Horino et al. Renal Replacement Therapy (2016) 2:32
DOI 10.1186/s41100-016-0041-3

Renal Replacement Therapy

CASE REPORT Open Access

Kocuria kristinae septic arthritis associated with infectious endocarditis in a hemodialysis patient with diabetes mellitus: a case report and literature review

Taro Horino*, Yoshiko Shimamura, Koji Ogata, Kosuke Inoue and Yoshio Terada

Abstract

Background: *Kocuria kristinae* is an aerobic Gram-positive coccus that is considered ubiquitous and non-pathogenic in healthy individuals. Furthermore, only 27 reports have described cases of critical infections with this microorganism, which is notoriously difficult to identify.

Case presentation: We report the case of a 61-year-old male hemodialysis patient with diabetes mellitus, who developed severe septic arthritis that was associated with infectious endocarditis, which was identified using ^{18}F-fluorodeoxyglucose positron-emission tomography/computed tomography (FDG-PET-CT). *K. kristinae* was identified in two separate blood cultures. The patient recovered immediately after being treated using piperacillin followed by ampicillin/sulbactam and gentamicin.

Conclusions: To the best of our knowledge, this is the first case of *K. kristinae* septic arthritis associated with infectious endocarditis in a hemodialysis patient with diabetes mellitus. We suggest that physicians consider the pathogenic potential of *K. kristinae*, which can cause fatal infections, such as septic arthritis and infectious endocarditis, in immunocompromised patients. FDG-PET-CT is a useful and safe diagnostic tool for determining the cause of inflammatory disease in dialysis patients.

Keywords: *Kocuria kristinae*, Septic arthritis, Infectious endocarditis, Hemodialysis, FDG-PET-CT

Background

In Japan, the number of patients with diabetes is increasing every year, as is the rate of renal replacement therapy (RRT) for treating diabetic nephropathy. Both end-stage renal disease (ESRD) and diabetes mellitus are severe immunocompromised states, and dialysis patients are prone to infectious complications. Furthermore, we believe that patients with diabetes mellitus are predisposed to invasive infections. Therefore, we describe the case of a hemodialysis patient who had a wound infection and bacteremia with *Kocuria kristinae*, which is a microorganism that is considered non-pathogenic in healthy individuals. In addition, we provide a review of case reports regarding *K. kristinae* infections in humans.

Case presentation

A 61-year-old male hemodialysis patient with a history of diabetes mellitus and polyarthralgia was referred to our hospital. Two months before the referral, the patient had arthralgia of the right knee, which exhibited swelling, tenderness, and restricted motion. The patient subsequently experienced severe polyarthralgia that involved both shoulders, wrists, hip joints, foot joints, and the left knee joint, as well as rapidly progressing severe lumbago. At the admission, his temperature was 36.6 °C. A physical examination revealed swelling and warmth in all joints that the patient identified as being painful. The laboratory test results are shown in Table 1. A chest radiograph revealed mild cardiomegaly without other remarkable findings. Joint radiographs revealed no deformities. Magnetic resonance imaging (MRI) of the left hip joint revealed high-intensity lesions surrounding

* Correspondence: horinott@yahoo.co.jp
Department of Endocrinology, Metabolism and Nephrology, Kochi Medical School, Kohasu, Okoh-cho, Nankoku 783-8505, Kochi, Japan

Table 1 Laboratory data at admission

CBC (normal range)		Blood chemistry		Tumor markers		Blood culture	
RBC (400–552)	$239 \times 10^4/mm^3$	FBS (70–110)	179 mg/dL	CEA (<3.4)	0.8 ng/mL	*Kocuria kristinae*	(+)
Hb (13.2–17.2)	6.9 g/dL	Alp (104–338)	501 U/L	CA19-9 (<37)	2.8 U/mL	Antimicrobial susceptibilities (MIC)	μg/mL
Ht (40.4–51.1)	21.9 %	γ-GTP (5–70)	41 U/L	Infection		Penicillin	0.12
Plt (14.8–33.9)	$46.8 \times 10^4/mm^3$	t-Bil (0.2–1.2)	0.3 mg/dL	HBs Ag	(–)	Oxacillin	≤2.0
WBC (3.6–9.6)	$5.6 \times 10^3/mm^3$	d-Bil (0–0.4)	0.0 mg/dL	HCV Ab	(–)	Ampicillin	≤0.25
neu	73.0 %	ChE (168–470)	123 U/L	HTLV-I Ab	(–)	Cefazolin	≤8.0
lym	8.0 %	ALT (8–42)	24 U/L	EBV		Cefotaxime	≤8.0
mon	13.0 %	AST (13–33)	37 U/L	VCA-IgG	×640	cefepime	≤8.0
eos	6.0 %	LDH (119–229)	125 U/L	VCA-IgM	×<10	Cefotiam	≤8.0
bas	0.0 %	CPK (62–287)	10 U/L	EBNA	×20	Cefpirome	≤8.0
aty-ly	0.0 %	Crn (0.6–1.1)	10.3 mg/dL	Mumps	(–)	Cefozopran	≤8.0
Electrolytes		BUN (8–20)	52 mg/dL	Herpes zoster	(–)	Cefmetazole	≤16.0
Na (136–145)	137 mmol/L	UA (3.4–7.8)	8.5 mg/dL	Parvovirus B19	(–)	Cefdinir	≤1.0
K (3.4–4.5)	4.4 mmol/L	Amy (37–125)	71 U/L	Syphilis	(–)	Cefditoren	≤1.0
Cl (100–108)	99 mmol/L	TG (30–150)	106 mg/dL	QFT	(–)	Flomoxef	≤8.0
Ca (8.7–11.0)	10.2 mg/dL	H-Chol (>40)	17 mg/dL	Candida Ab	(–)	Imipenem/cilastatin	≤4.0
P (2.5–4.5)	4.7 mg/dL	L-Chol (<140)	61 mg/dL	β-D-glucan	(–)	Meropenem	≤4.0
Mg (1.8–2.4)	2.5 mg/dL	Serological examinations		Chlamydia trachomatis		Amoxicillin	≤4.0
Proteinogram		CRP (<0.3)	35.5 mg/dL	IgG	(–)	Gentamicin	≤4.0
TP (6.7–8.1)	7.1 g/dL	SAA (<8.0)	1843.9 mg/mL	IgA	(–)	Amikacin	≤16.0
Alb (3.9–4.9)	2.6 g/dL	MMP-3 (36.9–121.0)	586.4 ng/mL	Chlamydia pneumoniae		Arbekacin	≤4.0
Glb	4.5 g/dL	CH50 (30–45)	47.0 U/mL	IgG	(–)	Erythromycin	≤0.5
α1	11.2 %	C3 (65–135)	131.5 mg/dL	IgA	(–)	Clarithromycin	≤2.0
α2	16.6 %	C4 (13–35)	31.7 mg/dL	Synovial fluid		Clindamycin	≤0.5
β	10.7 %	ANA	(–)	Glossly purulent material		Minocycline	≤4.0
γ	34.3 %	ds-DNA Ab	(–)	WBC	$13{,}200 \times 10^3/mm^3$	Linezolid	≤2.0
IgG (870–1700)	2519 mg/dL	SSA Ab	(–)	neu	95.0 %	Vancomycin	≤2.0
IgA (110–410)	497 mg/dL	SSB Ab	(–)	pla	5.0 %	Teicoplanin	≤8.0
IgM (35–220)	87 mg/dL	RF	(–)	Crystals	(–)	Fosfomycin	≤4.0

Table 1 Laboratory data at admission *(Continued)*

M-protein	(–)	ACPA	(–)	Culture	(–)	Levofloxacin	≤1.0
HbA1c (4.6–6.2)	7.9 %	MPO-ANCA	(–)			Sulfamethoxazole/trimethoprim	≤2.0
ESR (<20)	>140 mm/h	PR3-ANCA	(–)			Rifampicin	≤1.0

Abbreviations: *CBC* complete blood cell count, *RBC* red blood cell count, *Hb* hemoglobin, *Ht* hematocrit, *Plt* platelet, *ESR* erythrocyte sedimentation rate, *WBC* white blood cell count, *neu* neutrophils, *lym* lymphocytes, *mon* monocytes, *eos*eosinophils, *bas* basophils, *aty-ly* atypical lymphocytes, *Na* sodium, *K* potassium, *Cl* chlorine, *Ca* calcium, *P* phosphorus, *Mg* magnesium, *TP* total protein, *Alb* albumin, *Glb* globulin, *IgG* immunoglobulin G, *IgA* immunoglobulin A, *IgM* immunoglobulin M, *FBS* fasting blood sugar, *Alp* alkaline phosphatase, *γ-GTP* γ-glutamyltransferase, *t-Bil* total bilirubin, *d-Bil* direct bilirubin, *ChE* cholinesterase, *ALT* alanine transaminase, *AST* aspartate aminotransferase, *LDH* lactate dehydrogenase, *CPK* creatine phosphokinase, *Crn* creatinine, *BUN* blood urea nitrogen, *UA* uric acid, *Amy* amylase, *TG* triglyceride, *H-Chol* high-density lipoprotein cholesterol,
L-Chol low-density lipoprotein cholesterol, *CRP* C-reactive protein, *SAA* serum amyloid A, *MMP-3* matrix metalloproteinase-3, *CH50* complement activity, *C3* complement 3, *C4* complement 4, *ANA* antinuclear antibody, *SSA Ab* anti-SS-A/Ro antibody,*SSB Ab* anti-SS-B/La antibody, *RF* rheumatoid factor, *ACPA* anti-cyclic citrullinated peptide antibody, *MPO-ANCA* myeloperoxidase-anti-neutrophil cytoplasmic antibody, *PR3-ANCA* serine proteinase3-anti-neutrophil cytoplasmic antibody, *HBs Ab* hepatitis B surface antigen, *HCV Ab* hepatitis C virus antibody, *HTLV-I Ab* human T-lymphotropic virus type 1 antibody,*pla* plasma cell, *EBV* Epstein-Barr virus, *VCA* viral capsid antigen, *EBNA* Epstein-Barr nuclear antigen, *QFT* QuantiFERON-TB, *pla* plasma cell

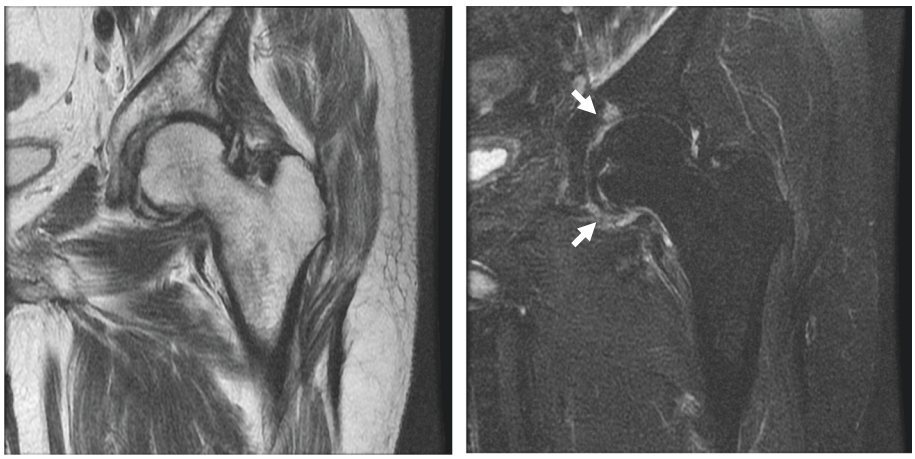

Fig. 1 The magnetic resonance imaging findings. The magnetic resonance findings from the left hip joint reveal high-intensity lesions (*white arrows*) surrounding the head of the femoral bone in the T2-weighted image (*right panel*)

the head of the femoral bone on the T2-weighted image (Fig. 1).[18]F-fluorodeoxyglucose positron-emission tomography/computed tomography (FDG-PET-CT) revealed short segments of increased FDG uptake within the shoulders, wrists, hip joints, knees, and foot joints (Fig. 2). We had performed arthrocentesis at the admission, and an aspiration specimen from the right knee joint yielded grossly purulent material. Synovial fluid white cell counts were 13,200/mm^3, with 95 % polymorphonuclear leukocytes and 5 % monomorphonuclear leukocytes. Although the synovial fluid culture was negative, clusters of Gram-positive cocci were isolated from a blood specimen that was taken at the admission. Two blood cultures were both positive for *K. kristinae*, and these results were confirmed using the Vitek 2 system. The isolate was sensitive to all tested antibiotics (Table 1). We started treatment using intravenous piperacillin (2 g once per day), and the polyarthritis immediately improved. After 14 days of antibiotic treatment, the

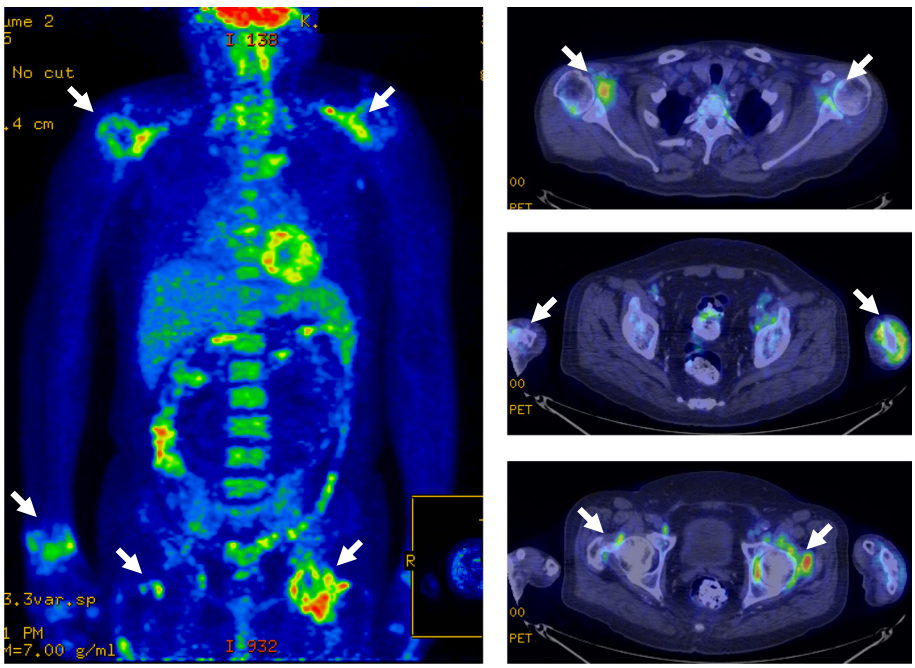

Fig. 2 The [18]F-fluorodeoxyglucose positron-emission tomography/computed tomography findings. [18]F-fluorodeoxyglucose positron-emission tomography/computed tomography reveals short segments of increased fluorodeoxyglucose uptake (*white arrows*) within the shoulders, wrists, hip joints, knees, and foot joints

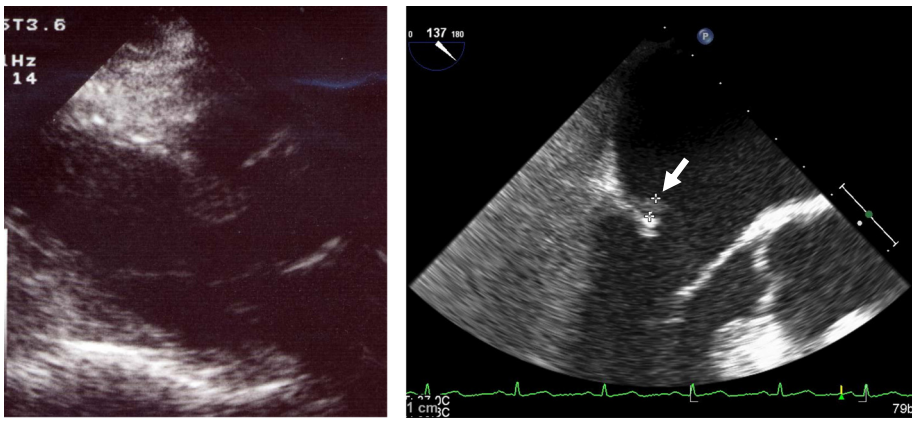

Fig. 3 The echocardiography findings. (*Left panel*) Echocardiography reveals no remarkable findings at 2 years before the presentation. (*Right panel*) Echocardiography at 1 month after the admission reveals mitral valve vegetation

patient's C-reactive protein levels were 0.8 mg/dL and the other laboratory data were normalized.

Electrocardiography detected no remarkable findings during his clinical course. However, 1 month after the admission, echocardiography detected vegetation on the mitral valve, which was compatible with infectious endocarditis (Fig. 3, right panel), although there had been no findings of vegetation at 2 years before the admission (Fig. 3, left panel). Physical examination after the echocardiography revealed Janeway's lesions, although we did not detect Osler's nodes or Roth's spots. Therefore, we stopped the intravenous piperacillin therapy and started treatment using intravenous ampicillin/sulbactam (1.5 g every other day) and intravenous gentamicin (100 mg every other day). The patient's clinical course was uneventful, and he was later discharged to our outpatient clinic (Fig. 4).

Discussion

K. kristinae was first described as *Micrococcus kristinae* in 1974 and was subsequently classified as *Micrococcus* spp., which is now separated into *Micrococcus*, *Nesterenkonia*, *Kytococcus*, *Dermacoccus*, and *Kocuria* [1, 2]. *K. kristinae* is a strictly aerobic species that is commonly identified among skin and oral flora [3]. Most physicians consider *Micrococcus* spp. (including *Kocuria*) nonpathogenic, and we initially assumed that the blood cultures from the present case were negative for pathogenic

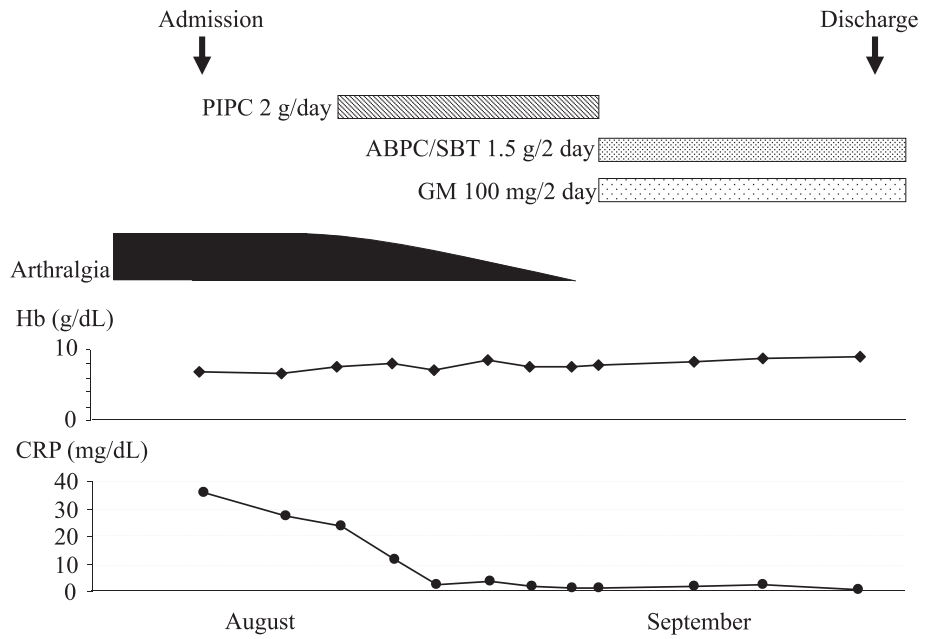

Fig. 4 The patient's clinical course. *PIPC* piperacillin, *APBC* ampicillin, *SBT* sulbactam, *GM* gentamicin, *Hb* hemoglobin, *CRP* C-reactive protein

bacteria. However, reports of infectious diseases that were caused by this species are gradually being published. The clinical characteristics of the 28 reported cases of *K. kristinae* infections are summarized in Tables 2 and 3 [4–16]. Most *K. kristinae* infections are reported as infections in immunocompromised hosts or patients with underlying malignancy, ESRD requiring dialysis, diabetes mellitus, and prematurity. Although published reports of *K. kristinae* infections are rare, the prevalence of this infection, especially among immunocompromised hosts, may be underestimated, as most physicians likely do not consider this organism as a pathogen.

Patients who are receiving RRT often have infectious diseases, although the identification of their causative microorganism(s) and focus of infection are difficult to accurately diagnose. Furthermore, patients who are receiving RRT are frequently immunocompromised and may have atypical infectious organisms that are considered normal bacterial flora in healthy individuals [17]. Among patients who are undergoing RRT, the host's immunity may be compromised by uremia that interferes with T cell and B cell function, macrophage phagocytosis, and antigen presentation, which can increase the risk of infection [17]. The population of patients with ESRD who receive RRT has been increasing worldwide, and this population included 314,180 Japanese patients in December 2013 [18]. Furthermore, the number of patients with diabetes mellitus is increasing, and diabetic nephropathy has recently become the leading cause (43.8 %) of RRT in Japan [18]. Because ESRD and diabetes mellitus can both lead to an immunocompromised status and increase susceptibility to unusual infections,

Table 2 Demographic data, treatments, and outcomes for 28 patients with *Kocuria kristinae* infections

Case	Antibiotic regimen	Course (days)	TPN	Outcome	Ref.	Year
1	Meropenem + glycopeptide → ciprofloxacin + clindamycin → catheter removal	NA	NA	Recovery	4	2002
2	Levofloxacin	14	No	Recovery	5	2005
3	Ceftriaxone + ofloxacin	16	NA	Death	6	2008
4	Azithromycin + ceftriaxone + vancomycin + oseltamivir → vancomycin + clindamycin → oxacillin	42	Yes	Recovery	7	2011
5	Vancomycin → teicoplanin → oxacillin	NA	Yes	Recovery	8	2011
6	Piperacillin/tazobactam → ciprofloxacin	NA	Yes	Recovery	8	2011
7	Oxacillin + vancomycin	NA	Yes	Recovery	8	2011
8	Oxacillin	NA	Yes	Recovery	8	2011
9	Tobramycin → cefotaxime + tazobactam → ciprofloxacin + teicoplanin + amoxicillin/clavulanic acid	24	No	Recovery	9	2011
10	Cefazolin + cefepime	14	No	Recovery	10	2011
11	Ceftriaxone + vancomycin	14	Yes	Recovery	11	2012
12	Ceftriaxone + metronidazole → sulbactam/ampicillin + gentamicin	25	No	Death	12	2013
13	No antibiotics	No	No	Unknown	13	2013
14	Dicloxacillin + vancomycin + levofloxacin	21	No	Recovery	14	2014
15	Ceftriaxone + amikacin + cefotaxime	16	No	Recovery	14	2014
16	Vancomycin	10	NA	Recovery	15	2014
17	Vancomycin	7	NA	Recovery	15	2014
18	Levofloxacin	7	NA	Recovery	15	2014
19	Linezolid	14	NA	Recovery	15	2014
20	Vancomycin	5	NA	Recovery	15	2014
21	Vancomycin + ceftazidime	10	Yes	Recovery	16	2015
22	Vancomycin	10	Yes	Recovery	16	2015
23	Vancomycin + ceftazidime	10	Yes	Recovery	16	2015
24	Oxacillin + vancomycin	9	Yes	Recovery	16	2015
25	Vancomycin + cefotaxime	12	Yes	Recovery	16	2015
26	Vancomycin + cefotaxime	10	Yes	Recovery	16	2015
27	Vancomycin + piperacillin/tazobactam	10	No	Recovery	16	2015
28	Piperacillin → ampicillin/sulbactam + gentamicin	30	No	Recovery	Current	2015

TPN total parenteral nutrition, *NA* not available

Table 3 Clinical, demographic, and bacterial data from 28 patients with *Kocuria kristinae* infections

Case	Age, year (month)	Sex	Country	Underlying disease	Isolation site	Catheters	Clinical presentation
1	51	F	Italy	Ovarian cancer, chemotherapy	Blood, catheter tip	CVC	Febrile neutropenia, sepsis
2	56	M	Hong Kong	Gallstones	Biliary fluid	No	Acute cholecystitis
3	68	M	France	MDS, acute myelogenous leukemia, tuberculosis, chemotherapy	Blood	CVC	Sepsis
4	29	F	USA	Pregnancy, hyperemesis gravidarum	Blood	CVC	Suppurative thrombosis
5	89	F	Taiwan	Post-resection ischemic bowel status, short bowel syndrome	Blood	CVC	Endocarditis
6	37	F	Taiwan	Gastric cancer	Blood	CVC	Bacteremia
7	2	M	Taiwan	Congenital short bowel syndrome, hypogammaglobulinemia	Blood	CVC	Bacteremia
8	68	F	Taiwan	Gastric cancer	Blood	CVC	Bacteremia
9	78	M	Italy	ESRD on CAPD	Peritoneal fluid	PDC	Peritonitis
10	69	M	China	ESRD on CAPD	Peritoneal fluid	PDC	Peritonitis
11	0 (4.0)	F	Turkey	Prolonged diarrhea, severe failure to thrive	Blood	CVC	Black hairy tongue, fever, bacteremia
12	74	M	Italy	Diabetes mellitus	Blood	No	Foot ulcer, endocarditis, sepsis
13	20	M	India	Urethral stricture	Urine	UC	Malaise
14	20	F	Mexico	ESRD on HD	Blood	CVC	Bacteremia
15	68	M	Mexico	ESRD on CAPD	Peritoneal fluid	PDC	Peritonitis
16	65	M	India	Lung small cell carcinoma	Sputum	NA	Leukocytosis (neutrophillia)
17	65	M	India	Carcinoma soft palate	Sputum	NA	Leukocytosis (neutrophillia)
18	41	F	India	Carcinoma lip and gingival sulcus	Sputum	NA	Leukocytosis (neutrophillia)
19	39	M	India	Squamous cell carcinoma of the buccal mucosa	pus	NA	Leukocytosis (neutrophillia)
20	42	F	India	Ductal carcinoma of the breast	pus	NA	Leukocytosis (neutrophillia)
21	0 (1.4)	M	Taiwan	Prematurity	Blood	CVC	Sepsis
22	0 (0.6)	M	Taiwan	Prematurity	Blood	CVC	Sepsis
23	0 (0.7)	F	Taiwan	Prematurity	Blood	CVC	Sepsis
24	0 (1.1)	F	Taiwan	Prematurity	Blood	CVC	Sepsis
25	0 (0.6)	F	Taiwan	Prematurity	Blood, catheter tip	CVC	Sepsis
26	0 (0.6)	F	Taiwan	Prematurity	Blood, catheter tip	CVC	Sepsis
27	0 (2.5)	F	Taiwan	Leukemia	Blood, catheter tip	CVC	Neutropenic fever
28	61	M	Japan	ESRD on HD, diabetes mellitus	Blood	No	Septic arthritis, endocarditis

Age is presented as year (month)
Abbreviations: *ESRD* end-stage renal disease, *CAPD* chronic ambulatory peritoneal dialysis, *HD* hemodialysis, *CVC* central venous catheter, *PDC* peritoneal dialysis catheter, *UC* urinary catheter

patients with ESRD and diabetes mellitus are likely very susceptible to uncommon and usually non-pathogenic microorganisms. However, *Kocuria* spp. infections are rarely reported among patients with diabetes and RRT. We speculate that there are two reasons for this phenomenon. First, most physicians believe that *Kocuria* spp., *Micrococcus* spp., and especially *K. kristinae* are non-pathogenic and might not consider these organisms as potentially causative organisms, despite observing positive culture results. Second, most *Kocuria* spp. and *Micrococcus* spp. are sensitive to almost all antibiotics (except ampicillin and erythromycin), and monotherapy often results in good outcomes [19]. In this context, Japanese investigators have reported that the causes of fever of unknown origin (FUO) are infection (27.7 %), non-infectious inflammatory disease (18.4 %), malignancy

(10.2 %), other conditions (14.8 %), and unknown causes (28.9 %) [20]. Thus, given the frequency of an unknown cause of FUO, we usually use empirical antibiotics to treat infections of unknown origin until culture results are available. Therefore, it is possible that repeating cultures will not identify the same microorganisms in each culture, as the empirical antibiotics might cure the infection without severe clinical events (e.g., sepsis) before the second sample is collected. Interestingly, infection is the primary cause of death among Japanese patients who are receiving RRT (26.5 %) [19], and it is likely that the number of patients with diabetes and RRT is going to continue increasing. Furthermore, the population of elderly Japanese patients is also increasing, which will likely result in a larger number of immunocompromised hosts. Based on these factors, it is possible that an increasing number of patients may have underlying *K. kristinae* infection that is not diagnosed until they develop severe complications, such as bacteremia and endocarditis. Therefore, it may be prudent to assess the pathogenic capacity of *K. kristinae* and any other unusual microorganisms, even if an infection of unknown origin improves after antibiotic monotherapy in patients who are elderly, have diabetes, or are receiving RRT. Moreover, clinicians should not underestimate the importance of a positive culture result for *K. kristinae*, as patients may experience repeated infections, develop severe infectious complications, and possibly die from this infection.

During recent years, many investigators have reviewed the diagnostic value of FDG-PET-CT. Although FDG was first developed to trace brain metabolism [21], FDG-PET-CT has subsequently been recognized as an early diagnostic tool that has high sensitivity for malignancies, such as melanoma and cervical, lung, breast, gastrointestinal, ovarian, and prostatic cancers [22]. Furthermore, recent studies have found that FDG-PET-CT could detect the existence of inflammatory diseases and determine their severity and extent [23]. Although contrast-enhanced CT and/or MRI are performed for diagnosing and examining malignancies, infections, and autoimmune diseases, the use of contrast agents for these techniques is often contraindicated in patients with severe renal insufficiency, as these agents increase the incidence of their adverse effects. In contrast, FDG-PET-CT is considered safer and simpler for dialysis patients, compared to contrast-enhanced CT or MRI. Therefore, we suggest that FDG-PET-CT should be used for diagnosing and evaluating dialysis patients if the origin of their disease is unclear and that physicians should not delay this diagnostic imaging, which can lead to a poor prognosis.

Conclusions

We report the first case of *K. kristinae* septic arthritis associated with infectious endocarditis in a chronic hemodialysis patient with diabetes mellitus. We suggest that physicians should consider the pathogenic potential of *K. kristinae*, as it can cause fatal infections (e.g., septic arthritis and infectious endocarditis) in immunocompromised patients or patients with diabetes and RRT. We also suggest that FDG-PET-CT is a very useful and safe diagnostic tool, which can help identify the focus of inflammatory disorders in dialysis patients.

Consent

Written informed consent was obtained from the patient for publication of this case report and the accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

Competing interests

The authors declare that they have no competing interests.

Authors' contributions

TH, YS, KO, and KI performed the laboratory testing. TH and YT drafted the manuscript. All authors read and approved the final manuscript.

Received: 28 November 2015 Accepted: 8 April 2016
Published online: 13 June 2016

References

1. Kloos WE, Tornabene TG, Schleifer KH. Isolation and characterization of micrococci from human skin, including two new species: *Micrococcus lylae* and *Micrococcus kristinae*. Int J Syst Evol Bacteriol. 1974;24:79–101.
2. Stackebrandt E, Koch C, Gvozdiak O, Schumann P. Taxonomic dissection of the genus *Micrococcus*: *Kocuria*gen. nov., *Nesterenkonia*gen. nov., *Kytococcus*gen. nov., *Dermacoccus* gen. nov., and *Micrococcus* Cohn 1872 gen. emend. Int J Syst Bacteriol. 1995;45:682–92.
3. Cordero MR, Zumalacárregui JM. Characterization of micrococcaceae isolated from salt used for Spanish dry-cured ham. Lett Appl Microbiol. 2000;31:303–6.
4. Basaglia G, Carretto E, Barbarini D, et al. Catheter-related bacteremia due to *Kocuria kristinae*in a patient with ovarian cancer. J Clin Microbiol. 2002;40:311–3.
5. Ma ES, Wong CL, Lai KT, et al. *Kocuria kristinae* infection associated with acute cholecystitis. BMC Infec Dis. 2005;5:60.
6. Martinaud C, Gaillard T, Brisou P, Gisserot O, de Jaureguiberry JP. Bacteremia caused by *Kocuria kristinae* in a patient with acute leukemia. Med Mal Infect. 2008;38:165–6 [in French].
7. Dunn R, Bares S, David MZ. Central venous catheter-related bacteremia caused by *Kocuria kristinae*: case report and review of the literature. Ann Clin Microbiol Antimicrob. 2011;10:31.
8. Lai CC, Wang JY, Lin SH, et al. Catheter-related bacteraemia and infective endocarditis caused by *Kocuria*species. Clin Microbiol Infect. 2011;17:190–2.
9. Calini A, Mattei R, Lucarotti I, Bartelloni A, Rosati A. *Kocuria kristinae*: an unusual cause of acute peritoneal dialysis-related infection. Perit Dial Int. 2011;31:105–7.
10. Cheung C, Cheng N, Chau C, Li CS. An unusual organism for CAPD related peritonitis: *Kocuria kristinae*. Perit Dial Int. 2011;31:107–8.
11. Karadag Oncel E, Boyraz MS, Kara A. Black tongue associated with *Kocuria* (*Micrococcus*) *kristinae* bacteremia in a 4-month-old infant. Eur J Pediatr. 2012;171:593.
12. Citro R, Prota C, Greco L, et al. *Kocuria kristinae* endocarditis related to diabetic foot infection. J Med Microbiol. 2013;62:932–4.
13. Tewari R, Dudeja M, Das AK, Nandy S. *Kocuria kristinae* in catheter associated urinary tract infection: a case report. J Clin Diagn Res. 2013;7:1692–3.
14. Chávez Valencia V, Orizaga de la Cruz C, Aguilar Bixano O, Huerta Ruíz MK, Sánchez Estrada EE. Infections due to *Kocuria kristinae*: case reports of two patients and review of the literature. Gac Med Mex. 2014;150 Suppl 2:183–5 [in Spanish].

15. Ahmed NH, Biswal I, Roy P, Grover RK. *Kocuria kristinae*, an unusual pathogen causing opportunistic infections in patients with malignancy. Indian J Med Microbiol. 2014;32:456–8.
16. Chen HM, Chi H, Chiu NC, Huang FY. *Kocuria kristinae*: a true pathogen in pediatric patients. J Microbiol Immunol Infect. 2015;48:80–4.
17. Leone S, Suter F. Severe bacterial infections in haemodialysis patients. Infez Med. 2010;18:79–85.
18. Masakane I, Nakai S, Ogata S, et al. An overview of regular dialysis treatment in Japan (as of 31 December 2013). Ther Apher Dial. 2015;19:540–74.
19. Szczerba I. Susceptibility to antibiotics of bacteria from genera *Micrococcus*, *Kocuria*, *Nesternkonia*, *Kytococcus* and *Dermacoccus*. Med Dosw Mikrobiol. 2003;55:75–80 [in Polish].
20. Yamanouchi M, Uehara Y, Yokokawa H, et al. Analysis of 256 cases of classic fever of unknown origin. Intern Med. 2014;53:2471–5.
21. Reivich M, Kuhl D, Wolf A, et al. The [18F] fluorodeoxyglucose method for the measurement of local cerebral glucose utilization in man. Circ Res. 1979;44:127–37.
22. Gallamini A, Zwarthoed C, Borra A. Positron emission tomography (PET) in oncology. Cancers (Basel). 2014;6:1821–89.
23. Balink H, Verberne HJ, Bennink RJ, van Eck-Smit BL. A rationale for the use of F18-FDG PET/CT in fever and inflammation of unknown origin. Int J Mol Imaging. 2012. doi: 10.1155/2012/165080.