (Tanner et al., 2000). Additionally, the occurrence of SIG in dog owners was found to be significantly higher than in other people (Guardabassi et al., 2004a). Therefore, MRSP infection may be a greater risk for dog owners rather than other people.

This has led to the recommendation to consider topical eradication measures for the decolonization of MRSP carrier animals to prevent both, animal re-infection and transmission of multidrug-resistant *staphylococci* to susceptible humans (Loeffler et al., 2008).

Sequence typing revealed that MRSP isolated in this study belong to ST71, which has been described as the dominant ST in Europe in the recent past (Bannöhr et al., 2007; Ruscher et al., 2010), whereas ST68 seems to be the predominant genetic lineage in the USA (Black et al., 2009).

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