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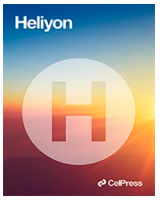

Rhodococcus equi bacteremia with necrotizing pneumonia and brain abscess in a newly diagnosed HIV patient in Saudi Arabia: A case report and review of literature

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ABSTRACT

Rhodococcus equi is a Gram-positive coccobacillus that falls within the category of aerobic actinomycetes. The *Rhodococcus* genus belongs to the nocardioform bacteria group. This microorganism has been found in various settings, including natural environments, animals, and particularly in individuals with compromised immune systems, such as those living with HIV. Notably, there is an increasing number of reports concerning *R. equi* infections in transplant recipients and even individuals with a normally functioning immune system.

Traditionally, *R. equi* has been primarily associated with pulmonary infections, but there is a growing body of evidence documenting its involvement in extrapulmonary infections. In this report, we present a case involving a newly diagnosed HIV patient who experienced *R. equi* -induced necrotizing pneumonia, bacteremia, and a brain abscess in newly diagnosed HIV patient. It is important to note that a direct Gram stain may potentially lead to misclassification of such microorganisms as contaminants. Microbiologists should therefore prioritize the careful examination of colony morphology, biochemical reactions, and consider the limitations of automated machine databases. Furthermore, they should correlate their identification findings with clinical data to ensure optimal patient care and management, especially in the context of an immunocompromised state.

1. Introduction

Rhodococcus equi is a Gram-positive coccobacillus, belonging to the group of aerobic actinomycetes. Originally isolated from a human patient suffering from pulmonary infection and underlying autoimmune hepatitis while receiving steroid treatment in 1967 (Golub et al., 1967), it has since gained recognition as a zoonotic pathogen affecting foals. Over the years, the incidence of *R. equi* infections has risen among various patient groups, including those with HIV [1],transplant patients [2], and individuals with intact

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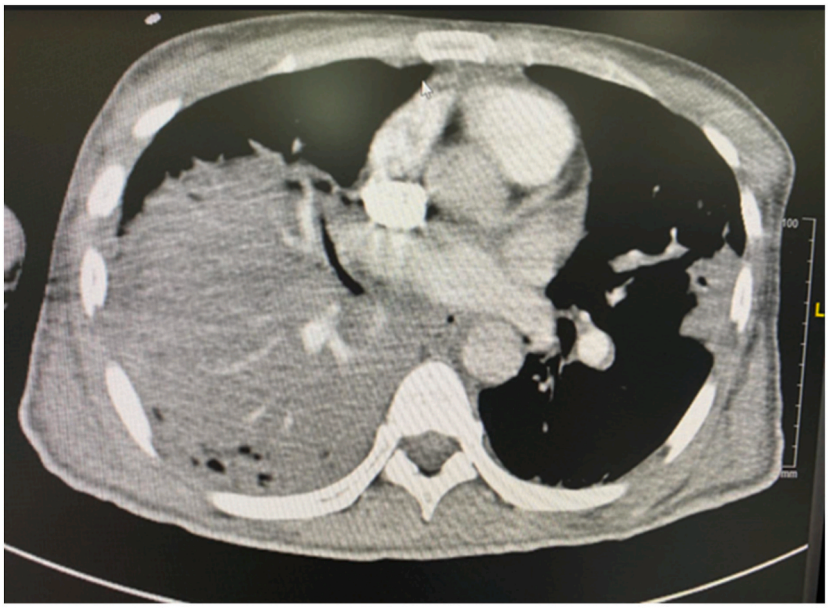
immune systems [3].While its primary target is the respiratory system, *R. equi* has demonstrated the capacity to disseminate to other bodily sites, including the brain, skin, soft tissues, bones, and bloodstream (Herath et al., 2013; Topino et al., 2010). Despite the absence of established treatment guidelines for this pathogen, its documented ability for intracellular multiplication and recurrence necessitates prolonged and combination antimicrobial therapy. In immunocompromised individuals, it may even require lifelong prophylaxis [4]. In this case report, we present the first documented instance of *R. equi* infection in Saudi Arabia (SA). The case involved a patient with pulmonary infection and bacteremia, which subsequently led to brain abscess formation. Furthermore, we conduct a comprehensive review of rhodococcal brain infections documented over the last decade. We also delve into the potential



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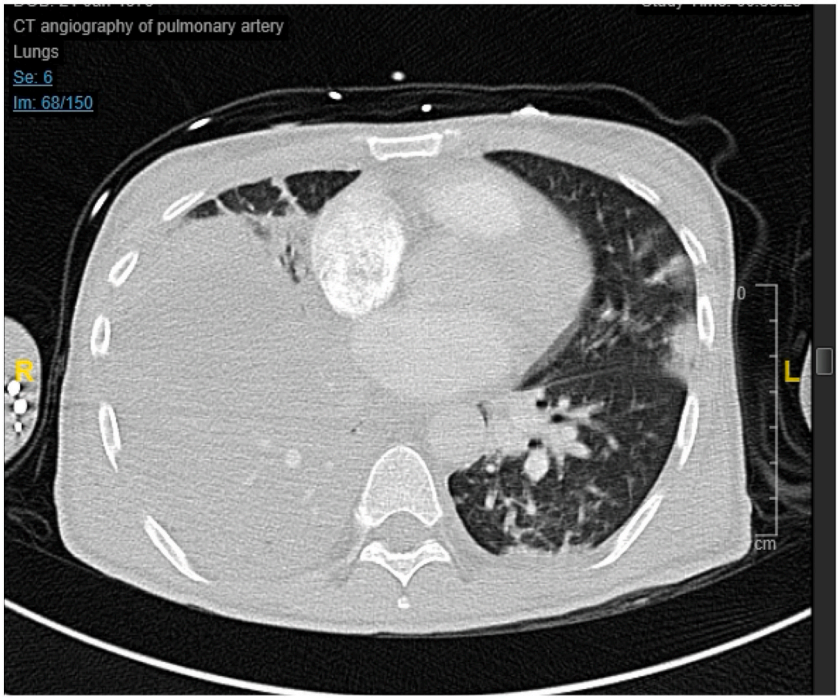

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Fig. 1. (A, B): CT Angiography (Day of admission) No evidence of pulmonary embolism, The right lower lobe demonstrates a large area of non-enhancement within the lung parenchyma; this is likely liquefication related to infection versus necrosis.

causes of laboratory misidentification and, consequently, the mismanagement of these pathogens.

2. Case presentation

2.1. Clinical data

We documented the case of a 41-year-old Saudi male who was unemployed and originally from the Eastern province of Saudi Arabia. He was brought to the emergency department due to lethargy. His initial presentation included a fever, which had reached 38.5 °C, and a progressively worsening chronic dry cough that was accompanied by shortness of breath. This cough significantly limited his physical activity, even at distances shorter than usual. Additionally, he reported night sweats and a notable unintentional weight loss of approximately 7 kg over the past two months. The patient had a history of homosexuality, smoking, and kept cats as pets at home.Upon his arrival at the emergency room, the patient was febrile, with a temperature of 38.9 °C. His vital signs were as follows: a heart rate of 128 beats per minute, a respiratory rate of 28 breaths per minute, blood pressure measuring 108/65 mmHg, and an oxygen saturation level of 88 % on room air. Furthermore, he appeared cachectic, displayed muscle wasting, and had poor oral hygiene, including oral thrush. His Glasgow Coma Scale score was 13 out of 15, and his pupils were reactive and equal, with no signs of focal neurological deficits.

During the chest examination, there were no observed limitations in chest wall movement or chest expansion. Percussion revealed dullness over the right lower lung zone, accompanied by reduced tactile vocal fremitus. Chest auscultation revealed decreased breath sounds at the same location, along with some fine basal crepitations and increased vocal resonance.

Other systems examination was unremarkable.

2.1.1. Initial Laboratory Findings

In the initial set of laboratory tests, the patient's white blood cell count was measured at 13.3 k/μl, with 86 % being neutrophils. His hemoglobin level was critically low at 6.1 g/dL, and his platelet count was 86,000/μl, with a reticulocyte count of 0.3 %. Venous blood gases (VBG) indicated a pH of 7.45, a PCO2 of 37 mmHg, a PO2 of 25 mmHg, and an HCO3 of 25 meq/L. Fibrinogen was elevated at 760 mg/dL, while fibrinogen degradation products were at 5.9 mg/dL, and D-Dimer was recorded at 1 μg/ml. Other laboratory findings included a lactic acid level of 3.65 mmol/L, glucose at 93 mg/dL, C-reactive protein levels of 40 mg/dL, an ESR of 79, and Procalcitonin at 1.5 ng/ml. Urea was measured at 36 mg/dL, creatinine at 0.9 mg/dL, and electrolyte levels were within the normal range. Liver function tests were unremarkable, except for an elevated lactate dehydrogenase (LDH) at 206 U/L. The coagulation profile revealed a Prothrombin Time of 16.7 seconds, a Partial Thromboplastin Time of 57.7 seconds, and an International Normalized Ratio of 1.25. Toxicology screening returned positive results for opiates. A SARS-CoV-2 PCR test yielded negative results, while urine analysis was unremarkable. Blood cultures were sent for further evaluation. Upon initial evaluation in the emergency room, a chest X-ray revealed opacification in the right hemithorax, along with pleural effusion. Additionally, the left lung exhibited a mid-wedge-shaped opacification. Further imaging was requested, and a chest CT angiography was subsequently performed (refer to Fig. 1).

2.1.2. Clinical Deterioration and Intensive Care Admission

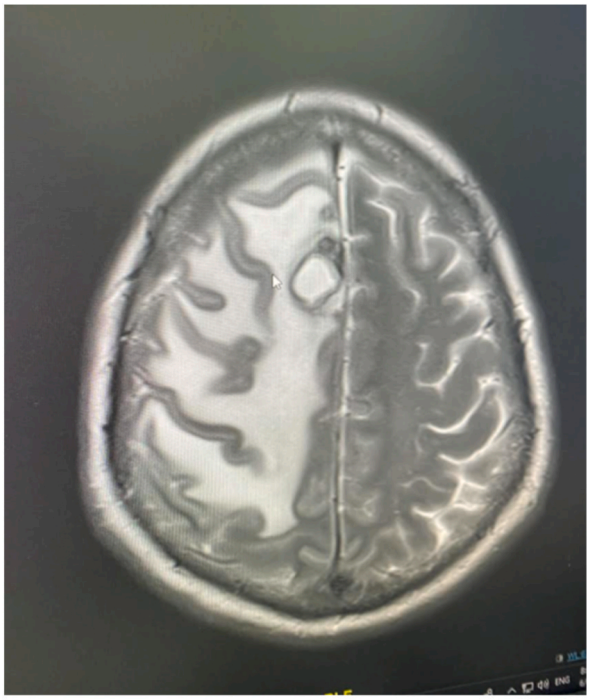
On the same day, during the night, the patient's condition deteriorated, leading to worsening hypoxia and hypotension. This necessitated admission to the intensive care unit (ICU), where he was intubated, and mechanical ventilation was initiated. Empiric treatment was initiated, including meropenem (1g IV every 8 hours), vancomycin (2g IV loading followed by 1g IV every 12 hours), and levofloxacin (750 mg IV once daily). Further laboratory investigations revealed a repeatedly positive HIV test result through fourth-generation immunoassay, which was subsequently confirmed by a positive Western blot test. After two days, blood cultures and bronchoalveolar lavage cultures grew *R. equi*. Repeated blood cultures on the second, third, and fifth day of admission continued to grow the same organism. The absolute CD4 lymphocyte count was measured at 8, and the HIV viral load was not available. Bronchoalveolar lavage (BAL) was sent for acid-fast bacilli (AFB) staining and *Mycobacterium tuberculosis* culture, both of which returned negative results. The patient continued on intravenous antibiotics, including meropenem (1g IV every 8 hours), vancomycin (1g IV every 12 hours) for three weeks, and levofloxacin (750 mg orally) for six weeks.

2.1.3. Resolution of Respiratory Symptoms

Following this treatment regimen, the patient's respiratory symptoms gradually improved, and a subsequent chest X-ray showed regression of the lung lesion. He was discharged with a prescription for daily Bactrim double strength as prophylaxis against Pneumocystis pneumonia (PCP) and was started on antiretroviral therapy (ART) consisting of emtricitabine (200mg), tenofovir alafenamide (25mg), darunavir (800 mg), and cobicistat (150 mg), to be taken daily.

2.1.4. Seizure Episode

Six months later, the patient presented to the emergency room with an episode of loss of consciousness and a seizure lasting for 2 min, characterized by tonic-clonic movements, up-rolling of the eyes, drooling saliva, and biting of the tongue. This episode spontaneously resolved. During this presentation, the patient was afebrile, had a Glasgow Coma Scale (GCS) score of 15 out of 15, exhibited no neck stiffness, and had negative Kernig's and Brudzinski signs. He displayed decreased power in the left upper and lower limbs, graded at 3–4 out of 5 (active against gravity), along with exaggerated reflexes, while sensation remained intact. Laboratory investigations at this time showed a white blood cell count of 13 k/μl, with neutrophils accounting for 80 %. Hemoglobin was measured at 15.7 g/dL, and platelet count was 191*109/μl. The C-reactive protein was 0.4 mg/dL, the ESR was 79 mm/h, urea was 12 mg/dL,



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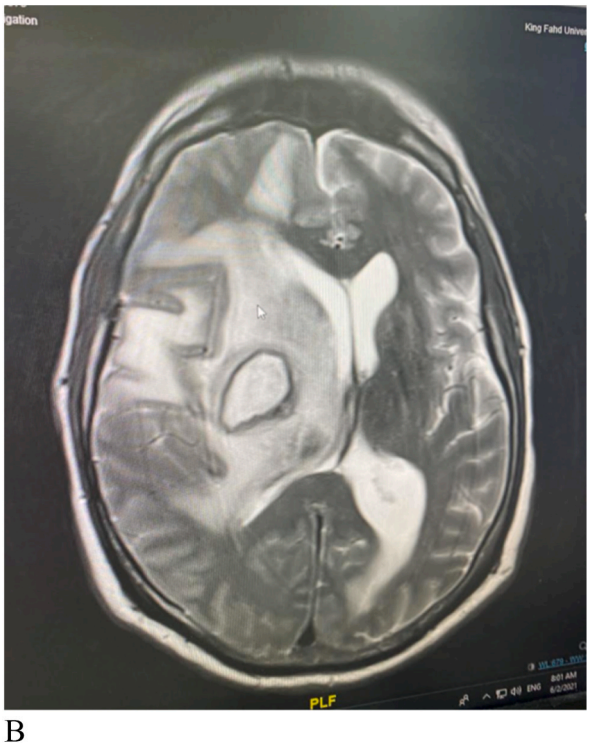

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Fig. 2. (A, B): Brain MRI. Three well defined lesions within the right basal ganglia and medial aspect of the right superior frontal gyrus, measuring: 10.8*1.8*1.4 cm and 0.7*0.8*0.5cm and 1.4*1.4*0.8cm in maximum diameter respectively which display hyperintense T2, hypointense T1 with diffusion restriction and peripheral nodular pattern of enhancement associated with extensive perifocal edema involving right frontotemporal parietal lobe.

creatinine was 0.7 mg/dL, and electrolyte levels were within the normal range. Liver function tests were normal, except for an elevated lactate dehydrogenase (LDH) level of 258 U/L. Toxoplasmosis serology (IgG and IgM) yielded negative results, and urine analysis, as well as blood cultures, returned negative results. A brain CT scan revealed a well-defined hypodense lesion located in the right basal ganglia, accompanied by a hyperdense rim, extending to the right frontal *para*-falci region. To further characterize this lesion, as recommended by the radiologist, a magnetic resonance imaging (MRI) of the brain was conducted. The MRI raised suspicions of several potential differentials (refer to Fig. 2 A, B), including infectious causes such as toxoplasmosis, tuberculosis (TB), or a brain abscess caused by *R. equi*.

2.1.5. Treatment and Clinical Progress

Following a second seizure episode, the patient was initiated on dexamethasone and phenytoin. Treatment with pyrimethamine (50 mg) and sulfadiazine (1000 mg) once daily, alongside leucovorin (25 mg) once daily, was also commenced for a duration of three weeks. Unfortunately, there was no clinical or radiological improvement noted during this period. A repeat CT brain scan with contrast showed progression of the existing lesions and the appearance of new lesions. Considering the patient's history of previous bacteremia and the well-documented propensity of *R. equi* to cause brain abscesses and relapse, a decision was made to restart treatment with

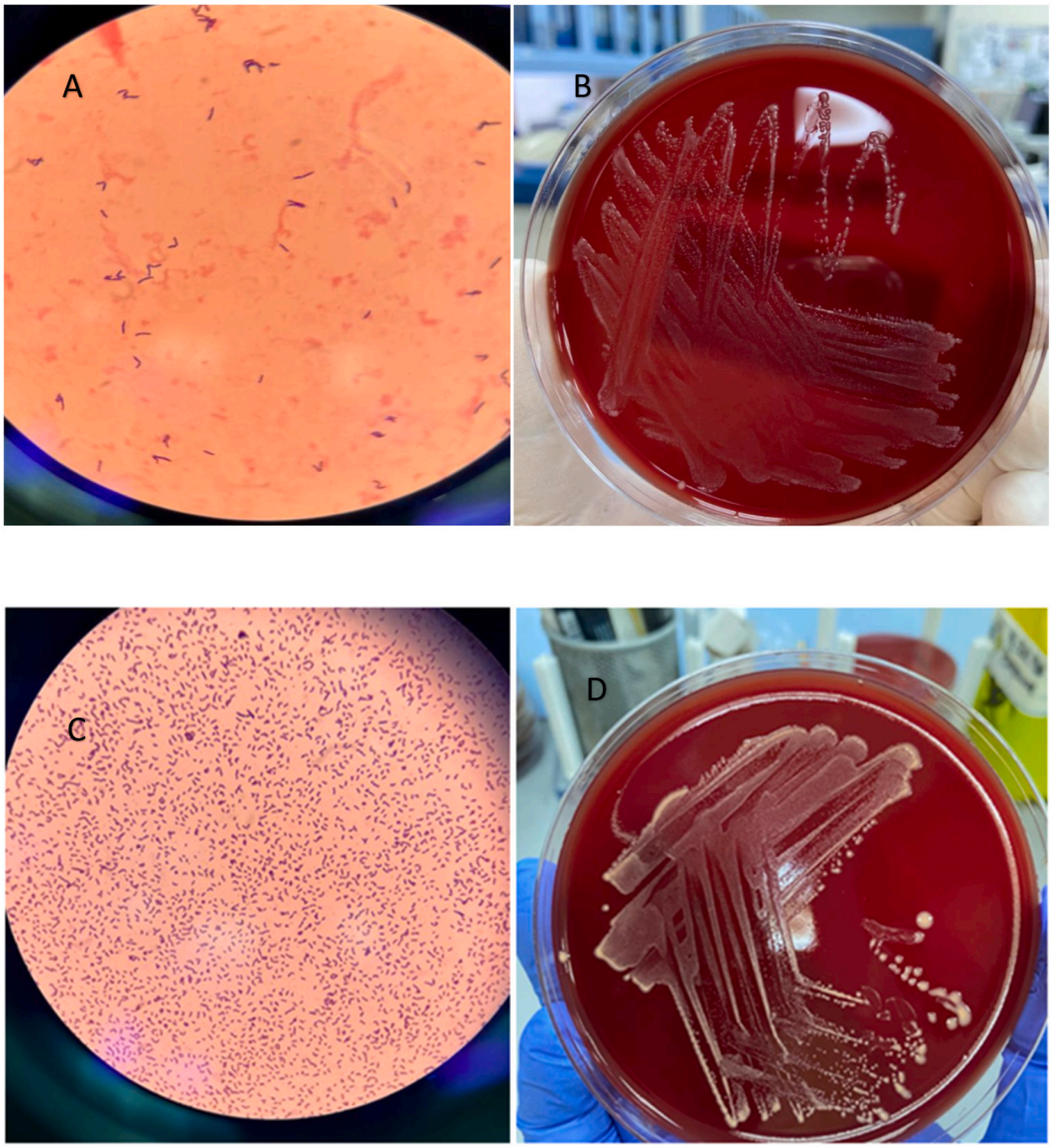

Fig. 3. Microbiological findings of *R. equi*. A: Direct gram staining of flagging blood culture showing Gram positive rods, B: nonhemolytic, creamy colonies on sheep blood agar, C: Gram stain from colonies showed Gram positive coccobacilli, D: After three days of incubation, the salmon pink color of the colonies appeared.

meropenem (1 g IV every 8 hours) and vancomycin (1 g IV every 12 hours) as a possible brain abscess caused by *R. equi*. Although brain biopsy was recommended, it was deemed clinically infeasible by neurosurgeons and, therefore, not performed. After four weeks of this combination therapy, the patient demonstrated improvement, with resolution of left-sided weakness and a reduction in lesion size and brain edema observed on a repeat CT brain scan. Consequently, the patient was discharged with a prescription for oral maintenance therapy, which included Azithromycin (500 mg) once daily and trimethoprim-sulfamethoxazole (double strength) every 12 hours daily for an additional two weeks, followed by Azithromycin (1200 mg) once weekly and trimethoprim-sulfamethoxazole (double strength) once daily.

After eight months of antiretroviral therapy (ART), the patient exhibited clinical improvement, with an absolute CD4 count of 158 cells/μl and an undetectable HIV viral load. A repeat MRI demonstrated near-complete resolution of the previous brain lesions, with some evidence of scarring.

2.2. Microbiological findings

When the blood culture bottle flagged positive on the second day of admission (after 30 hours of incubation in the blood culture system BACT/ALERT), a direct Gram staining procedure revealed Gram-positive rods, often arranged in a V formation (refer to Fig. 3-A). Subsequently, the blood was inoculated onto various agar plates, including sheep blood agar plate (BAP), MacConkey agar, chocolate agar, anaerobic blood agar, and CNA agar. After 24 hours of incubation at 37 °C, nonhemolytic creamy colonies were observed on BAP and chocolate agar (refer to Fig. 3-B), while there was no growth on MacConkey agar. Gram staining of the colonies showed Gram-positive coccobacilli (refer to Fig. 3-C), and Acid fast staining revealed a non-acid-fast result. For organism identification, MALDI-TOF MS (VITEK MS; bioMérieux) was employed, yielding a confidence value of 99.9 % for the identification of the organism as *R. equi*, utilizing the database (version 3.0). On the same day, the transtracheal aerobic culture also yielded colonies with a similar morphology, although no identification was obtained through MALDI-TOF MS. Therefore, it was further processed for identification using VITEK-2 with the ANC card, which indicated *Turicella otitidis* with a 99.9 % probability. However, due to incongruent clinical data, the absence of *R. equi* in the VITEK-2 ANC card database, and a growing suspicion of *R. equi* infection, another attempt at identification using MALDI-TOF MS, conducted by a senior technologist, correctly identified the organism as *R. equi*, with a confidence value of 99.9 %. Additional biochemical reactions, including catalase positivity, DNase negativity, and oxidase negativity, were in line with *R. equi,* as compared to *Turicella* (as presented in Table 1). Furthermore, the isolate exhibited non-motility in the motility test. After three days of incubation, the characteristic salmon pink color of *R. equi* colonies became apparent, confirming its identity (refer to Fig. 3-D). Unfortunately, antibiotic susceptibility testing was not performed due to the unavailability of the broth microdilution method (the recommended method according to CLSI M24-A2) in our laboratory (Woods & Clinical and Laboratory Standards Institute, 2011).

3. Literature review

Cases of *R. equi* infection have predominantly manifested as pulmonary infections. Notably, bacteremia has been more frequently observed in immunocompromised individuals compared to those with intact immune systems, with incidence rates of 80 % and 30 %, respectively [4]. Topino et al. conducted a comprehensive review of reported cases involving HIV patients with rhodococcal infections spanning from 1986 to 2008. Within this extensive dataset of 272 HIV-infected cases, it was found that brain abscesses were documented in only 14 cases, while 7 cases involved bloodstream infections, and 2 were marked by sepsis. In contrast, the overwhelming majority, comprising 97 % of cases, exhibited lung infections, primarily in the form of pneumonia [1]. Extrapulmonary involvement has been observed not only in HIV patients but has also been reported in immunocompetent individuals. A study by Hareth et al., in 2013 examined cases from 1989 to 2011 and identified approximately 26 instances of rhodococcal infections. Among these cases, 17 individuals displayed extrapulmonary infections, and four specifically presented with brain abscesses, all of whom were immunocompetent individuals [3]. In a study conducted by Roda et al., a review of 17 cases of brain abscesses caused by R. equi revealed several noteworthy findings. Predominantly, this condition affected males, with the most frequent site of brain involvement being

Table 1
Clinical and microbiological characteristics of *Rhodococcus equi* and *Turicella otitidis*.References: [4,5,6] *Some reports documented urease negative *Rhodococcus* isolates as in our case (Méndez-Cruz et al., 2022; Prescott, 1991).

	Rhodococcus equi	Turicella otitidis
Clinical presentation	Mostly affect immunocompromised and present in more than 80 % as lung infection	Ear infection, auricular abscess, mastoiditis and bacteremia
Direct Gram stain	Gram positive coccoid to bacillary form	long Gram-positive bacilli
AFB stain	Weakly positive or negative	Negative
Growth requirement	Grow well in nonselective media	Grow well in nonselective media
Colonial morphology	White mucoid or rough that become pigmented with age characteristically salmon-pink in 3–5 days	Whitish, creamy, and convex with entire edges in 48 hours
Biochemical reactions	Catalase positive	Catalase positive
	CAMP positive	CAMP positive
	Mostly urease positive*	Urease negative
	DNase negative	DNase positive

supratentorial. Furthermore, 41 % of the patients included in the study were HIV-positive, and 61 % of these cases exhibited concurrent pulmonary involvement [7]. Notably, when comparing extrapulmonary involvement between immunocompetent and immunocompromised patients, Gundelly and colleagues observed intriguingly higher rates in the former group. This disparity held true in both European and US studies, with statistically significant differences indicated by a P value of 0.0002 and 0.0001, respectively [8]. In our search for English-written published cases on PubMed within the last decade, we identified a limited number of instances. Specifically, only five cases, including our own, were documented as cases of brain *R. equi* infections. These cases have been succinctly summarized in Table-2.

4. Discussion

The *Rhodococcus* genus belongs to the *Nocardiaceae* family and encompasses a total of 53 species. Although various species, including *R. erythropolis, R. ruber, R. gordoniae, and R. facsians*, have been reported in the medical literature as human pathogens, *R. equi* stands out as the most frequently documented [13]. Notably, there have been over one hundred reported cases of *R. equi* infections in HIV patients, and this spectrum has expanded to encompass transplant recipients and immunocompetent individuals. Infections can be acquired through ingestion, direct inoculation, or inhalation [4]. While many cases have reported animal exposures, this is not universally the case [3]. In our case, the patient had a history of exposure to cats, which could have served as the source of infection. Recent data have confirmed that *Rhodococcus* can be isolated from cat feces, establishing cats as reservoirs of *Rhodococcus*, thus corroborating our assumption [14]. The treatment of *R. equi* infection has presented a significant challenge, primarily due to the absence of a standardized regimen. The intricacy of treating this pathogen can be attributed to its intracellular growth and elevated resistance, even though susceptibility testing standards for this organism remain less well-established [4,13]. Clinically, both initial response and subsequent relapse are not uncommon, as seen in numerous cases initially presenting as pulmonary infections that progress to brain infections after cessation of antibiotic treatment, mirroring our own case [9,11]. This underscores the necessity for

Table 2
Rhodococcus brain infections from 2011 until this case, AMK: amikacin, AZM: azithromycin, CRO: ceftriaxone, CPX: ciprofloxacin, DM: diabetes mellitus, DOX: doxycycline, F: female, HIV: human immunodeficiency virus, ITP: Immune thrombocytopenia, IT: intrathecal, IV: intravenous, M: male, MEM: meropenem, RIF: rifampicin, SLE: Systemic lupus erythematosus, VAN: vancomycin.

case	Reference	Age/ Sex	Comorbidities	Brain imaging	Diagnosis	Diagnosing rhodococcus brain infections	Treatment for brain infection and relapses	Final outcome
1	[9]	49/F	HIV (CD4 count 118)	Extracranial abscess then relapsed as ring enhancement lesion	Pneumonia, bacteremia, brain abscess and relapsed as brain abscess	Previous bacteremia, imaging and clinical response to anti-rodococcal regimen	Surgical removal + IV VAN, IMP, CRO and CPX for 8 weeks then oral CP and CLM for 15 weeks. After 20 weeks > relapse: IV VAN, CRO and CPX for 3 weeks.	Survived for years with no other relapses
2	[10]	52/ M	Hypertension Moderate heart failure	Frontal-temporal mass	Bacteremia and brain abscess	Brain lesion biopsy and culture	IV AMK and MEM	Died in few days
3	[11]	48/ M	SLE ITP DM Immunosuppressant drugs	Multiple aggregated enhancing lesions and later enhancement of the meninges	Necrotizing pneumonia, brain abscess, three times relapses as recurrent meningitis	CSF culture grew *Rhodococcus* on fourth admission (no growth initially)	Multiple regimens used last episode treated with: IV & IT VAN, IT AMK that shifted later to AZM, oral RIF, oral DOX (different time frames for each)	Survived after 4 months of last relapse > no further relapses recorded
4	[12]	40/ M	Post trauma CNS rhinorrhea	Normal	Purulent meningitis	CSF culture	IV MEM for 33 days	Survived after 2 months with no relapse
5	Our case	41/ M	HIV (CD4 count 29) Depression	Ring enhancement lesions	Necrotizing pneumonia, bacteremia, relapsed as brain abscesses	Previous bacteremia, imaging and clinical response to anti-rodococcal regimen	IV VAN and MEM for 4	Survived after 9 months > no relapse weeks maintained on AZM and trimethoprim-sulfamethoxazole

prolonged antimicrobial therapy in managing this organism, along with prophylactic measures, particularly in immunocompromised states. For instance, one case reported *R. equi* sepsis in a renal transplant patient who required treatment for up to 9 months, even in the absence of brain involvement [2]. To ensure *R. equi* eradication, clinicians should contemplate the restoration of the immune system in HIV patients by initiating antiretroviral therapy (ART). It is worth noting that mortality in such individuals has been reduced significantly, from 56 % to 8 %, following the introduction of highly active ART [8]. In our case, the patient received two months of antibiotic treatment for bacteremia and necrotizing pneumonia, resulting in positive clinical, microbiological, and radiological responses. However, the discontinuation of antimicrobials, non-compliance with antiretroviral therapy, and persistent low CD4 counts may have collectively contributed to the recurrence of *R. equi* infection and subsequent brain involvement.

In our presented case, the feasibility of lumbar puncture (LP) was contraindicated due to midline shift and the presence of brain lesions, thus precluding the collection of cerebrospinal fluid (CSF) for culture. Despite these limitations, including the inability to perform a brain biopsy due to the deep-seated location of the lesion and concerns about potential neurological deterioration, we believe that the clinical and radiological improvement observed following the administration of vancomycin and meropenem provides strong evidence supporting the diagnosis of brain rhodococcosis. The fact that the patient exhibited no response to anti-toxoplasmosis therapy over three weeks, with the emergence of new lesions and an increase in the size of existing ones, lends further support to this hypothesis. While most of the five reported cases of brain rhodococcosis had positive cultures, as summarized in Table-2, it's worth noting that, similar to our case, Ferretti et al. reported an instance of an HIV patient with rhodococcal bacteremia and imaging suggestive of a brain abscess. This patient did not respond to anti-toxoplasmosis therapy but demonstrated improvement upon initiation of an anti-rhodococcal regimen, even though no positive CSF culture was obtained [9].

Identification of *R. equi* can be challenging in the laboratory setting for several reasons. Firstly, its appearance in Gram stain can lead to misidentification as diphtheroid and may be erroneously considered a blood contaminant [15]. Secondly, the fact that *R. equi* can be acid-fast on Ziehl-Neelsen stain can result in misidentification as *Mycobacterium*, especially given that both organisms can present with similar pulmonary symptoms [16]. Thirdly, misidentification can also occur due to the absence of *R. equi* in some automated identification system databases. In four reported cases, *R. equi* was misidentified as Kocuria spp. when utilizing the VITEK-2 GP ID card (Afonso et al., 2022; Savini et al., 2011; Spiliopoulou et al., 2014; Vechi et al., 2018, and in one case, it was misidentified as *Turicella otitidis* using the VITEK-2 ANC ID card, mirroring our experience [17]. Correct identification in such scenarios often necessitates a combination of clinical correlation, review of colonial morphology, assessment of biochemical reactions, and identification by another automated system that includes the organism in its database, such as MALDI-TOF MS or gene sequencing. This underscores the crucial role of microbiologists, despite the availability of advanced laboratory tools, and emphasizes the critical importance of effective communication between laboratory physicians and clinicians.

5. Conclusion

R. equi has emerged as a recognized infection that affects both immunocompromised and immunocompetent individuals. Healthcare professionals face various challenges when dealing with this pathogen, including selecting appropriate treatment options, determining the optimal duration of treatment, accurate identification, assessing susceptibility, and addressing antimicrobial resistance. Recognizing the limitations of automated identification systems within the laboratory is crucial, whether you are a microbiologist or an infectious diseases specialist. This awareness is vital for achieving accurate identifications of rarely encountered organisms like *R. equi*. Effective communication between clinicians and laboratory physicians plays a pivotal role in preventing misidentifications and, consequently, the improper management of such infrequently encountered pathogens. Furthermore, standardization of susceptibility testing, treatment regimens, and therapy durations is imperative for addressing *R. equi* infections. This is particularly important given the increasing number of reports involving immunocompromised and even immunocompetent individuals in recent times.

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Ethical approval

Informed consent was obtained from the patient's relative for the publication of all images, clinical data and other data included in the manuscript. No ethical approval was sought as additional cases were compiled from published case reports. The institution operates in accordance with the 1964 Helsinki declaration and its later amendments.

Data availability statement

Data included in article/supp. material/referenced in article.

Additional information

No additional information is available for this paper.

CRediT authorship contribution statement

Wala Alkhalifa: Writing – review & editing, Writing – original draft, Visualization, Validation, Supervision, Resources, Methodology, Investigation, Conceptualization. **Batool Abu Ali:** Writing – review & editing, Writing – original draft, Methodology, Investigation, Conceptualization. **Hassan AlDandan:** Writing – original draft, Investigation, Conceptualization. **Hosam Aljehani:** Supervision, Investigation, Conceptualization. **Marwan Alwazzeh:** Writing – original draft, Supervision, Methodology, Investigation, Conceptualization. **Asim Diab:** Supervision, Methodology, Investigation, Conceptualization.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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References

[1] S. Topino, V. Galati, E. Grilli, N. Petrosillo, *Rhodococcus equi* infection in HIV-infected individuals: case reports and review of the literature, AIDS Patient Care STDS 24 (4) (Apr. 2010) 211–222, https://doi.org/10.1089/apc.2009.0248.
[2] E. Macken, et al., *Rhodococcus equi* sepsis in a renal transplant recipient: a case study, Transplant Direct 1 (3) (Apr. 2015), https://doi.org/10.1097/TXD.0000000000000519.
[3] S. Herath, C. Lewis, M. Nisbet, THE NEW ZEALAND MEDICAL JOURNAL Increasing Awareness of *Rhodococcus equi* Pulmonary Infection in the Immunocompetent Adult: a Rare Infection with Poor Prognosis, 2013.
[4] D.M. Weinstock, A.E. Brown, *Rhodococcus equi*: an Emerging Pathogen, 2001 [Online]. Available: https://academic.oup.com/cid/article/34/10/1379/328195.
[5] J.F. Prescott, *Rhodococcus equi*: an Animal and Human Pathogen, 1991 [Online]. Available: https://journals.asm.org/journal/cmr.
[6] H. J, M.A. P, C.K.C. Jorgensen, Manual of Clinical Microbiology, eleventh ed., 2015.
[7] R.H. Roda, M. Young, J. Timpone, J. Rosen, *Rhodococcus equi* pulmonary-central nervous system syndrome: brain abscess in a patient on high-dose steroids-a case report and review of the literature, Diagn. Microbiol. Infect. Dis. 63 (1) (Jan. 2009) 96–99, https://doi.org/10.1016/j.diagmicrobio.2008.08.024.
[8] P. Gundelly, Y. Suzuki, J.A. Ribes, A. Thornton, Differences in *Rhodococcus equi* infections based on immune status and antibiotic susceptibility of clinical isolates in a case series of 12 patients and cases in the literature, in: BioMed Research International, vol. 2016, Hindawi Limited, 2016, https://doi.org/10.1155/2016/2737295.
[9] F. Ferretti, et al., Disseminated *Rhodococcus equi* infection in HIV infection despite highly active antiretroviral therapy, BMC Infect. Dis. 11 (Dec) (2011), https://doi.org/10.1186/1471-2334-11-343.
[10] Velázquez Benito A, Tejero Juste C, Pérez Lázaro C, Santos Lasaosa S. Absceso cerebral por Rhodococcus equi con comportamiento seudotumoral en paciente inmunocompetente [Cerebral abscess due to Rhodococcus equi with pseudotumour presentation in an immunocompetent patient]. Neurologia. 2013 Oct;28(8):522-4. Spanish. doi: 10.1016/j.nrl.2012.04.007. Epub 2012 Jun 14. PMID: 22703632.Velázquez Benito A., Tejero Juste C., Pérez Lázaro C., Santos Lasaosa S., NEUROLOGÍA Cerebral abscess due to Rhodococcus equi with pseudotumour presentation in an immunocompetent patient, 2011 [Online]. Available:.
[11] K. Lee, et al., A case of recurrent meningitis caused by *Rhodococcus* species successfully treated with antibiotic treatment and intrathecal injection of vancomycin through an ommaya reservoir, Infect Chemother 47 (3) (2015) 183–189, https://doi.org/10.3947/ic.2015.47.3.183.
[12] L.X. Cui, L.N. Gu, Y.B. Hou, B.L. Zhang, J.Y. Liu, Purulent meningitis caused by *Rhodococcus equi* a case report, Medicine (United States) 97 (24) (Jun. 2018), https://doi.org/10.1097/MD.0000000000011156.
[13] M. Majidzadeh, M. Fatahi-Bafghi, Current taxonomy of *Rhodococcus* species and their role in infections, 11., in: European Journal of Clinical Microbiology and Infectious Diseases, vol. 37, Springer Verlag, 2018, pp. 2045–2062, https://doi.org/10.1007/s10096-018-3364-x. Nov. 01
[14] C.L. de Paula, et al., First microbiological and molecular identification of *Rhodococcus equi* in feces of nondiarrheic cats, BioMed. Res. Int. 2019 (2019), https://doi.org/10.1155/2019/4278598.
[15] F.F. Tuon, et al., Rhodococcus equi bacteremia with lung abscess misdiagnosed as corynebacterium: a report of 2 cases, Clinics (Sao Paulo) 62 (6) (2007) 795–978, https://doi.org/10.1590/s1807-59322007000600022.
[16] T. Le, et al., Diagnosing *Rhodococcus equi* infections in a setting where tuberculosis is highly endemic: a double challenge, J. Clin. Microbiol. 53 (4) (Apr. 2015) 1431–1433, https://doi.org/10.1128/JCM.02284-14.
[17] A.R. Méndez-Cruz, G.E. Félix-Bermúdez, D.V. Aguilar-Escobar, L. Vega-Vega, A.I. Morales-Estrada, A. Contreras-Rodríguez, Bloodstream infection by *Rhodococcus* corynebacterioides in a pediatric patient diagnosed with high-risk retinoblastoma, Rev. Argent. Microbiol. (2022), https://doi.org/10.1016/j.ram.2022.06.001.