Journal of Taibah University Medical Sciences (2015) 10(4), 492–495

Taibah University

Journal of Taibah University Medical Sciences

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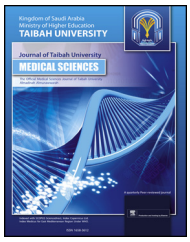

Case Report

Kodamaea (*Pichia*) *ohmeri* peritonitis in a nine-year-old child in Saudi Arabia treated with caspofungin

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Received 10 March 2015; revised 14 August 2015; accepted 14 August 2015; Available online 1 October 2015

الملخص

كودامايا (بيتشيا) أوميري هو فطر انتهازي ناشئ يتسبب في عدوى قاتلة. نعرض في هذا التقرير حالة طفل يبلغ من العمر ٩ أعوام يعاني من المتلازمة الكلوية المقاومة للكورتيزون. كان الطفل على الغسيل البريتوني والسيكلوسبورين وعُرض علينا بحالة التهاب الصفاق بسبب كودامايا أوميري. استجاب الطفل للعلاج بكاسبوفنجين الذي كان الفطر متحسس له والتي وُصفت له بعد الإزالة الفورية للقثطار البريتوني.

الكلمات المفتاحيه: كودامايا أوميري؛ كاسبوفنجين؛ التهاب الصفاق

Abstract

Kodamaea (*Pichia*) *ohmeri* is an emerging fatal opportunistic fungal infection. We describe the case of a 9-year-old boy known to have steroid-resistant nephrotic syndrome. The child, on peritoneal dialysis and cyclosporine, presented with peritonitis related to *K. ohmeri*. The patient's condition was cured by immediate removal of the peritoneal catheter and treatment with Caspofungin, which was prescribed for susceptible fungal infection.

Keywords: *Kodamaea ohmeri*; Caspofungin; Peritonitis

Introduction

Kodamaea (*Pichia*) *ohmeri* is a yeast belonging to the fungi kingdom and the *Saccharomycetes* class, *Kodamaea* genus, and *K. ohmeri* species, which was previously called *Yamadazma ohmeri*.[1]

It is the teleomorphic form of *Candida guilliermondii var. membranaefaciens* and is widely used in the food industry for the fermentation of fruits, pickles, and rinds.[1]

The genus *Kodamaea* currently comprises five species: *K. anthrophila*, *K. kakaduensis*, *K. laetipori*, *K. nitidulidarum*, and *K. ohmeri*.[2] Twenty-seven cases have been reported in the medical literature with different presentations and risk factors (Table 1).[6,9,10,12–19] Most of the patients were immune-compromised and diabetic, but a few cases were immune-competent. Among them, only one patient was treated with caspofungin,[4] and another patient was successfully treated with micafungin.[3] There were 10 reported deaths despite antifungal therapy with amphotericin and fluconazol (Table 1).[6,8,9,12,15,17,20,23]

In this report, we describe the case of a 9-year-old child with ESRD on peritoneal dialysis complicated by *Kodamaea* (*pichia*) *ohmeri* peritonitis that was successfully treated with caspofungin.

The case

A nine-year-old boy was referred to our hospital with a diagnosis of steroid-resistant nephrotic syndrome. He was

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Peer review under responsibility of Taibah University.

ELSEVIER Production and hosting by Elsevier

 http://dx.doi.org/10.1016/j.jtumed.2015.08.003

Table 1: A summary of the clinical characteristics of the cases of *K. ohmeri* infection in the most recent medical literature.[3]

Case	Reference Year	Country	Age Sex	Risk factors	Source	Antifungal	Outcome
1	8 (2007)	Korea	11/12 M	Burkitt's lymphoma Neutropenia, CVC	Blood	FCZ	Died
2	8 (2007)	Korea	41/M	Alcohol, KA, TB, CVC	Blood	No antifungal	Survived
3	8 (2007)	Korea	47/M	DM, CRF, CVC	Blood	FCZ, AMB	Died
4	8 (2007)	Korea	4/F	TOF, CVC	Blood	FCZ, AMB	Died
5	8 (2007)	Korea	Neonate F	Premature, CVC	Blood	No antifungal	Survived
6	20 (2007)	Spain	82/F	DM, CRF	Blood	AMB	Died
7	21 (2008)	–	38/F	AML, Hemochromatosis	Blood	FCZ, AMB	Survived
8	23 (2008)	India	Neonate M	Prematurity	Blood	FCZ, AMB	Died
9	22 (2009)	Brazil	3/F	Ascaris, Peritonitis, CVC, Multiple ABX	Blood	Liposomal AMB	Survived
10	11 (2009)	Taiwan	71/M	DM, TinaPedis, Cellulitis	Blood	FCZ, AMB	Survived
11	4 (2009)	Taiwan	55/M	Alcoholic, Hepatitis, Duodenal ulcer, CVC	Blood	FCZ, Caspofungin	Survived
12	24 (2009)	India	38/F	HIV, CD4-7%, Absolute CD4 −111 cell/mm^3	Oral Swab	FCZ	–
13	3 (2010)	USA	34/M	Asthma, Alcohol, Thrombophlebitis	Blood	Micafungin	Survived
14	Present case (2014)	Saudi Arabia (SA)	9/M	CRF, Peritoneal dialysis, Cyclosporine	Peritoneal fluid	FCZ, AMB, Caspofungin	Survived

PM: pacemaker, DM: diabetes mellitus, CVC: central venous catheter, SCC: Squamous cell carcinoma, VP: ventriculoperitoneal shunt, KA: ketoacidosis, CRF: chronic renal failure, ALL: acute lymphoblastic leukaemia, CML: chronic lymphoblastic leukaemia, HCV: hepatitis C virus, PAC: porta catheter, AMB: amphotericin B, FCZ: fluconazol, ABX: antibiotics, TOF: tetralogy of fallot, TB: tuberculosis, and NEC: necrotizing enterocolitis. Table adapted and modified with permission received from the publisher. Mycopathologia (2010) 170:223–228.

previously diagnosed with focal segmental glomerulonephritis based on a renal biopsy prior to his presentation to us. He was receiving 50 mg of cyclosporine every 12 h, 12.5 mg of captopril every 8 h, and 100 mg of labetalol every 12 h and was discharged for follow up in the outpatient clinic.

Seven months ago, the patient was admitted to a local hospital complaining of cough, vomiting, decreased oral intake, abdominal pain, and generalized oedema with a fever up to 39 °C for a duration of 1 month. Peritoneal dialysis was started every other day, but his abdominal pain was severe and did not improve. A peritoneal fluid culture was obtained, and a *Candida* infection was identified; the patient was started on 100 mg of intravenous fluconazole once daily and was transferred to our institute for peritoneal catheter removal.

On admission to our centre, his temperature was 36.6 °C, and he had a pulse rate of 118 bpm, an RR of 36/min, a Bp of 128/96 mmHg, a Wt of 19.4 Kg, an Ht 120 cm, and a BMI of 13.5 kg/m^2. During hospitalization, his fever spiked to 39.4 °C; he was started on pipracillin–tazobactam (tazocin) and vancomycin in addition to fluconazole, and the peritoneal catheter was removed. Investigations found a WBC count of 17, a PLT count of 465, and a negative blood culture. The chest X-ray showed bilateral small pleural effusion. Peritoneal fluid was collected for culture and sensitivity (c/s). The fluconazole was changed to ambisome, and the peritoneal dialysis was switched to haemodialysis (HD) through a femoral line inserted in the left side. The organism was identified as yeast, and the ambisome was changed to caspofungin. An abdominal ultrasound was performed and found no evidence of collection, although the kidneys were small and echogenic with multiple cortical cysts related to end-stage renal disease, without hepatic or splenic lesions, and diffuse mild small bowel thickening, suggestive of enteritis. The final identification of the yeast isolated from the peritoneal fluid was *K. ohmeri* (previously known as *Pichia ohmeri*).

Our microbiology lab detected *K. ohmeri* using the Vitek 2 biosysteme and sub culturing on sabouraud dextrose agar, which showed white rough colonies of yeast (Figure 1) that failed to grow in mycological agar. Additionally, the sample turned blue-grey in response to *CHROMagar Candida chromogenic agar growth medium. The API 20C assay was used for confirmation.*

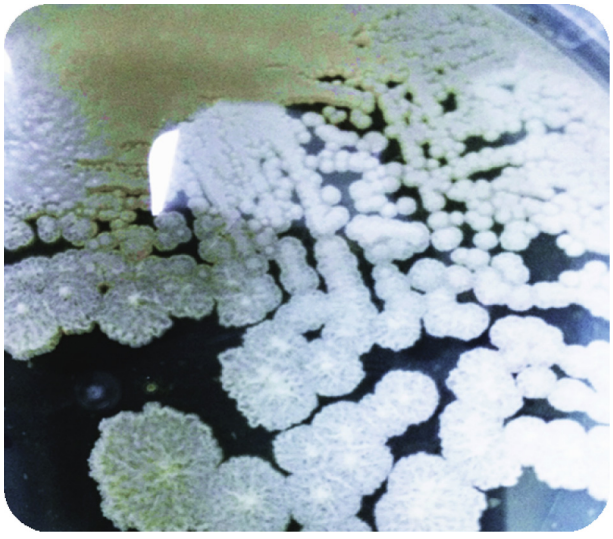

Figure 1: Growth of *K. ohmeri* in sabouraud dextrose agar, KFSH&RC.

Table 2: The *Kodamaea* (*Pichia*) *ohmeri* susceptibility test (*KFSH&RC*).

Amphotericin B	0.5 mg/l
Anidulafungin	0.12
Micafungin	0.12
Caspofungin	0.5
Voriconazol	0.03
Itraconazol	0.06
Fluconazol	4
Posaconazol	0.03
5 Flurocytosine	≤0.06

The susceptibility test (Table 2) using micro-broth dilution showed that the organism was susceptible to caspofungin and amphotericin B, and according to the CLSI definition of the break point,[5] it was susceptible to all other antifungal therapy.

The patient's general condition improved, and the patient became a febrile. We continued treatment with caspofungin for three weeks and achieved complete recovery.

Discussion

Kodamaea ohmeri yeast has been described in the literature and mostly causes blood stream infections (Table 1).[6,9,12,14–19] Nearly all of the reported cases are associated with risk factors such as diabetes mellitus, neutropenia, malignancy, catheter-related infections such as those of the central venous catheter or the ventriculo-peritoneal-shunt, and peritoneal dialysis (Table 1).[6,9,10,12–19] Our patient presented with peritonitis secondary to peritoneal dialysis. Additionally, the patient was an immune-compromised child on cyclosporine. As the child was treated in a local hospital, their laboratory results did not identify the yeast or provide treatment such as fluconazol. After the removal of the peritoneal catheter, the antifungal was changed from fluconazol to ambisome, and then caspofungin was started based on the identification of the fungus as a yeast. The general condition of the child improved in regard to his abdominal pain and the disappearance of the fever.

The patient completed three weeks of caspofungin, achieved a complete recovery from his peritonitis and was discharged home.

The *K. ohmeri* yeast was first discovered in an immunocompromised patient in 1998.[6] It is an emerging opportunistic pathogen and should not be considered to be a contamination.[7] There is limited data on the risk factors, laboratory analyses, treatment and prevention of *K. ohmeri* infection.[7] A literature review (Table 1)[6,9,10,12–19] indicated that *K. ohmeri* can be resistant to fluconazole, is associated with poor outcomes and treatment with amphotericin B is optimal. However, our case was successfully managed with caspofungin, which was selected because the patient was still symptomatic (including fever and abdominal pain) despite ambisome therapy for a week. Until now, no standard therapeutic regimen had been identified.

In 2009, a 55-year-old Taiwanese man presented with alcoholic hepatitis and peptic ulcer disease with CVC for parental nutrition, and he developed fungemia, which was successfully treated with the removal of the CVC and caspofungin for a total of 2 weeks.[4]

The identification of *K. ohmeri* yeast in the laboratory is possible with the commercially available Vitek and API 20C assays, but the API 20C is considered the gold standard for the identification of *K. ohmeri* and other yeast species.[8] CHROMagar Candida chromogenic agar growth medium is a useful tool for the detection of yeast species.[8] This tool is based on the growth of coloured colonies in which the colour changed from pink to blue.[8–10]

In children, three cases have been reported in premature neonates, and six additional cases have been reported among patients aged 8 months to 14 years old, indicating that there is no age specification for this infection but that the underlying risk factors play a major role in acquiring the infection (Table 1).[8,9,18] The source of the infection in our case was most likely the dialysis fluid. Seventy percent of the reported cases were catheter related, indicating that a breakdown of the skin mucosal barrier is a significant risk factor.[11] The duration of the treatment is varied in the previous reports, as there is no recognized number of cases yet available. The duration may be longer in cases of endocarditis with prophylactic antifungal therapy.[14]

Conclusion

Kodamaea ohmeri is an emerging fatal opportunistic fungal infection that should be specifically included in the differential diagnoses of immune-compromised patients and those with indwelling catheters. A high index of suspicion and early diagnosis and treatment are necessary for optimal recovery.

Author contribution

All authors approved the final manuscript.

Conflict of interest

All authors have no conflicts of interest to declare.

Funding

None.

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