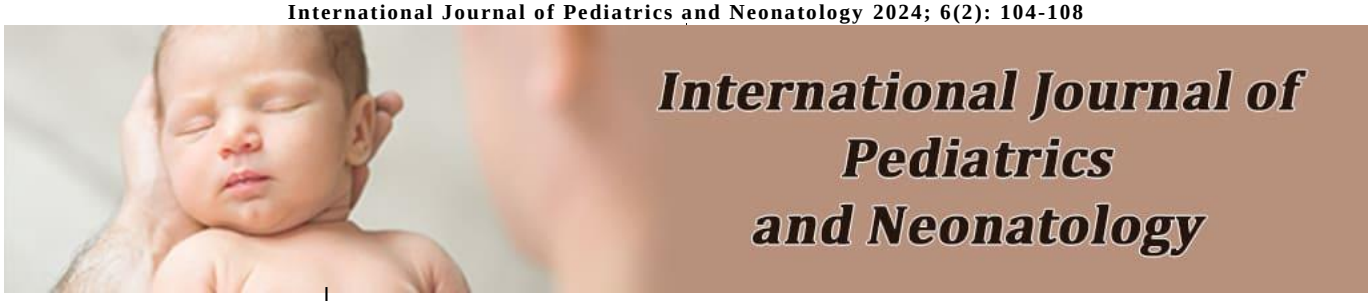

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A case series of *Kodamaea ohmeri* sepsis with long term outcomes in neonates

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Abstract
Kodamaea ohmeri (*K. ohmeri*) is a fungal pathogen causing severe infections in humans. We had six cases of neonatal infections caused by *Kodamaea ohmeri*, which has otherwise been rare. Two of the babies were born by normal vaginal route and four by cesarean section. All of them developed clinical symptoms and apparent worsening within 48 hours of life. Blood culture showed growth of *K. ohmeri* in all, with one of them developing meningitis. Three babies were treated with fluconazole, and two required amphotericin B, while one baby had a rapid deterioration and death. Three neonates had seizures with encephalopathy and required antiepileptics, who later developed neurological sequelae. *Kodamaea ohmeri* is an emerging pathogen in NICU with devastating outcomes. The utmost care and judicious treatment are required to overcome the deleterious effect caused by this agent.

Keywords: Neonate, sepsis, Kodamaea, ohmeri, fungal, fluconazole

Introduction
Kodamaea ohmeri, previously known as *Yamazadyma ohmeri*, is an ascomycetous yeast that belongs to the sachromycetaceae family [1]. The first authentic case of *K. ohmeri* fungemia was reported in an adult [2]. Since then, this species has emerged as a critical opportunistic agent of fungemia, especially in immunocompromised patients [3]. More recently, this fungus has emerged as an opportunistic infectious agent in NICU, primarily reported in very low birth weight neonates after a prolonged NICU stay. There have not been reports about this organism causing sepsis among term neonates or as a cause of early-onset neonatal sepsis (EONS). We report a case series of six neonatal infections caused by *Kodamaea ohmeri* from a tertiary hospital in Kerala, India. The institute ethics committee approved the study.

Case Report
Case 1
A term, female neonate, with a birth weight of 3 kilograms (Kgs), was delivered in an outside hospital by emergency cesarean section for fetal distress. Mother was a primigravidae who had regular antenatal visits. The baby cried at birth, had an Apgar score of 9 at 5 minutes of life, and was breastfed within the first hour of life. At 12 hours of life, she had respiratory distress, with tachypnea and retractions. She was referred to our unit as the symptoms worsened. On arrival, the baby had a saturation of 92% in room air, respiratory rate (RR) of 72 per minute (Min), severe intercostal retractions, and an audible grunt (Distress score of 5/10 as per Downes scoring). She was admitted to NICU and initiated on Continuous positive airway pressure (CPAP) with FiO_2 of 30% and positive end-expiratory pressure (PEEP) of 4. Saturation improved to 98% after starting on CPAP support. Fluid support was given with 10% dextrose. Initial blood gas showed respiratory acidosis (pH - 7.15, pCO_2- 68 mmHg, PaO_2- 54 mmHg, HCO_3- 18 mmHg and base deficit (BD) of-18 meq/l). X-ray imaging showed unilateral homogenous opacification on the right side, suggesting congenital pneumonia. With blood count showing leucocytosis (21000, neutrophils- 82%) and elevated C-reactive protein (CRP) of 35, we started her on intravenous (IV) antibiotics per our unit protocol (Ciprofloxacin 50 mg/kg/day and amikacin 15 mg/kg/day, respectively after sending blood culture).

Within the next 6 hours, respiratory distress aggravated, with a downe score of 8/10, saturation falling to 82%, and blood gas showing worsening acidosis (pH- 7, PCO_2 70 mmHg, and HCO_3 14 mmHg). She required ventilatory support in Synchronised intermittent mandatory ventilation (SIMV) mode with pressure support (PS). With an initial setting of FiO_2- 65%, RR- 55/min, peak inspiratory pressure (PIP) - 18, and peak end-expiratory pressure (PEEP) of 5, she showed improvement in clinical symptoms with saturation rising to 95%. After two hours, she again had desaturation (87%), tachypnea, and a requirement of increment in FiO_2 to 90%. With the baby requiring significantly high FiO_2, we did a two-dimensional (2D) echo, and the findings were consistent with persistent pulmonary hypertension (PPHN) - right ventricular hypertrophy (RVH), deviation of the inter ventricular septum (IVS) to the left, tricuspid regurgitation (TR), and bidirectional shunting at the patent foramen ovale (PFO). PPHN was managed with supportive care, sildenafil (IV loading dose of 4mg/kg, followed by a continuous infusion), and ionotropic agent dobutamine at 10 microgram /kilogram/minute (mcg/kg/min). Over the next 24 hours, the baby developed sclerema. Considering the clinical worsening and a further rise in CRP to 110, we changed the antibiotics to vancomycin and meropenem. Since the baby continued to have desaturations on the conventional ventilation mode, we changed the support to high-frequency oscillatory ventilation (HFOV). Blood culture, after 48 hours, reported the growth of *Kodamaea ohmeri*, sensitive to fluconazole. Upon starting the baby on IV fluconazole (6mg/kg/day), she had a dramatic improvement in the clinical symptoms. We could gradually taper the ventilatory support and extubate her to CPAP within 48 hours of starting fluconazole. Cerebrospinal fluid (CSF) was sterile. IV antibiotics were discontinued, and dobutamine was tapered and stopped. Sildenafil was continued orally for a week. She was weaned off from CPAP after 48 hours, initiated on breastfeed, and shifted to the mother's side on day 8 of admission. After completing the antifungal course of 14 days, we discharged her. Repeat 2D echo done at one month of life was normal. She has been on regular follow-up, and at three years of age, she has attained age-appropriate milestones and is neurologically normal.

Case 2

An outborn female baby with a birth weight of 2.7 kgs, was referred to our unit at 24 hours of life due to lethargy, poor feeding, and seizure of tonic posturing type. She was born at 38 weeks of gestation, by vaginal route to a second gravidae mother, who had regular antenatal visits. The baby cried soon after birth, and after routine newborn care, was breastfed and shifted to the mother's side. She developed the symptoms after 12 hours of birth and was observed in the NICU there. Once the baby had a seizure episode, she was referred to our unit. On arrival, the baby was in shock, with poor peripheral perfusion, prolonged capillary refill time (CFT- > 3 seconds), and mean blood pressure of 18 mmHg. She had features of acute renal failure with oliguria (o.4 ml/kg/hour) and a creatinine of 1.4, recurrent seizures, and severe encephalopathy. We could reverse the shock with intravenous fluids (Normal saline bolus of 20 ml/kg followed by 10% dextrose as maintenance) and ionotropic (Dobutamine at 10 mcg/kg/minute) support. With the initial septic workup being positive (Whole blood count (WBC)-18000 cells/cu mm, neutrophils- 82%, CRP- 28), antibiotics (Ciprofloxacin and amikacin) were started. Recurrent seizures required phenobarbitone (loading dose of 40 mg/kg and a maintenance dose of 5 mg/kg/day), phenytoin (loading dose of 40 mg/kg and a maintenance dose of 5 mg/kg/day), and levetiracetam (Loading dose of 20 mg/kg and a maintenance dose of 10 mg/kg/day) for control. Electroencephalogram (EEG) showed abnormality with epileptiform discharges arising from the left temporal region. Blood culture grew *Kodamaea ohmeri* after 48 hours, sensitive to Amphotericin B and resistant to fluconazole. With the cerebrospinal fluid (CSF) analysis suggesting meningitis (total CSF count 250 cells/cumm, CSF protein 250, and sugar), magnetic resonance imaging (MRI) of the brain was done which showed diffuse cerebritis. After starting the baby on IV Amphotericin B, her clinical status gradually improved. She was restarted on direct breastfeeding after 48 hours of starting antifungal treatment and could be shifted to the mother's side on day 6 of admission. Seizures did not recur, and the anti-epileptics could be tapered and stopped in the first month of life. After completing 21 days of antifungal course, she was discharged. Encephalopathy had improved, and the baby was accepting feeds well at the time of discharge. A repeat EEG after one month was normal. She was on regular follow-up till five months of age when she had infantile spasms. The spasms progressed on to West syndrome. After starting the child on adrenocorticotropic hormone (ACTH), spasms could be controlled. At two years of age, the child has global developmental delay and spastic paraparesis.

Case 3

This was an outborn male baby, delivered by cesarean section for fetal distress. The mother was a primigravidae, and she had fever one week prior to delivery and she was treated with oral antibiotics (Amoxycillin). Baby had a birth weight of 2.7 kgs with an Apgar score of 9 at 5 minutes of life. He was breastfed immediately. After an uneventful postnatal course of 2 days, he had an episode of hypoglycemia (GRBS- low) and tonic posturing on day 3 of life. The baby was referred to our unit after a dose of midazolam (0.1 mg/kg) and a 10% dextrose bolus (2mg/kg). On admission, heel prick glucose level was 27 gram/decilitre (g/dl), and we started the baby on a glucose infusion rate (GIR) of 6mg/kg/mt. The baby continued to have seizure episodes that required phenobarbitone (loading dose of 20mg/kg followed by 5 mg/kg/day) for control. He also had encephalopathy following seizures. As part of the workup for hypoglycemia, blood culture showed *Kodamaea ohmeri* after 48 hours. The initial antifungal fluconazole had to be changed to Amphotericin B as per the sensitivity report, received after 24 hours. CSF study was suggestive of meningitis (Cells 110, polymorphs 60, lymphocytes 40, protein 120 mg/dl, and sugar 20 mg/dl). The baby received a total of 3 weeks of intravenous Amphotericin B. He had mild encephalopathy at the time of discharge. Antiepileptic was tapered and stopped, after the EEG showed a normal study at 45 days of life. This baby has been on regular follow-up on a monthly basis. He had hypertonia of the upper limb muscles at six months of life (Amyl Teesons angle). No further seizures were noted. Currently, at 18 months of age, the child has developed spastic quadriparesis with global developmental delay.

Case 4

This was an outborn baby born to a hypothyroid mother on thyroid supplementation (50 mcg daily), with ambiguous genitalia (Micropenis, bilateral undescended

testis, and poorly formed scrotum). The mother underwent an emergency caesarean section for abruption of the placenta. The baby did not require any resuscitation at the time of birth (Apgar- 9 at 5 minutes) and has been otherwise well. On day 2 of life, the baby was referred to our unit for lethargy and poor feeding. Blood workup suggested probable sepsis (Total count - 18000 cells/cumm, platelet - 1.4 lakh/cumm, C reactive protein - 15 mg/dl), and we started the baby on injection ciprofloxacin and amikacin as per unit protocol. Ambiguous genitalia workup was also carried out simultaneously. After 48 hours of starting the antibiotic, the baby was asymptomatic and taking feeds well. Neonatal jaundice, which developed on day 3 of life, required phototherapy for 48 hours. With a repeat blood count being normal (Total count - 8000 cells/cumm, platelet - 1.8 lakh/cumm, C reactive protein - <6 mg/dl) and blood culture after 48 hours being sterile, we stopped the antibiotics and discharged the baby back to the referring hospital on day 5 of life. To our surprise, blood showed growth of *Kodamaea ohmeri* after five days of culture. After obtaining a normal CSF study (3 cells/cumm, protein- 20 mg/dl, sugar- 50 mg/dl), a 14 days course of oral fluconazole was given for the baby. The baby has been doing well upon follow-up. The child is currently 18 months of age and is neurologically normal. Karyotyping showed a 46 XY male chromosomal pattern with normal internal and external genitalia.

Case 5

A female outborn baby presented to our unit on day two of life with a history of lethargy, poor feeding, and seizure-tonic posturing of upper limbs. She was born by normal vaginal route, with a good Apgar score (9 at 5 minutes) to a primigravida mother, who had no significant antenatal complaints. The baby had hypoxia (Saturation of 85% in room air and 100% with 5 liters of oxygen) and shock (poor peripheral perfusion, CFT- > 3 seconds, and mean blood pressure of 18 mmHg) on admission. Blood gas showed metabolic acidosis (pH- 7.2, PC02-29, HCO_3- 12, and base deficit of -17). Initial stabilization of the baby was done with a normal saline bolus of 10 ml/ kg, oxygen support (5 litres), IV fluid (Isolyte P), and vasopressor pressor (Dobutamine at 10 mcg/kg/minute) support. She was started on antibiotics ciprofloxacin (50 mg/kg/day) and amikacin (15 mg/kg/day) with blood work up suggesting sepsis (total count - 22000 cells/cumm, platelet - 25000/cumm, C reactive protein - 36 mg/dl). After a loading dose of IV phenobarbitone(20 mg/kg), the seizure was controlled and did not recur. Thrombocytopenia required a platelet transfusion of 15 ml/kg. Blood culture showed growth of *Kodamaea ohmeri* after 48 hours, and we started her on injection fluconazole. CSF study was normal (1 cell (lymphocyte), sugar- 58 mg/dl and protein 68 mg/dl). She was shifted to the mother's side and was active and breastfeeding well. We discharged the baby after completing 14 days of antifungals. The baby has been doing well on follow-up, with a normal neurological examination at one year of age.

Case 6

A preterm (34 weeks) female baby with a birth weight of 1.5 kgs was referred to our unit on day 3 of life, with complaints of respiratory distress. The mother had pregnancy-induced hypertension, and early termination was done at 34 weeks as she developed pre-eclampsia. Born by the vaginal route, the baby cried soon after birth with an Apgar score of 8 at 5 minutes of life. However, she had respiratory distress in the first hour of life (Respiratory rate 78/minute, lower chest retractions and grunt - Silvermann Anderson score of 4) and was admitted to the NICU for further care and treatment. She was given oxygen support through nasal prongs (2 liters/minute) and was started on injection cefotaxime. The baby's clinical condition worsened after 48 hours of life and hence was referred to our unit. The baby was in respiratory failure and hypotensive shock on admission, with a saturation of 55% on 12 liters of oxygen, severe chest retractions, audible grunt, poor peripheral perfusion, CFT > 3 seconds, and a mean BP of 15 mmHg. She was immediately intubated and given ventilatory support (SIMV mode with FiO_2- 100%, RR- 55, TV- 10 ml, and PEEP 5). The initial blood gas showed severe acidosis (pH- 6.9, PCO_2- 62, HCO_3- 10, and base deficit -20). After an initial bolus of 10 ml/kg of normal saline, IV fluid (Isolyte P), and pressor support with dobutamine at 10 mcg/kg/min was continued. After sending a blood culture, she was given meropenem. Unfortunately, the baby continued to deteriorate further with a repeat blood gas after one hour, showing further worsening (pH- 6.0, PCO_2- 70, HCO_3- 10, and base deficit -21). Despite our continued efforts, she succumbed to illness within 5 hours of admission. The blood culture on admission showed *Kodamaea ohmeri*, sensitive to amphotericin B, after four days.

Discussion

Kodamaea ohmeri is a rare clinical isolate that has recently become known to cause various human infections. This yeast, commonly used in the food industry for its fermentation properties in pickles, has been implicated in causing fungemia, peritonitis, and wound infection over the years, mostly in adults [4, 5]. Infections due to *K. ohmeri* occur in patients of different age groups and immune profiles. The patient group includes neonates and children, immunocompromised cancer patients, patients with other chronic illnesses such as diabetes, and immunocompetent patients [6]. In the pediatric population, this fungal pathogen is known to cause severe infections [7, 8]. Very few cases of neonatal K Ohmeri infections has been reported worldwide.

The first K Ohmeri infection case in a neonate born prematurely was reported in 2006 [9]. Lee *et al*., in 2007, reported a case of a premature neonate affected with Kodamea Ohmeri sepsis wherein they identified a central venous catheter as the source of infection [10]. The first published case of neonatal K Ohmeri infection in India was in a premature neonate who had received broad-spectrum antibiotics through a central line [11]. In 2011, a late-onset fungal infection with K Ohmeri in a premature neonate was published. This neonate had received two courses of broad-spectrum antibiotics for bacterial sepsis, which was stated as the possible source of fungal infection [12]. In the published case reports on *K. ohmeri* in neonates, almost all of them had risk factors of being premature, presence of invasive devices (e.g., umbilical catheter), and prolonged antibiotic usage(6-8). Contrary to this, all the neonates in our series who had K Ohmeri infection acquired it in the perinatal period, and none had such obvious risk factors.

All the six neonates in our study presented in the early neonatal period with varying symptoms. Biswal *et al.* reported a case of K Ohmeri in the first week of life in a term neonate whose mother also had the same organism identified on the vaginal swab [6]. In another case report of K Ohmeri in 33 weeks preterm neonate, they determined the organism on

day 18 of life after receiving multiple courses of antibiotics [13]. A similar finding of late expression of K Ohmeri was noted in other case reports as well [9-12]. It is very unusual for a neonate to present with an early onset fungal sepsis unless there is an underlying antenatal or perinatal risk factor. All the neonates in this case series were outborn and thus we had limited information with regards to the maternal risk factors. The infection may have been aquired during the intraparum or peripartum period, but we could not aquire any substantial information from the referred hospitals to corroborate our postulation.

Necrotizing enterocolitis and lethargy were the predominant presenting symptoms in the first case of neonatal K Ohmeri sepsis [9]. A similar presentation with feed intolerance was seen in another preterm of 33 weeks [13]. Respiratory distress and shock have also been reported in two cases [11, 12]. Lethargy and poor feeding were consistent in our patients, while other clinical features were varying. Three neonates had seizures which required anti epileptics for control.

We use Bactec method of culture for identification of organisms, which has a high sensitivity and specificity [14]. When a case of neonatal sepsis is suspected, we start the baby on ciprofloxacin and amikacin, as this combination had a good coverage over the predominant organsism isolated from our unit [15]. Once the blood culture report is available, usually by 48 hours, we decide upon the further course of antibiotics. We do not follow the practice of prophylactic antifungals for neonates at risk due to the low incidence of fungal sepsis. Since there were no risk factors for fungal sepsis in these neonates, we started antifungals only after receiving the blood culture reports. Although Amphotericin B was the main line of treatment in the previous cases due to the clinical course of the neonates [6, 9, 11, 12], voriconazole and echinocandins, such as caspofungin and micafungin, also showed results against *K. ohmeri (16, 17)*. In our study, we could successfully treat three of the cases with fluconazole. In one neonate, we had to complete the course with oral fluconazole because of the late culture growth of K Ohmeri. Amphotericin B had to be used in two neonates due to the severity of illness and the sensitivity pattern. To the best of our knowledge, this is the first case series showing successful treatment with fluconazole monotherapy in such neonates.

There are no published reports on long term follow up of the neonates who were treated for K Ohmeri. We routinely follow a multidisciplinary approach for the NICU graduates to assess their growth and well-being. We use DASII [18] (Performed by pediatrics and neonatology team) and Bayley scoring system [19] (by psychologist) for evaluating the development of the child on follow up. Out of the five neonates who survived, three had neonatal seizures. Two of them required more than one anti-epileptic for seizure control and had encephalopathy, while the seizure in the third neonate was controlled with a single loading dose of phenobarbitone. Neonates who had multiple seizures and encephalopathy during the course of illness had significant neurodevelopmental sequelae. It appears that the initial presentation with seizure and encephalopathy could have a poor long-term prognosis.

In a neonate, who has predisposing risk factors like prolonged antibiotic usage, in-dwelling catheter and total parenteral nutrition for more than 2 weeks, there is always a danger of fungal sepsis. When a neonate presents with sepsis that is not responding to the usual antibiotics, we usually do not presume fungal sepsis and start treatment until culture results come in. But considering the increasing cases of fungal sepsis, it would be worthwhile to consider a prophylactic line of management for it.

Conclusion

Kodamaea ohmeri is an emerging pathogen in NICU with potentially devastating outcomes. The mode of presentation could be varying and quite possible even in term/near-term good weight babies in the initial days of life. Treatment with fluconazole has a good success rate and initial presentation with severe CNS symptoms seems to be a poor prognostic factor.

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Statement of ethics

The institute ethics committee approved the study and the necessary consent was obtained from the parents of the new borns.

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Author contributions

Dr. Mohamed Reshad collected the patient data, did the data entry, and wrote the manuscript. Dr. Binesh Balachandran planned the study, collected the data, reviewed and edited the manuscript. Dr. Moideen Sharief and Dr Rajeswari helped in collection of data, and writing the manuscript.

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