Clinical Presentation, Management, and Outcomes of Patients With Brain Abscess due to Nocardia Species

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Background. Nocardial brain abscesses are rare, and published literature describing brain abscesses due to Nocardia species is limited to individual case reports or small series. We report one of the largest contemporary retrospective studies describing risk factors, diagnostic evaluation, management, and outcomes of nocardial brain abscess.

Methods. Retrospective review of all adults with brain abscess due to culture-confirmed Nocardia species at our institution between January 1, 2009, and June 30, 2020.

Results. Overall, 24 patients had nocardial brain abscesses during the study period. The median age at presentation was 64 years, and 62.5% were immunocompromised. Pulmonary and cutaneous infections were the most common primary sites of nocardial infection. All 24 patients had magnetic resonance imaging performed, and the frontal lobe was the most commonly involved. The most common organism isolated was Nocardia farcinica, followed by Nocardia wallacei and Nocardia cyriacigeorgica. Thirteen patients were managed with antimicrobial therapy alone, while 11 had both medical and surgical management. In all patients, dual therapy was recommended for the initial 6 weeks of treatment, and 22 patients received at least 1 oral agent as part of their final antibiotic regimen, predominantly trimethoprim-sulfamethoxazole and linezolid. Fourteen patients achieved complete clinical and radiographic resolution of infection.

Conclusions. Nocardia is an important cause of brain abscess in the immunocompromised host. Early diagnostic and therapeutic aspiration may help healthcare providers confirm the diagnosis, choose an appropriate antimicrobial regimen, and achieve source control.

Keywords. brain abscess; management; Nocardia; risk factors.

Brain abscesses are rare, with a worldwide estimated incidence ranging from 0.3 to 1.3 per 100 000 persons per year [1]. However, they tend to occur with considerably higher frequency in immunocompromised patients. Nocardial brain abscesses are exceedingly uncommon and comprise only 2% of all intracranial abscesses [2]. Nocardia has a unique tropism for the brain [3]. Infection is usually acquired through inhalation. Central nervous system (CNS) involvement may occur via hematogenous spread or direct extension from a contiguous cranial infection site following head trauma [4, 5]. The spectrum of CNS infection ranges from diffuse cerebral infiltration, meningitis, spinal cord infection, to brain abscess.

Over the last 30 years, the introduction of new antibiotics and diagnostic procedures has considerably changed the management of brain abscesses. However, even with the advancement of imaging technologies and antimicrobial therapy, mortality rates for nocardial brain abscesses remain high, up to 30%, compared with 10% for other bacterial causes [6].

Published data on the Nocardia spp. causing brain abscesses are dated or limited to case reports or small case series that often lack details about diagnostic and management interventions and clinical outcomes data. Understanding the interplay between patient factors and laboratory and radiologic findings of nocardial brain abscesses may improve the identification of individuals at risk. Therefore, we aimed to describe the clinical presentation, treatment, and outcomes of patients with Nocardia brain abscess in a contemporary cohort at a large referral center.

METHODS

We retrospectively reviewed all adult patients (≥18 years old) with an International Classification of Diseases, Ninth Revision and Tenth Revision, Clinical Modification diagnosis of brain abscess at our institution from January 1, 2009, through June 30, 2020. A brain abscess was defined as a localized intracerebral collection of necrotic material surrounded by a well-vascularized capsule associated with at least 1 of the following 3 characteristics: (a) positive blood cultures for Nocardia spp., (b) positive cultures for Nocardia spp. in brain abscess
aspirate, (c) presence of Nocardia organisms on histopathology of the excised brain material.

Clinical specimens from blood and brain aspirate were cultured on-site in BD Bactec mycobacteria growth indicator tube (MGIT) 960 broth in mycobacterial growth indicator tubes (Becton, Dickinson and Company, Franklin Lakes, NJ, USA) and on Middlebrook 7H11/7H11S agar biplates incubated at 35°C to 37°C for up to 6 weeks. Positive MGIT broth was subcultured to a Middlebrook 7H11 agar plate, and isolated colony growth was identified using Sanger sequencing of a 500-bp region of the 16S rRNA gene. A 100% match to the database entry was required for identification. From August 2014, matrix-assisted laser desorption ionization time-of-flight mass spectrophotometry (MALDI-TOF MS) was added to supplement species identification using Sanger sequencing [7, 8].

Brain abscess cases were further categorized as health care-associated infections (HAIs) if brain abscess developed ≥48 hours after admission, was not present at the time of admission, and the patient was admitted for a cause other than Nocardia [9]. Immunocompromised patients were defined as solid organ or bone marrow transplant recipients, patients with hematologic or solid malignancy, and those currently on antineoplastic chemotherapy, immunomodulators, or other immunosuppressive drugs, including corticosteroids (≥5 mg/d for >14 days). Final antibiotic therapy was defined as an antibiotic regimen used for >50% of the total duration of therapy. Species were labeled susceptible if >80% of isolates tested were susceptible. Relapsed antibiotic therapy was defined as an antibiotic regimen used for >50% of the total duration of therapy. Species were labeled susceptible if >80% of isolates tested were susceptible. Relapsed infection was defined as the association of clinical and radiological signs of nocardiosis with the isolation of the same Nocardia species after the cessation of antimicrobial treatment for nocardiosis. Clinical, laboratory, and radiographic data were extracted from the electronic health record. The study was approved by the Mayo Clinic Institutional Review Board (IRB# 20-000488).

Categorical variables were reported as frequencies and proportions. Continuous variables were reported as median (interquartile range [IQR]). Five-year mortality was reported with a Kaplan-Meier curve. Statistical analysis was performed using JMP, version 14.1.0 (SAS Institute Inc., Cary, NC, USA).

RESULTS

Demographic Characteristics
A total of 247 adult patients with brain abscess were screened, and 24 (9.7%) patients met the study criteria. The median age at presentation (IQR) was 64 (58.5–71.2) years, and 75% were males. The most common underlying medical conditions were chronic kidney disease (45.8%), hypertension (33.3%), and diabetes mellitus (29.1%). Fifteen (62.5%) patients were immunocompromised, including 3 patients (20%) with head and neck malignancy. Other malignancies included prostate cancer (20%), lung adenocarcinoma (13.3%), and lymphoma (13.3%). Two patients (8.3%) had head and neck surgery. Nine patients (37.5%) were on prednisone, with a median dose (IQR) of 10 (8.7–23) mg for >2 weeks. Seven (29.2%) patients were on other immunomodulatory therapies as summarized in Table 1. The median time from solid organ transplant (SOT) to diagnosis of Nocardia spp. brain abscess (IQR) was 876 (261–1698) days. A total of 4 patients were on trimethoprim-sulfamethoxazole (TMP-SMX) for Pneumocystis pneumonia prophylaxis. The median Charlson Comorbidity Index (CCI) score (IQR) was 7 (4.25–10).

Clinical and Radiographic Presentation
Lung and cutaneous infections were the most common primary sites of nocardial infection (37.5% and 12.5%, respectively). Lung infections were more frequent in immunocompromised than immunocompetent hosts (71% vs 29%). The immunocompetent host was more likely to have nocardiosis due to presumed direct inoculation secondary to trauma than the immunocompromised patients (62% vs 38%). Three (12.5%) cases were HAIs, and the infectious diseases (ID) team was consulted on all patients during their hospitalization.

All 24 patients had magnetic resonance imaging (MRI) performed, and 19 (79.1%) patients had a head computed tomography (CT). Of the patients who had a head CT, 13 (68.4%) had similar findings, but 6 (31.6%) had fewer lesions when compared with the MRI. Eight (33.3%) patients had >1 intracranial fluid collection. The most common location was the frontal lobe (41.6%), followed by parietal (37.5%), temporal (33.3%), and occipital (12.5%). Two patients had cerebellar involvement. The fluid collections’ median diameter size (IQR) was 14 (10–21) mm, and midline shift was observed in 8.4% of cases.

Microbiology
All 24 patients had a positive culture result from direct brain abscess sampling. The most common species isolated were Nocardia farcinica (n = 9; 37.5%), N. wallacei (n = 3; 12.5%), N. cyriacigeorgica (n = 3; 12.5%), N. abscessus (n = 1; 4.1%), N. otitidiscaviarum (n = 1; 4.1%), N. transvalensis (n = 1; 4.1%), and N. argensis (n = 1; 4.1%). Similarly, all 24 patients had peripheral blood cultures performed, but only 3 patients had a positive blood culture for Nocardia spp. (Table 2).

Management and Outcomes
Thirteen (54.2%) patients were managed solely by medical management, while 11 (45.8%) had both medical and surgical management (Table 2). The median time from nocardial brain abscess diagnosis to surgical therapeutic intervention (IQR) was 3 (0.2–6) days.

Corticosteroids were used as part of the medical treatment in 10 (41.6%) patients, mostly due to midline shift and neurologic deficits. Dexamethasone was most commonly used (8; 33.3%), with a median dose (IQR) of 6 (4–12) mg per day and a median duration (IQR) of 14 (5–71.5) days.
The most common empiric antibiotic therapy used alone and/or in combination was TMP-SMX in 41.6% of cases, followed by vancomycin (37.5%), linezolid (33.3%), metronidazole (33.3%), and meropenem (21.1%). A total of 20 patients (83.3%) had at least 1 active antibiotic started empirically. The most common definitive antibiotic regimen based on susceptibilities is summarized in Table 2. Susceptibility testing varied among different species. All isolates were susceptible to TMP-SMX. Additional trends included different species. All isolates were susceptible to TMP-SMX and linezolid. The final median antibiotic duration (IQR) was 322 (180.5–365) days. Thirty-six percent of patients receiving immunosuppressive therapy had reductions in immunosuppression as part of their management.

Fourteen (58.3%) patients achieved complete resolution of the clinical manifestations and radiographic resolution of brain fluid collection. Two (8.3%) had permanent neurological deficits (left hemiparesis and seizure, respectively), 3 (12.5%) patients relapsed, and 1 (4.1%) patient progressed despite medical therapy requiring surgical intervention at a later time during his hospital course. A total of 7 (29.1%) patients died, with a median time from diagnosis to death of 169 days. Four of these (16.6%) deaths were related to underlying chronic conditions including malignancy and cardiovascular disease. The average follow-up time for this cohort was 19 months. Figure 1 shows that ~60% of patients with nocardial brain abscess survived at 5 years.

**DISCUSSION**

The current study is one of the largest contemporary cohorts to describe the risk factors, clinical and radiographic features, management interventions, and outcomes of nocardial brain abscess. *Nocardia* brain abscess occurs mostly in the fifth to sixth decades of life [10, 11], with the peak occurrence between ages 43 and 75 years. Purported risk factors for developing nocardial brain abscess include age, gender, and immunocompromised status [12, 13]. In our cohort, patients who presented with a nocardial brain abscess were older and predominantly Caucasian males, consistent with prior reports [14–16]. Higher incidence of nocardial brain abscesses in older age may be due to immunosenescence, a physiological part of aging linked to a higher risk of infection in part due to the excessive production of pro-inflammatory cytokines by macrophages and fibroblasts that may impact the innate and adaptive immune systems, which are crucial in the development of nocardial brain abscess [17, 18].

Systemic infection with *Nocardia* spp. most often occurs in immunosuppressed patients and rarely in immunocompetent individuals [4, 19, 20]. In our cohort, the percentage of immunocompromised individuals, including 10 transplant patients and those receiving corticosteroids, was higher than in other published studies [21–26]. Cell-mediated immune deficiencies, in particular, seem to be a major predisposing factor for *Nocardia* infections [20, 27]. As seen in our series, steroids or other immunosuppressive medications such as calcineurin inhibitors, antiproliferative agents, and mTOR inhibitors can suppress cell-mediated immunity, likely contributing to the high prevalence of *Nocardia* infections [20, 27, 28]. As the overall population ages and more patients receive various immunosuppressive therapies due to expanding indications, we may see more nocardial abscesses in the future.

Chronic corticosteroid therapy, especially at higher doses and with prolonged durations, has been associated with
| Cases | Age, Gender | Immune-compromised Host | Primary Source | Brain Abscess Location | Diameter, mm | Positive Blood Cultures | Nocardia Spp. on Direct Brain Abscess Sampling | Final Antibiotic Therapy | Final Antibiotic Route | Duration of Therapy, d | Type of Surgical Intervention | Outcome |
|-------|-------------|--------------------------|----------------|------------------------|-------------|------------------------|---------------------------------|---------------------------|----------------------|----------------------|--------------------------------|----------|
| 1     | 80, M       | Yes                      | Pulmonary      | Frontal                | 12          | No                     | N. wallacei                     | Moxifloxacin, amikacin         | Oral/IV              | 84                   | None                | Died[^2]                        |          |
| 2     | 72, M       | Yes                      | Skin           | Frontal, parietal and midbrain | 17          | Yes                    | N. farcinica                    | TMP-SMX                   | IV                   | 180                 | Open aspiration            | Relapsed and died |          |
| 3     | 74, M       | No                       | Unknown        | Parietal, brain stem and cerebellum | 13          | No                     | N. farcinica                    | TMP-SMX                   | Oral                 | 336                 | Open aspiration            | Permanent neurologic deficit |          |
| 4     | 63, F       | No                       | Pulmonary      | Frontal, occipital and temporal | 21          | No                     | N. cyriacigeorgica              | Ceftriaxone                | IV                   | 168                 | None                | Cured                           |          |
| 5     | 77, F       | No                       | CNS trauma     | Temporal               | 24          | No                     | Nocardia spp.                  | TMP-SMX, doxycycline         | IV/oral              | 168                 | Open aspiration            | Cured                           |          |
| 6     | 50, M       | Yes                      | Unknown        | Temporal               | 41          | No                     | N. farcinica                    | Linezolid, amoxicillin-clavulanate | Oral                 | 224                 | Open aspiration            | Relapse and died |          |
| 7     | 94, M       | Yes                      | Pulmonary      | Temporal and parietal  | 15          | No                     | N. farcinica                    | Linezolid, imipenem          | Oral/IV              | 14                  | Stereotactic             | Died[^3]                       |          |
| 8     | 73, F       | No                       | Unknown        | Parietal               | 8           | No                     | N. wallacei                     | Linezolid, TMP-SMX, minocycline | Oral                 | 308                 | None                | Relapse and died[^4]       |          |
| 9     | 65, M       | Yes                      | Pulmonary      | Cerebellum and fore-brain | 10          | No                     | N. farcinica                    | TMP-SMX, moxifloxacin, doxycycline | Oral                 | 280                 | None                | Cured                           |          |
| 10    | 69, M       | No                       | Pulmonary      | Frontal                | 10          | No                     | N. cyriacigeorgica              | TMP-SMX                   | Oral                 | 365                 | None                | Required delayed surgical intervention |          |
| 11    | 50, M       | Yes                      | Unknown        | Parietal               | 5           | No                     | N. otitidisavarium             | TMP-SMX, amikacin           | Oral/IV              | 260                 | None                | Cured                           |          |
| 12    | 66, M       | Yes                      | Skin           | Brain stem             | 4           | No                     | Nocardia spp.                  | TMP-SMX, amoxicillin-clavulanate | Oral                 | 365                 | None                | Cured                           |          |
| 13    | 49, M       | No                       | Pulmonary      | Parietal               | 6.6         | Yes                    | N. farcinica                    | TMP-SMX, moxifloxacin        | Oral                 | 365                 | None                | Cured                           |          |
| 14    | 61, M       | Yes                      | Skin           | Frontal                | 4           | No                     | Nocardia spp.                  | TMP-SMX                   | Oral                 | 365                 | None                | Cured                           |          |
| 15    | 64, M       | Yes                      | Unknown        | Midbrain               | 21          | No                     | Nocardia spp.                  | TMP-SMX                   | Oral                 | 365                 | None                | Permanent neurologic deficit |          |
| 16    | 60, F       | Yes                      | Unknown        | Temporal and parietal  | 10          | No                     | N. argensis                     | TMP-SMX, moxifloxacin       | Oral                 | 365                 | Open aspiration            | Cured                           |          |
| 17    | 34, M       | Yes                      | Pulmonary      | Temporal               | 26          | No                     | Nocardia spp.                  | TMP-SMX                   | Oral                 | 365                 | Open aspiration            | Cured                           |          |
| 18    | 64, M       | Yes                      | Unknown        | Occipital and temporal | 17          | No                     | N. cyriacigeorgica              | Linezolid, TMP-SMX          | Oral                 | 504                 | Open aspiration            | Cured                           |          |
| 19    | 50, M       | Yes                      | CNS trauma     | Frontal, occipital, temporal, and parietal | 13          | No                     | N. farcinica                    | TMP-SMX, ceftriaxone        | Oral/IV              | 196                 | None                | Died                            |          |
| 20    | 61, F       | No                       | Unknown        | Frontal                | 10          | No                     | N. farcinica                    | Linezolid, ceftriaxone      | Oral/IV              | 182                 | None                | Cured                           |          |
| 21    | 69, M       | No                       | Unknown        | Parietal               | 22          | No                     | N. farcinica                    | TMP-SMX                   | Oral                 | 365                 | Stereotactic             | Cured                           |          |
| 22    | 69, F       | Yes                      | Pulmonary      | Frontal                | 20          | No                     | N. transvalensis                | TMP-SMX, doxycycline        | Oral                 | 365                 | None                | Cured                           |          |
| 23    | 58, M       | No                       | Unknown        | Frontal                | 20          | No                     | N. abscessus                    | TMP-SMX, amoxicillin-clavulanate | Oral                 | 365                 | Open aspiration            | Cured                           |          |
| 24    | 63, M       | Yes                      | Pulmonary      | Frontal                | 25          | Yes                    | N. wallacei                     | TMP-SMX, imipenem          | Oral/IV              | 20                  | Stereotactic             | Died                            |          |

Abbreviations: CNS, central nervous system; F, female; IV, intravenous; M, male; TMP-SMX, trimethoprim-sulfamethoxazole.

[^2]: Diameter of the largest lesion in cases when more than 1 lesion was present.

[^3]: Death related to *Nocardia* infection.
increased risk of opportunistic infections such as Pneumocystis pneumonia—thus the recommendation for TMP-SMX prophylaxis [5, 29]. Daily TMP-SMX prophylaxis also may prevent nocardiosis and account for the reduced prevalence of this organism in patients with AIDS and SOT recipients [30, 31]. However, breakthrough nocardial infections may occur in the context of low-dose or intermittent TMP-SMX prophylaxis [32–34]. Interestingly, in our series, 4 patients were on 1 TMP-SMX double-strength tablet daily for prophylaxis and still developed nocardial brain abscess. Therefore, if the suspicion for *Nocardia* infection is high, use of TMP-SMX prophylaxis should not dissuade clinicians from considering nocardial abscesses in differential diagnosis.

Primary lung infection, likely due to inhalation, was the most frequent mechanism of infection acquisition in our cohort, similar to earlier observations [26, 35]. Hematologic spread with a high propensity to the skin and subcutaneous tissue or CNS has also been described [5, 19, 36]. In our series, *Nocardia* spp. grew in the blood cultures of only 3 patients. Detailed skin examination and CT of the chest can be helpful to identify the primary site of infection. Interestingly, although ID was consulted in all cases in our series and a thorough evaluation was performed, the primary infection source could not be identified in 41.7% of the cases. We hypothesize that infection may have started as a direct inoculum from trauma or inhalation but infection at the primary site was subclinical and therefore no primary source was identified.

Nocardial brain abscesses may present as a hyperenhancing multiloculated ring lesions. As the infection usually occurs through inhalation and from direct spread from the sinuses [37], the frontal lobe is commonly involved. However, location, size, and appearance of nocardial brain abscesses alone cannot be used to differentiate these from other causes of bacterial abscesses. Sometimes it is difficult to differentiate nocardial abscesses from intracranial metastatic malignancy on CT or MRI [38]. Therefore, while MRI continues to be the preferred radiologic method for imaging of suspected nocardial abscesses [39], diagnostic aspiration is necessary to confirm the diagnosis and for selection of appropriate antimicrobial therapy.

The importance of isolating and culturing the *Nocardia* spp. is due to concern for resistance to specific antimicrobial agents [40]. However, *Nocardia* spp. have relatively slow growth, and they can be difficult to culture in the laboratory, making the 16S rRNA gene sequencing method a reliable alternative for identification [19]. Kiska et al. [41] concluded that no single method could accurately identify all *Nocardia* spp. associated with human infections. A combination of the antimicrobial susceptibility pattern, colony pigment, biochemical tests, and molecular techniques could potentially identify all isolates to the species level. To date, no specific brain tropism has been identified to a particular species. The present study was concordant with previous reports [3, 42–45] where *N. farcinica* and *N. abscessus* were the most commonly encountered species.

In general, the most active agents against *Nocardia* species include TMP-SMX, amikacin, minocycline, and imipenem [46, 47]. However, no randomized trials have been performed to compare the efficacy of different antibiotic regimens for nocardiosis. A study by Brown-Elliott et al. involving 522 clinical isolates reported that only 2% of the isolates demonstrated

![Figure 1. Survival curve is patients with Nocardia spp. brain abscess.](image)
in vitro resistance to TMP-SMX [40]. Hamdi et al. reported similar results [46]. Sulfonamides and trimethoprim are small lipophilic antibiotics. At high doses, the penetration into the cerebrospinal fluid, in both the absence and the presence of meningal inflammation, is considered sufficient for the treatment of CNS infections with susceptible bacteria [48]. Based on these observations, TMP-SMX is considered the mainstay of therapy [26, 49]. Acknowledging the high morbidity and mortality associated with nocardial brain abscesses, TMP-SMX should be used in combination with another highly bioavailable antimicrobial with good CNS penetration for induction therapy for nocardial brain abscess. Given the small number of patients in our study and earlier publications, no definitive recommendations can be made, and larger, multicenter studies are needed to determine the optimal treatment regimen.

Despite the limited literature, surgical excision is considered necessary in most cases [2, 50]. Lee et al. reported aspiration alone in 90.9% of their patients with no reported deaths [51]. In a study by Hall et al. [52], surgical aspirations alone were considered appropriate as the initial management of brain abscess. Others have proposed that craniotomy and excision of the entire abscess and wall are more effective than aspiration and drainage [2]. In our patient population, 54.2% of the cases had no surgical management, likely due to the smaller median brain abscess diameter size in our cohort (14 mm). In general, surgical aspiration is recommended and preferred in lesions larger than 2.5 cm in diameter [2].

Patients with nocardial brain abscess may have residual motor deficits and hearing impairment even with successful treatment for underlying infection [2]. In our cohort, hemiparesis and seizure were encountered only in few cases. The majority (62.5%) of our patients had a good outcome. Poor outcomes, including death, were mainly due to patients’ underlying comorbidities. This is further supported by a high median CCI score in our cohort. Due to the small size, we were unable to conclude if medical management vs a combination of medical and surgical management impacts patient outcomes. However, it stands to reason that early diagnosis and surgical management may be associated with reduced morbidity and mortality due to early and effective source control.

**Limitations**

The retrospective nature of the study with a case determination that was based on the claims data set is the primary limitation. Decisions regarding diagnostic testing and therapeutic interventions were left to the discretion of treating physicians and were not based on any standardized protocol. Due to the small size of our cohort, we were unable to assess the statistical significance of our observation. Finally, even though the Mayo Clinic in Minnesota receives patients from other cities and rural areas, the high prevalence of immunocompromised patients and those presenting from the Midwest limits our ability to generalize our findings.

**CONCLUSIONS**

Older, immunocompromised, and high-morbidity populations are at increased risk for nocardial brain abscesses. Early diagnostic and therapeutic aspiration may help health care providers confirm diagnosis, choose an appropriate antimicrobial regimen, and reduce morbidity and mortality due to early and adequate source control.

**Supplementary Data**

Supplementary materials are available at Open Forum Infectious Diseases online. Consisting of data provided by the authors to benefit the reader, the posted materials are not copyedited and are the sole responsibility of the authors, so questions or comments should be addressed to the corresponding author.

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**Patient consent.** The design of the work was approved by the local institutional review board (IRB# 20-000488). The study was considered minimal risk, and patient consent requirements were waived.

**Author contributions.** Cristina Corsini Campioli, MD: conception or design of the work, data collection, data analysis and interpretation, drafting the article, critical revision of the article, final approval of the version to be published; Natalia E. Castillo Almeida, MD: drafting the article, critical revision of the article, final approval of the version to be published; John C. O’Horo, MD: conception or design of the work, critical revision of the article, final approval of the version to be published; John Raymond Go, MD: critical revision of the article; Daniel C. DeSimone, MD: critical revision of the article; M. Rizwan Sohail, MD: conception or design of the work, drafting the article, critical revision of the article, final approval of the version to be published.

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