**Background**

*Trichosporon* species are basidiomycetous yeast-like fungi widely distributed in nature, predominantly found in tropical and temperate areas [1]. *Trichosporon* spp. may be found in substrates such as soil, decomposing wood, air, rivers, lakes, seawater, cheese, scarab beetles, bird droppings, bats, pigeons and cattle [1]. These organisms also are present in the human microbiota of skin and gastrointestinal tract [2]. *Trichosporon* spp. are phenotypically characterized by colonies of white or cream coloring, with dry appearance, cerebriform or radiated surface [3] and microscopically by the presence of blastoconidia, arthroconidia, pseudophyphae and true hyphae [4].

*Trichosporon* spp. are usually associated with superficial mycosis such as white *piedra* [1, 4, 5], onychomyco-
sis [1, 5–7] interdigital and inguinocrural lesions [5, 7]. Invasive trichosporonosis is a deep-seated infection which...
may be observed in leukemia or lymphoma patients who developed severe neutropenia, associated with broad spectrum antibiotic therapy [2, 4]. *Trichosporon asahii* and *T. mucoides* are the most frequently isolated species in invasive trichosporonosis [4, 8]. Pubmed searches using the terms “Trichosporon inkin”, “invasive” and “infection” only retrieved six publications [9–14].

The establishment of fungal infection is associated with host immune conditions as well as virulence attributes of the microorganism involved. For instance, adhesion to epithelial cells [15], the production and secretion of hydrolytic enzymes such as phospholipases and hemolysins [16] and the capacity of biofilm formation [17] contribute to yeasts pathogenicity and have been demonstrated in *Trichosporon* spp.

Central nervous system (CNS) trichosporonosis is a rare clinical manifestation associated with immunocompromised patients [18]. In fact, only a few clinical cases of this medical condition have been reported in the literature [2, 4, 18–25] and none of them was due to *T. inkin*.

The gold standard diagnosis of CNS trichosporonosis is the isolation of *Trichosporon* in culture of tissue samples or cerebrospinal fluid (CSF) [1, 19]. The reference method for *Trichosporon* species identification is based on Intergenic Spacer 1 (IGS1) region of the ribosomal DNA sequencing [8, 26]. However, Matrix-Assisted Laser Desorption/ Ionization Time-of-Flight (MALDI-TOF) Mass Spectrometry has been shown to be a valuable alternative to *Trichosporon* species identification [1, 27].

Despite some controversies, triazoles appear to be the best first line antifungal therapy for invasive trichosporonosis, especially when *T. asahii* is involved [1, 10, 18, 19, 27]. In vitro studies suggest that voriconazole exhibits the best antifungal activity against different *Trichosporon* species, when compared to amphotericin B and fluconazole [10]. Echinocandins are associated with high MIC values [19, 28] and indicates no action on *Trichosporon* cells. Therefore, they are not indicated in clinical practice [19, 27].

To the best of our knowledge, we describe the first case of meningoencephalitis due to *T. inkin* in a previously healthy female patient under corticosteroids regimen after having undergone microsurgery of neuroma in Natal, Rio Grande do Norte State, Northeast Brazil. We also describe the antifungal susceptibility profiling and characterization of the expression of virulence factors in vitro of two *T. inkin* isolates sequentially obtained from the CSF of this patient and also a review of meningoencephalitis clinical cases due to *Trichosporon* spp. reported in the literature.

Case presentation

MECM, a 49-years-old previously healthy woman, married and childless, was admitted at a private hospital in Natal City, Rio Grande do Norte State, Brazil, in June, 2014 for a microsurgery of neuroma. She used to live in a flat with a parrot who had an unknown disease that caused loss of feathers. The microsurgery was performed via the cranial middle fossa to remove a left sided acoustic neuroma. After 40 days of the procedure, she presented a predominantly and intensive occipital holocranial headache, followed by vomiting. She was managed with analgesia and prednisone 20 mg/day for 5 days. The patient also had hyporexia that was accentuated with the worsening of headache, 12 kg of weight loss, asthenia, irritability, difficulty to concentrate and rotator vertigo. She did not have a fever. On physical examination, the patient presented classic signs of irritability of meningeal inflammation.

On the 50th postoperative day, she was diagnosed with a cerebrospinal fistula in the occipital region and submitted to a surgical correction. The CSF analysis revealed 126 cells/mm³, composed by 63% of lymphomonocytes, 13 mg/dl of glucose levels (89 mg/dl of glycemia) and 189 mg/dl of proteins. Direct examination and CSF microbiological culturing (including common bacterial, mycobacterial and fungal procedures) did not detect any pathogen. Hemogram and biochemical examination of blood were normal. Vancomycin and ceftriaxone were prescribed for 14 days, dexamethasone, 16 mg/day, for 10 days, followed by 15 days of prednisone weaning. She was discharged with partial improvement of headache, without vomiting and presenting normal CSF. After 3 weeks, the headache intensified and vomiting returned. Prednisone 80 mg/day, for 7 days, followed by 30 days of weaning was prescribed, resulting in mild improvement of headache, but with persistent vomiting and return of rotational vertigo. Therefore, cinnarizine, esomeprazole, bromopride and paracetamol/codeine were prescribed. As no relief was obtained after 30 days, the patient was re-hospitalized and CSF analysis revealed: 245 cells/mm³, 88% of lymphomonocytes, 23 mg/dl of glucose levels and proteins of 324 mg/dl. Microbiological cultures for bacteria and fungi were negative. Hemogram and biochemical examination of blood were still normal. She was diagnosed again with occipital liquoric fistula and submitted to clinical treatment. She was under the same antimicrobial and corticoid regimen of the last hospitalization and was discharged with mild headache. Dexamethasone 16 mg/day, for 10 days, followed by 30 days of weaning with prednisone was prescribed. At that moment, the CSF still had 68 cells/mm³, with 100% of lymphomonocytes, 56 mg/dl of glucose levels and 78 mg/dl of proteins. Prednisone was prescribed for 30 days.

When the corticoid was discontinued, headache worsened and vomiting returned. After 5 months of the onset of the disease, a new computed tomography (CT) scan of the skull showed a CSF fistula on the same topography. She was hospitalized and submitted to a surgery to correct the fistula. She had leukocytosis on admission (16,000 leukocytes/mm³, with 88% segmented cells) and CSF analysis
showed 280 cells/mm³, being 88% of lymphomonocytes cells, 12 mg/dL of glucose levels and 312 mg/dL of proteins. Bacterial and fungal cultures were negative. Empirical treatment with vancomycin and cefepime was introduced for 21 days and dexamethasone 16 mg/day for 10 days, followed by 20 days of weaning with prednisone. As the headache worsened, she was again hospitalized and submitted to surgical correction of the fistula. New CSF showed 184 cells, 63% of lymphomonocytes, 41 mg/dL of glucose levels and 285 mg/dL of proteins. Vancomycin, meropenem and dexamethasone, 10 mg/day were initiated. On the 5th day of treatment, headache remained intense and frequent vomiting. A new CT suggested hydrocephalus and the patient was submitted to a ventriculoperitoneal (VP) shunt. After 3 days of VP, the patient continued to present with vomiting and leukocytosis and the CSF pressure was above 300 mmH2O. She was admitted to the intensive care unit. A magnetic resonance imaging (MRI) of the skull suggested meningeal thickening, spinal cord compression at the level of C5-C6 and the alteration of the CSF signal was compatible with viral or fungal disease (Fig. 1). The initial suspicion was cryptococcosis. Liposomal amphotericin B (300 mg/day) and acyclovir therapy were empirically initiated. After several invasive procedures, broad spectrum antibiotics and corticosteroids, CSF culture showed growth of Trichosporon spp. After 2 weeks, another Trichosporon CSF positive culture was obtained. As there was progressive worsening of the clinical condition, voriconazole (200 mg/12 h) was added to the previous prescription. On the 20th day of hospitalization, the patient died (Table 1).

Culturing procedures and molecular identification of the pathogen
The CSF was centrifuged at 2500 rpm for 10 min and the sediment was used for direct examination and culture. Direct examination was performed with India ink which revealed no encapsulated blastoconidia. The sediment of 2 CSF samples collected at different days (14th and 28th of April, 2015) were plated on Sabouraud Dextrose Agar at room temperature (28 + 2 °C) and yielded positive yeast cultures after 72 h of incubation. The two cultures were send to the Medical and Molecular Mycology Laboratory, Clinical and Toxicological Analyses Department, Federal University of Rio Grande do Norte State for further molecular identification. Of note, both colonies had a mucoid aspect. Besides, because Cryptococcus spp. are the main etiological fungal agents obtained from meningitis, that was the first suspicion. Yeast isolates from original cultures were plated onto CHROMagar Candida (CHROMagar Microbiology, Paris, France) and corn meal-Tween 80 (to induce sporulation). Surprisingly, both isolates had a macroscopic wrinkled appearance, were able to produce arthroconidia, as revealed by their micromorphology, and to hydrolyze urea (Fig. 2a to d). Therefore, they were considered to belong to the genus Trichosporon and named HGT198 and HGT914, respectively. Both strains were further identified by molecular techniques.

Molecular identification
A single colony of each strain was used for DNA extraction with PrepMan Ultra sample preparation reagent (Applied Biosystems, Foster City, CA) according to the manufacturer’s instructions. Genomic DNA concentration and purity were checked with a NanoDrop instrument (Thermo Scientific; Amersham Pharmacia Biotech, Wilmington, DE, USA). Both strains were further identified by a molecular method as detailed elsewhere [29]. DNA amplification was obtained by using the primer pair TRF (5’-AGAGGCTTA CCATGGTATCA-3’) and TRR (5’-TAAGACCCATA CGCCCTA-3’) [26]. Nucleotide sequences were submitted for BLAST analysis at the NCBI site (http://www.ncbi.nlm.nih.gov) for species identification. Only sequences deposited in GenBank showing high similarities with our query sequences and an E-value of lower than 10⁻⁵ were used in this study. BLAST searches showed the best match with T. inkin (FJ153608.1), 100% identity (619 of 619 bp without gap sites) for both strains (HGT198 and HGT914). IGS1
rDNA sequences of these strains have been deposited in GenBank under accession numbers KY807052 and KY807053, respectively. Of note, both strains were considered of 100% identity, after blastn analysis (all the 641 bp compared among them), with an E-value of 0 and no gaps found between the two IGS1 rDNA sequences.

Characterization of virulence factors and in vitro antifungal susceptibility

Strains HGT198 and HGT914 were evaluated according to their ability to adhere to human buccal epithelial cells, biofilm formation, hemolysins and phospholipase production by using the methods described by Zuza-Alves [30]. DNAse production was determined according to Montoya [16].

Table 1 Timeline of exposition to multiple risk conditions of a patient submitted to an acoustic neuroma surgery and further developed meningitis in Natal city, Rio Grande do Norte State, Northeast Brazil

| Period of time | Acoustic neuroma surgery | Corticosteroids usage | Appearance of the fistula | Antibiotics usage | CSF analysis performing | Surgical fistula correction | VP shunt | Antifungal therapy | Trichosporon positive culture | Death |
|---------------|--------------------------|-----------------------|---------------------------|------------------|------------------------|-----------------------------|--------|-------------------|-----------------------------|-------|
| Week 1        | X                        |                       |                           |                  |                        |                             |        |                   |                             |       |
| Week 6        | X                        |                       |                           |                  |                        |                             |        |                   |                             |       |
| Week 8        | X                        | X                     |                           |                  |                        |                             |        |                   |                             |       |
| Week 9        | X                        |                       |                           |                  |                        |                             |        |                   |                             |       |
| Week 13       | X                        |                       |                           |                  |                        |                             |        |                   |                             |       |
| Week 17       | X                        | X                     |                           |                  |                        |                             |        |                   |                             |       |
| Week 18       | X                        |                       |                           |                  |                        |                             |        |                   |                             |       |
| Week 19       | X                        |                       |                           |                  |                        |                             |        |                   |                             |       |
| Week 20       | X                        | X                     |                           |                  |                        |                             |        |                   |                             |       |
| Week 21       | X                        |                       |                           |                  |                        |                             |        |                   |                             |       |
| Week 22       | X                        |                       |                           |                  |                        |                             |        |                   |                             |       |
| Week 23       | X                        | X                     |                           |                  |                        |                             |        |                   |                             |       |
| Week 24       | X                        |                       |                           |                  |                        |                             |        |                   |                             |       |
| Week 25       | X                        |                       |                           |                  |                        |                             |        |                   |                             |       |
| Week 26       | X                        |                       |                           |                  |                        |                             |        |                   |                             |       |

*CSF Cerebrospinal fluid, VP Ventriculoperitoneal

![Fig. 2](image-url)

**Fig. 2** a Cream-colored, dull, wrinkled cerebriform colonies, after 48 h of incubation at 30 °C on Sabouraud dextrose agar. b Colonies with typical “dirty” grey-blue color on CHROMagar Candida® medium after 72 h of incubation at 35 °C. c Micromorphological aspects after incubation in cornmeal agar containing Tween 80 for 72 h at 30 °C, showing long true hyphae and arthroconidia. d Urease test of yeast cells grown in Cristensen’s urea Agar containing phenol red, showing positive results after incubation at 30 °C for 72 h.
Both strains did not produce phospholipase or DNAse. However, they showed high biofilm formation capability as compared to C. albicans ATCC90028 and T. asahii CBS2630 and similar levels of hemolysin production of the two reference strains. In addition, they were able to adhere to epithelial cells to the same extent of T. asahii reference strain (Table 2).

Both strains were tested against fluconazole, itraconazole and amphotericin B by using the CLSI protocol [31–33]. As illustrated on Table 3, they exhibited very low MIC values against all antifungal drugs tested.

**Discussion and conclusions**

Trichosporon species are present in the environment and may belong to the human microbiota but may associated with both superficial and deep infections [1, 18]. Infections associated with CNS due Trichosporon are rare in immunocompromised patients and extremely rare in immunocompetent patients [20].

The number of cases of invasive trichosporonosis reported worldwide may be still considered restrict once a limited number of publications may be found in the literature [27]. A recent epidemiological study conducted by De Almeida Júnior et al. [27] reviewing global cases of invasive trichosporonosis retrieved from PubMed from 1994 and 2015 only found a total of 203 cases of invasive trichosporonosis, where T. asahii accounted for 95 (47.6%) of all cases [27].

The first case of SNC infection due Trichosporon was described by Watson and Kallichurum in 1970 [25] in a 39-year-old African woman diagnosed with underlying bronchial adenocarcinoma and brain abscess due T. cutaneum [25]. It is important to mention that no molecular techniques were available at this time. Therefore, this could have led to an unreliable species identification. Since then, only other few case reports of Trichosporon meningitis have been published in the literature, but none of them was due to T. inkin (Table 4).

The geographic distribution of invasive trichosporonosis with CNS complications is higher in Asia (seven cases) [2, 18–22, 24]. There are cases described in Europe [23], Africa [25] and Central America (one case each) [4]. Clinical series with patients’ demographic data and underlying conditions may be found in Table 4.

There is only a single study reporting Trichosporon meningitis in an immunocompetent patient [22]. In this study, Rastoji et al. [22] reported a case of invasive trichosporonosis with CNS complications in a 18-years-old male, who presented fever, chills and rigor associated with headache, nausea, vomiting and altered sensorium. T. asahii was isolated from CSF and sputum of this patient [22].

Several clinical conditions are considered to be risk factors for invasive trichosporonosis including the history of intensive chemotherapy, high dose of corticosteroids, burns, neutropenia, broad spectrum antibiotics usage and hematological malignancies [8, 20, 27]. In fact, the patient described in this study was previously immunocompetent but further submitted to long periods of corticosteroids and broad-spectrum antibiotics, besides suffering the invasive medical procedure to remove an acoustic neuroma. Of note, the CSF fistula probably had an important role for local Trichosporon contamination. In addition, the fact that she had a parrot could have led to yeasts skin colonization, once these birds may harbor Trichosporon in their gastrointestinal tract [34]. A limitation of our study is that we did not perform her parrot’s droppings culture, trying to isolate Trichosporon and further checked with molecular techniques to determine the probable source of infection.

Our isolates were as able to adhere to epithelial cells as T. asahii CBS2630 that is considered the most virulent and frequently isolated species of the genus Trichosporon, as demonstrated by virulence studies with Galleria mellonella and murine models of systemic infection [35]. Of note, all Trichosporon isolates were less adherent than Candida albicans ATCC90028. This was expected, because C. albicans is widely recognized as the more adherent Candida species [36], but it was used as a reference strain for adhesion assay.

Both isolates (HGT198 and HGT194) had stronger hemolytic activities than reference strain T. asahii CBS2630. Our isolates were considered highly hemolytic according

| **Table 2** Evaluation of attributes of virulence factors in vitro of Trichosporon inkin isolates HGT198 and HGT194 obtained from a patient submitted to an acoustic neuroma surgery and further developed meningitis in Natal city, Rio Grande do Norte State, Northeast Brazil |
|-------------------------------------------------|-----------------|-----------------|
| **Candida albicans ATCC90028**                  | **N° of T. inkin cells adhered to 150 HBEC** | **Hemolytic index (HI)** |
|                                                 | 179.8 ± 2.05    | 0.63 ± 0.01     |
| **Trichosporon asahii CBS2630**                 | 34.3 ± 1.70     | 0.74 ± 0.01     |
| **Trichosporon inkin HGT198**                   | 36.7 ± 1.50     | 0.68 ± 0.01     |
| **Trichosporon inkin HGT194**                   | 37.3 ± 1.50     | 0.56 ± 0.01     |

**NT** Not Tested

*Statistically significant different from Candida albicans ATCC90028

*Statistically significant different from Trichosporon asahii CBS2630

*Statistically significant difference between HGT198 and HGT194
with the criteria established by Montoya et al. [16]. These authors reported a single strain of *T. asahii* with strong hemolytic activity, while the other 38 isolates did not produce this enzyme. These findings reinforce virulence attributes properties of the isolates from the present study that may have influenced clinical outcome.

Biofilm formation by microorganisms has gained attention within clinical practice because of its ability to increase mortality in patients with systemic infections by yeasts [37]. Studies on biofilm formation in *Trichosporon* have increased in recent years, since this yeast has been considered the second most common etiological agent of systemic infection in patients with hematological malignancies [38]. In the presence of biofilm, structured microbial communities remain embedded within an extracellular polymeric substance, where *Trichosporon* spp. show significantly greater resistance to antifungals, with MICs ranging from 128 to 1.024 μg/mL for *Amphotericin B* and 512–1.024 μg/mL for *Fluconazole*, *Itraconazole*, and *Voriconazole* [38]. Our isolates showed an optical density of 595 nm (which reflects biofilm biomass stained by crystal violet) at least two-fold higher than the readings found for *T. asahii* CBS6030. Of note, there is a trend of increased virulence attributes expression over time when both strains isolated 2 weeks apart were compared (Table 1). This phenomenon is observed for both hemolysins and biofilm production. This may reflect strain adaptation to the host in the progress of infection.

Our strains were not resistant to any of the antifungal drugs tested. In addition, MICs obtained from *T. inkin* HGT198 and HGT914 did not increase over time. This observation indicates that probably there was not induced antifungal resistance for the strains recovered within 2 weeks of difference. It is important to emphasize that antifungal susceptibility testing was only possible to be performed after patient’s death and was not used to drive antifungal therapy.

### Table 3

Determination of antifungal susceptibility testing of *Trichosporon inkin* isolates HGT198 and HGT914 obtained from a patient submitted to an acoustic neuroma surgery and further developed meningitis in Natal city, Rio Grande do Norte State, Northeast Brazil

| Strain                          | Fluconazole (24 h) | Itraconazole (48 h) | Amphotericin B (48 h) |
|---------------------------------|--------------------|---------------------|-----------------------|
| *Candida parapsilosis* ATCC 22019 | 1 μg/mL            | NT                  | NT                    |
| *Candida krusei* ATCC6258       | 16 μg/mL           | NT                  | NT                    |
| *Trichosporon inkin* HGT-198    | 0.5 μg/mL          | 0.062 μg/mL         | 0.5 μg/mL             |
| *Trichosporon inkin* HGT-914    | 0.5 μg/mL          | 0.062 μg/mL         | 0.5 μg/mL             |

### Table 4

Systematic review of *Trichosporonosis* meningitis cases published in the literature from to 1970 to 2018

| Country | Sex/Age | Diagnosis/Underlying diseases | Species isolated | Clinical Sample | Treatment/outcome | Year/Reference |
|---------|---------|-------------------------------|------------------|-----------------|------------------|-----------------|
| Brazil  |         |                               | *Trichosporon inkin* | CSF             | AMB, VOR, ITR, POS/Survived | 2016 [24]     |
| Singapore | F/50 | Disseminated trichosporonosis/ Aplastic Anemia | *T. asahii* | CSF, Blood | AMB/died | 2015 [20]     |
| India   | M/18   | Chronic meningo-ventriculitis and intraventricular fungal ball/immunocompetent | *T. asahii* | Intraventricular biopsy and CSF | AMB/died | 2012 [18]     |
| Iran    | M/34   | Brain abscess/ autoimmune hepatitis, hypothyroidism | *T. asahii* | Brain abscess | Surgical resection, AMB and ITC/ survived | 2012 [19]     |
| India   | NI     | Meningitis/Acquired Immunodeficiency Syndrome (AIDS) | *Trichosporon sp.* | CSF | AMB, FLU, survived | 2012 [19]     |
| Jamaica | F/44   | Meningitis and cerebral abscess/diabetes, burns | *T. asahii* | Facial wounds, sputum, and a meningeal swab | None/died | 2011 [4]     |
| Taiwan  | NI     | Meningitis/NI                 | *T. montevideense* | CSF | FLU/survived | 2009 [2]     |
| India   | M/18   | Disseminated trichosporonosis/ Immunocompetent | *T. asahii* | CSF | FLU/survived | 2007 [22]     |
| India   | F/36   | Chronic meningitis/ Chronic back pain after fall | *T. beigeli* | CSF | None/died | 1995 [21]     |
| Belgium | M/15   | Meningitis/acute lymphocytic leukaemia | *T. beigeli* | CSF | AMB, FC and FLU/died | 1990 [23]     |
| South Africa | F/39 | Brain abscess/adenocarcinoma | *T. cutaneum* | Brain lesions | None/died | 1970 [25]     |
There are some limitations about the interpretations of MIC values in our *Trichosporon* isolates. First of all, there are still no breakpoints established from Clinical & Laboratory Standards Institute (CLSI) and European Committee on Antimicrobial Susceptibility Testing (EUCAST) Antifungal Susceptibility testing to *Trichosporon* species [27], and second, there are no studies which determined MICs of *T. inkin* strains isolated from patients with meningocencephalitis.

Susceptibility testing to the antifungal drugs Amphotericin B, Itraconazole and Fluconazole were performed by Taj-Aldeen et al. [39] with three *T. inkin* clinical isolates obtained from urine and white *piedra*. MIC range was 1–4 μg/mL (Amphotericin B), 0.25–4 μg/mL (Fluconazole) and 0.013–0.125 μg/mL (Itraconazole). *T. inkin* clinical isolates from bone, urine, skin, subcutaneous abscess, peritoneal liquid and blood susceptibility profiling was determined and MIC range was 0.06–1 μg/mL (Amphotericin B), 1–32 μg/mL (Fluconazole) and 0.06–2 μg/mL (Itraconazole) [40]. In the present study, our *T. inkin* isolates had lower MIC values when compared with other publications [39, 40]. We observed that Itraconazole exhibited better in vitro effect against *T. inkin* isolates compared to Fluconazole and Amphotericin B and this finding was also observed by other studies involving *T. inkin* [39, 40] and other *Trichosporon* species [2, 35].

In conclusion, we may say this is the first case report of meningitis caused by *T. inkin* reported in the literature. Our female previously healthy patient was under corticosteroids and antibiotics therapy for a few months. In addition, she was submitted to an invasive procedure to remove a left sided acoustic neuroma and further developed a cerebrospinal fistula. All those factors are consolidated risk conditions for the infection caused by *T. inkin*. The certainty of the invasive infection by *T. inkin* was based on the isolation of the pathogen among two different occasions together with brain images and cytological findings suggestive of meningitis. Both strains showed strong ability to express virulence factors in vitro. These findings together with patient’s immunological status may have been crucial for the clinical outcome, because the strains were apparently not resistant to the antifungal drugs prescribed during her period of hospitalization. The physicians main suspicion was cryptococcal meningitis. Corroborating this idea, the strains obtained from CNF presented mucoid aspect in the primary isolation. However, the sending of these isolates to a reference Mycology center for accurate phenotypic and molecular identification revealed that the meningitis caused by *Trichosporon inkin*.

**Abbreviations**

ATCC: American Type Culture Collection; BLAST: Basic Local Alignment Search Tool; CBS: Centraalbureau voor Schimmel cultures; CLSI: The Clinical & Laboratory Standards Institute; CNS: Central nervous system; CSF: Cerebrospinal fluid; CT: Computed tomography; Fig.: Figure; IGS1: Intergenic Spacer 1; MALDI-TOF: Matrix-Assisted Laser Desorption/Ionization Time-of-Flight; MIC: Minimal Inhibitory Concentration; MRI: Magnetic Resonance Imaging; rpm: Revolutions per minute; VP: Ventriculoperitoneal

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**Availability of data and materials**

The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

**Authors’ contributions**

JJS, TUGF and ALAP were the attending physicians for the patient and collected medical data of the patient. MFA performed microbiological analysis and in vitro experiments. ECF and ASAM performed strains molecular identification. ALC made a critical review of the manuscript and provided financial support and structure for molecular identification. WPSR performed experiments and wrote the paper. EPM designed and wrote the paper and critically analyzed medical data. GMC designed all the experiments, structured and wrote the paper and critically reviewed the manuscript. All authors read and approved the final manuscript.

**Ethics approval and consent to participate**

All clinical and demographic data of the patient were collected in accordance with the Local Research Ethics committee from the Liga Norte-RioGrandense Contra o Câncer Hospital, approved under number 042/042/2012.

**Consent for publication**

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

**Competing interests**

The authors declare that they have no competing interests.

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