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Case report

FACIAL NERVE PARALYSIS AS A COMPLICATION OF ACUTE OTITIS MEDIA IN CHILDREN: A CASE REPORT AND LITERATURE REVIEW

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Abstract: **Introduction**: The facial nerve controls facial expression, and its dysfunction causes facial asymmetry and reduced mimic function. Facial nerve paralysis is significantly less common in children compared to adults. Children under the age of 10 are estimated to have an incidence rate of approximately 3 per 100,000 per year. The leading cause of facial paralysis in the pediatric population is idiopathic (Bell's palsy), similar to that in adults. In cases where the cause is not idiopathic, infections accompanied by trauma and congenital anomalies are the most frequent etiological factors of facial nerve paralysis in children. This condition is most often attributed to nerve edema and subsequent compression within the bony fallopian canal.

Case Report: This report presents a case of peripheral facial nerve paralysis as an extracranial complication of acute otitis media in a 2-year-old child. It also includes a literature review focusing on recent diagnostic protocols, evaluation, and therapeutic approaches.

Conclusion: As demonstrated in our case, the prognosis for facial nerve paralysis associated with acute otitis media (AOM) was generally good after appropriate therapy. Recovery from facial paralysis usually occurs within three months. This report delineates a rare complication of AOM in children. Timely recognition and appropriate therapy are crucial to achieve good outcomes.

Keywords: facial nerve paralysis, acute otitis media, complications.

INTRODUCTION

Acute otitis media (AOM) is an inflammation of the middle ear mucosa, characterized by the presence of effusion and symptoms that develop within a period of up to 48 hours (1). It is one of the most common infectious diseases affecting children worldwide. AOM usually develops as a viral upper respiratory tract infection caused by respiratory viruses, followed by a bacterial superinfection in the presence of a weakened immune response or inadequate treatment. The infection typically spreads through the rhinogenic route, that is, from the nasopharynx via the eustachian tube (2).

In most children older than one year, the condition resolves spontaneously within several days, even without the use of antibiotic therapy. However, in approximately 20% of cases, the disease progresses, and serial superinfections may lead to serious complications. The most common extracranial complication is acute mastoiditis, which typically occurs in the third week after disease onset, either in a latent or manifested form. Facial nerve paralysis, an extracranial complication of AOM, is also possible, although it is extremely rare and occurs in approximately 0.005% of affected children (3). The primary reason for the low incidence, particularly in developed countries, is mandatory immunization and the use of broad-spectrum antibiotics for the treatment of acute otitis media (AOM) (4). Severe intracranial complications, such as meningitis or brain abscesses, fortunately occur only rarely (Figure 1).

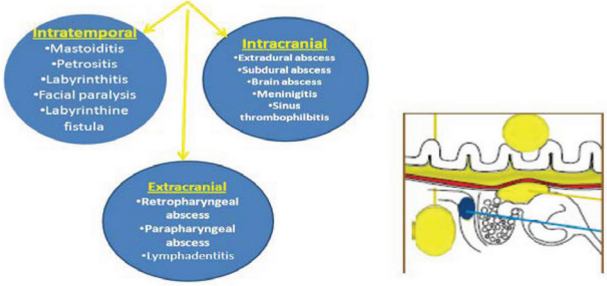

Figure 1. *Complications of otitis media (Wikipedia. Available at: https://en.wikipedia.org/wiki/Otitis_media)*

Considering the still not fully defined etiopathogenetic mechanisms of AOM and the rarity of peripheral FNP as a complication of this condition, there is an ongoing debate regarding diagnostic and therapeutic approaches. In this context, particular attention should be paid to the indications for surgical treatment of this complication (5).

Case Report

A two-year-old boy was brought to the otorhinolaryngology outpatient clinic by his parents due to an acute onset of paralysis on the left side of his face. The symptoms began four days before the examination and initially presented as occasional coughing and clear nasal discharge. The child did not have a fever. Before referral to an otorhinolaryngology specialist, the attending pediatrician initiated symptomatic therapy.

The patient was the first child, born from an uncomplicated pregnancy and delivered vaginally three weeks before term. The APGAR score was 9/10, and the birth weight was 2,900 g. He had received all vaccinations appropriate for his age. He had previously experienced one episode of bronchitis. There was no known history of allergies to food or medications. Family history revealed that the mother had experienced hearing impairment requiring hearing aids since the age of four, due to recurrent ear infections.

Upon admission to the pediatric department, the child was conscious, afebrile, eupneic, and hemodynamically stable, with the following vital signs: body weight, 12 kg; body temperature, 36.8°C; oxygen saturation, 99%; and heart rate, 90 beats/min. There was no rash, and the skin had normal coloration and turgor. Lung auscultation revealed normal breath sounds, with no additional findings. The heart rhythm was regular and the tone was clear, with no murmurs.

Neurological examination revealed weakness and asymmetry of the left facial muscles, sparing the forehead muscles. There was loss of the left nasolabial and frontal folds, a lag in the left corner of the mouth when crying, and a drooping of the left corner of the mouth at rest, accompanied by difficulty closing the left eye and infrequent blinking (Figure 2). The clinical presentation corresponded to grade IV peripheral facial nerve paralysis based on the House–Brackmann scale (6) (Table 1).

Figure 2. *Left-sided peripheral facial nerve palsy in a child at the time of initial examination (The image is from the authors' personal archive)*

Otorhinolaryngological examination revealed bilateral hyperemia of the tympanic membranes on otomicroscopy, with a visible fluid level in the middle ear and a deformed light reflex. There was no swelling or hyperemia in the retroauricular or mastoid regions, and no tenderness on palpation. Oropharyngoscopy showed mild hyperemia of the pharyngeal mucosa. Endonasally, there was a serous discharge in the nasal passages, whereas palpation of the neck revealed no enlarged lymph nodes.

Laboratory tests performed after admission indicated an elevated leukocyte count of 12.87/mm³, with

Table 1. *House-Brackmann facial nerve grading system. (Adapted from: House JW, Brackmann DE. Facial nerve grading system. Otolaryngol Head Neck Surg. 1985; 93(2): 146–7. (6))*

Grade	Description	Eye Closure	Forehead Movement	Mouth Movement	Synkinesis
I	Normal facial function in all areas.	Complete	Normal	Symmetrical	None
II	Slight weakness; normal tone & symmetry at rest.	Complete, slight effort	Normal	Slight asymmetry	Mild
III	Moderate dysfunction, no noticeable weakness at rest.	Complete, with effort	Slightly reduced	Asymmetry present	Noticeable
IV	Severe dysfunction, obvious facial weakness.	Incomplete	No movement	Asymmetry with effort	Present
V	Severe dysfunction, minimal facial motion.	Incomplete	No movement	Very asymmetrical	Present
VI	Total facial paralysis; no motion.	None	No movement	No movement	None

a predominance of 67% lymphocytes. The C-reactive protein (CRP) level was 0.9 mg/L, whereas all other biochemical parameters and urinalysis with sediment were within the reference ranges.

Tympanometry showed a flat Type B tympanogram, indicating immobility of the tympanic membrane and the presence of fluid in the middle ear. Head and temporal bone computed tomography (CT) was planned as an emergency procedure but was not performed due to the parents' refusal to consent to this diagnostic procedure.

After establishing the diagnosis of acute paralysis nervi Facialis (FNP) associated with AOM, the child was promptly started on intravenous antibiotic therapy (Ceftriaxone 500 mg i.v. every 12 hours), along with probiotic syrup, tapering corticosteroids (Methylprednisolone 20 mg i.v.), vitamin B infusion (every 24 hours), and an analgesic–antipyretic (Paracetamol syrup 5 ml every 6–8 hours).

On the second day of hospitalization, an ophthalmology consultation was performed to prevent the most serious complications of facial nerve paralysis, such as corneal damage due to reduced secretion and lagophthalmos. The on-call ophthalmologist noted that mydriasis was appropriate for the child's age, and all other ophthalmological findings were normal. Artificial tears were prescribed to keep the eye moisturized, and the mother was advised to monitor the child and ensure that the affected eye was closed during sleep. A follow-up ophthalmology appointment was scheduled for seven days later, at which point the findings remained normal.

For the purpose of further etiological diagnostics of peripheral facial nerve paralysis and identification of potential infectious agents, serological tests were performed for herpes simplex virus, varicella virus, coxsackievirus, and adenovirus, all of which returned negative results. The urine culture results were also negative.

On the same day, left-sided paracentesis was performed for both diagnostic and therapeutic purposes. Microbiological analysis of the purulent drainage yielded negative results. Follow-up laboratory tests conducted the following day revealed inflammatory marker levels within the reference range.

During hospitalization, the patient remained afebrile and hemodynamically stable. Intravenous antibiotics, corticosteroids, and symptomatic therapy were administered for several days. On the seventh day of hospitalization, the child was discharged in good general condition, with a recommendation to continue oral antibiotic therapy (Cefixime oral suspension, 5 ml once daily) for the next seven days. Additionally, facial muscle exercises were advised under the supervision of a physiotherapist and a speech therapist at the local health center. A follow-up examination was scheduled seven days later, with instructions to immediately contact an otorhinolaryngologist through the emergency service in case of any new symptoms.

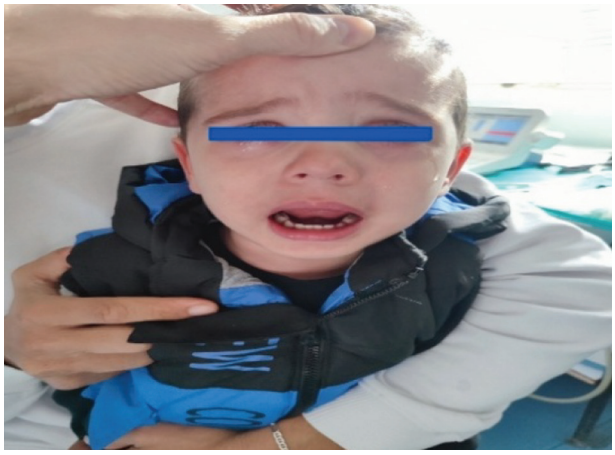

Figure 3. *Follow-up examination.Significant improvement in local findings*
(The image is from the authors' personal archive)

Figure 4. *One-month follow-up.*
Complete recovery of facial function
(The image is from the authors' personal archive)

At the follow-up visit, a significant improvement in local findings was observed. Inspection revealed only slight facial asymmetry, with normal closure of the left eye and forehead wrinkling (Figure 3). Otoscopic examination showed no hyperemia of the tympanic membranes; pink oropharyngeal mucosa was noted during oropharyngoscopy, and clear nasal passages were observed during anterior rhinoscopy. Palpation of the neck revealed no enlarged lymph nodes. The parents reported that the child remained afebrile throughout, with normal sleep and a preserved appetite.

At the next follow-up examination one month later, paralysis of the left side of the face had undergone complete regression (Figure 4).

The child was subsequently monitored over a six-month period with monthly check-ups, during which findings related to the seventh cranial nerve remained normal. Throughout this period, the child was in good general health and showed appropriate developmental progress for his age.

Two months later, the child developed a new episode of bilateral otitis media accompanied by low-grade fever, but without any cranial nerve deficits or other signs of intra- or extratemporal complications. The condition was treated conservatively following a ten-day course of antibiotic therapy according to protocol. At the follow-up examination, the tympanic membranes appeared normal, and the child was in good general condition.

A consultative examination by an otologic surgeon was also conducted at that time. The clinical findings were normal, and bilateral Type A tympanograms were recorded. Recommendations were made to maintain nasal patency, support the child's immune system, and schedule follow-up visits as needed.

Since then, there have been no recurrent ear infections or related complications, and the child, accompanied by his parents, has not returned to the otorhinolaryngology outpatient clinic or emergency pediatric services.

DISCUSSION

The facial nerve is the seventh cranial nerve, and its most prominent function is the innervation of the muscles involved in facial expression. Additionally, it has secretory and sensory functions. The nerve can be divided into six segments: intracranial, intrameatal, labyrinthine, tympanic, mastoid, and extracranial. In the final segment, after exiting the facial canal, it enters the parotid gland and divides into two main branches, which further split into the "pes anserinus" — five terminal branches. The ramus marginalis is particularly important in the facial musculature because it is the only branch that does not form anastomoses with neighboring branches (7).

Etiology

The possible etiological factors of FNP in children can be classified as congenital or acquired, with acquired causes further divided into infectious, traumatic, neoplastic, hypertension-related, and idiopathic causes (Bell's palsy).

If there is no definitive cause for acute unilateral facial paralysis (onset <72 h), the diagnosis is Bell's palsy. In children, the incidence of Bell's palsy is estimated at 6.1 cases per 100,000 children annually between the ages of 1 and 15 (8).

Acute otitis media remains the most common infectious cause of facial nerve paralysis in children (9). Epstein-Barr virus (EBV), human immunodeficiency virus (HIV), herpes simplex virus (HSV), among others, can also lead to facial paralysis.

Iatrogenic facial nerve paralysis may occur because of oromaxillofacial, parotid, or otologic surgery. In a case series of 35 pediatric patients with facial nerve paralysis, only three cases were iatrogenic, and all patients achieved full recovery. However, if a child does not show relatively rapid improvement, less common causes, such as malignancies and metabolic disorders, must be considered. Hypertension is a rare cause of facial nerve paralysis in children. FNP in children may, in very rare instances, be associated with systemic diseases such as granulomatosis with polyangiitis (GPA), sarcoidosis, and systemic lupus erythematosus. Due to the scarcity of pediatric cases, evidence is mostly restricted to individual case reports (9).

In the pre-antibiotic era, the incidence of facial nerve paralysis in acute otitis media (AOM) was 0.5%. In the era of antimicrobial therapy, the frequency of AOM dropped sharply. The etiology of facial nerve paralysis in patients with AOM remains unknown (10, 11). Nerve paralysis typically develops gradually in children, within 2–3 days of the onset of acute otitis media.

Diagnostic approaches in previous studies

The assessment of peripheral FNP is twofold: the cause and degree of the lesion must be examined, and the location of the lesion must be topographically assessed. The investigation of the cause, in addition to a thorough medical history (which must include details on prior ear treatments, injuries, or diagnosed systemic diseases), should include a detailed clinical examination (otorhinolaryngological, neurological, assessment of the degree of the lesion, internal medicine, and ophthalmological examinations), radiological investigations (CT of the temporal bones and MRI of the temporal region and pontocerebellar angle, ultrasound of the parotid glands), and electrodiagnostic tests such as electromyography.

During physical examination, a patient with FNP cannot raise the eyebrow or close the eyelid on the affected side. The nasolabial fold is typically absent, and the affected side of the mouth droops with possible drooling (12). During clinical examination, special attention should be given to examining the external auditory canal, auricle, and mastoid. Infections of the middle ear leading to facial nerve paralysis are commonly associated with middle ear effusion, redness of

the tympanic membrane, purulent ear discharge, and tenderness over the mastoid region. These symptoms typically precede the onset of facial weakness by approximately 5 to 8 days (13). Topographical assessment of the lesion site is performed using multiple tests, including the Schirmer test, stapedius reflex, gustometry, and sialography.

If other inflammatory etiologies are considered, studies on C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR) should be ordered.

A tympanogram should be performed for all children with acute FNP. As part of the diagnostic protocol, in patients with facial nerve paralysis caused by AOM, computed tomography (CT) of the head should be performed to exclude a radiological diagnosis of otomastoiditis and document the presence of associated intracerebral or extracerebral complications (14).

Therapeutic modalities in previous studies

After diagnosis, facial nerve paralysis associated with AOM should be treated appropriately according to the prescribed protocol, with eradication of the purulent process as the primary goal (15). The therapy should include a third-generation broad-spectrum cephalosporin (e.g., Ceftriaxone, as in the case of our patient) as the initial antibiotic therapy, which may be modified based on microbiological susceptibility. The actual efficacy of antiviral medications in cases of facial nerve paralysis is still not fully understood (16). Corticosteroids appear to reduce recovery time, especially when applied in the early stages of the disease (17). Eye care is extremely important to prevent corneal abrasion and vision loss. If incomplete eyelid closure is present, it is essential to use saline eye drops during the day and ophthalmic ointments during sleep.

Surgical approaches for the treatment of this condition remain unclear. Several authors have reached a consensus on the need for myringotomy with or without the placement of ventilation tubes in cases of otomastoiditis without tympanic membrane perforation, suggesting that mastoidectomy should be performed, or less commonly, decompression of the facial nerve should be considered if no improvement occurs within a few days (18).

Prognosis

Children with facial paralysis typically have a favorable prognosis. Complete recovery of facial function occurs in about 90% of Bell's palsy cases in children aged 14 or younger, whereas only around one-third of patients over the age of 60 experience full recovery (19).

As demonstrated in our case, the outcome for facial nerve paralysis associated with AOM was generally good after appropriate therapy, although the incidence of residual dysfunction was 6% (20). While functional recovery is common, a study by Biebl and colleagues showed that in 175 children with facial paralysis, although most had complete functional recovery, residual facial asymmetry persisted in up to half of the cases (21).

CONCLUSION

This case highlights the intricacy of peripheral facial nerve paralysis as an extracranial complication of acute otitis media in children. Recovery from facial paralysis usually occurs within three months. This integration of data contributes to a deeper understanding of the clinical course of peripheral facial nerve paralysis during AOM in children and reinforces the necessity for vigilant monitoring and proactive intervention to improve the quality of life of our patients.

Abbreviations

AOM – Acute Otitis Media
PNF – Paralysis Nervi Facialis

Conflict of Interest Statement: The authors declare that there is no conflict of interest related to this study.

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Sažetak

PARALIZA FACIJALNOG NERVA KAO KOMPLIKACIJA AKUTNOG ZAPALJENJA SREDNJEG UVA KOD DECE: PRIKAZ SLUČAJA I PREGLED LITERATURE

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Uvod: Facijalni nerv je živac čije oštećenje dovodi do poremećaja mimike i pojave asimetrije lica. Paraliza facijalnog nerva je značajno ređa kod dece u poređenju sa odraslima. Procenjuje se da deca mlađa od 10 godina imaju učestalostod približno 3 na 100.000 godišnje. Vodeći uzrok facijalne paralize u pedijatrijskoj populaciji je idiopatski (Bell-ova paraliza), slično kao i kod odraslih. U slučajevima kada uzrok nije idiopatski, infekcije praćene traumom i urođene anomalije su najčešći etiološki faktori paralize facijalnog nerva kod dece. Javlja se kao posledica edematozne reakcije živca i sledstvene kompresije u Falopijevom koštanom kanalu.

Prikaz slučaja: Prikazujemo slučaj periferne paralize facijalisa kao egzokranijalne komplikacije akutnog otitisa kod deteta uzrasta dve godine. Zatim će biti načinjen pregled literature sa fokusom na savremene dijagnostičke protokole, evaluaciju i terapijske pristupe.

Zaključak: Kao što je prikazano u našem slučaju, prognoza paralize facijalnog nerva povezane sa akutnim zapaljenjem srednjeg uva (AOM) je generalno dobra nakon odgovarajuće terapije. Oporavak od facijalne paralize obično se dešava u roku od tri meseca. Ovaj prikaz prikazuje retku komplikaciju AOM-a kod dece. Pravovremeno prepoznavanje i adekvatna terapija su ključni za postizanje dobrih ishoda.

Ključne reči: paraliza facijalnog nerva, akutni otitis medijalis, komplikacije.

REFERENCES

1. Lieberthal AS, Carroll AE, Chonmaitree T, Ganiats TG, Hoberman A, Jackson MA, et al. The diagnosis and management of acute otitis media. Pediatrics. 2013; 131(3): e964-e99. doi: 10.1542/peds.2012-3488.

2. Rosenfeld RM, Shin JJ, Schwartz SR, Coggins R, Gagnon L, Hackell JM, et al. Clinical practice guideline: Otitis media with effusion (update). Otolaryngol Head Neck Surg. 2016; 154(1 Suppl): S1–S41. doi: 10.1177/0194599815623467.

3. Ellefsen B, Bonding P. Facial palsy in acute otitis media. Clin Otolaryngol Allied Sci. 1996; 21(5): 393–5. doi: 10.1046/j.1365-2273.1996.00810.x.

4. Dissanayake G, Zergaw M, Elgendy M, Billey A, Saleem A, Zeeshan B, et al. Effectiveness of pneumococcal conjugate vaccines over antibiotic resistant acute otitis media in children: a systematic review. Cureus. 2024; 16(8): e67771. doi: 10.7759/cureus.67771.

5. Popovtzer A, Raveh E, Bahar G, Oestreicher-Kedem Y, Feinmesser R, Nageris BI. Facial palsy associated with acute otitis media. Otolaryngol Head Neck Surg. 2005; 132(2): 327–9. doi: 10.1016/j.otohns.2004.09.013.

6. House JW, Brackmann DE. Facial nerve grading system. Otolaryngol Head Neck Surg. 1985; 93(2): 146-7. doi: 10.1177/019459988509300202.

7. Lorch M, Teach SJ. Facial nerve palsy: etiology and approach to diagnosis and treatment. Pediatr Emerg Care. 2010; 26(10): 763–9. doi: 10.1097/PEC.0b013e3181f3bd4a.

8. Ozkale Y, Erol I, Saygi S, Yilmaz I. Overview of pediatric peripheral facial nerve paralysis: analysis of 40 patients. J Child Neurol. 2015; 30(2): 193–9. doi: 10.1177/0883073814530497.

9. Evans AK, Licameli G, Brietzke S, Whittemore K, Kenna M. Pediatric facial nerve paralysis: patients, management and outcomes. Int J Pediatr Otorhinolaryngol. 2005; 69(11): 1521–8. doi: 10.1016/j.ijporl.2005.04.025.

10. Gupta DK, Atam V, Chaudhary SC. Recurrent lower motor neuron type facial palsy: an unusual manifestation of SLE. BMJ Case Rep. 2011; 2011: bcr1220103564. doi: 10.1136/bcr.12.2010.3564.

11. D'Anna C, Diplomatico M, Tipo V. Facial palsy in a baby with acute otitis media. Arch Dis Child Educ Pract Ed. 2018; 103(3): 155–7. doi: 10.1136/archdischild2017312743.

12. Ciorba A, Corazzi V, Conz V, Bianchini C, Aimoni C. Facial nerve paralysis in children. World J Clin Cases. 2015; 3(12): 973–9. doi: 10.12998/wjcc.v3.i12.973.

13. Shargorodsky J, Lin HW, Gopen Q. Facial nerve palsy in the pediatric population. Clin Pediatr (Phila). 2010; 49(5): 411–7. doi: 10.1177/0009922809347798.

14. Chen XC, Lu CW, Liu CH, Wei CC. Facial palsy complicated by masked otomastoiditis in a 3 month old infant. J Emerg Med. 2014; 46(2): e47–50. doi: 10.1016/j.jemermed.2013.08.029.

15. Gaio E, Marioni G, de Filippis C, Tregnaghi A, Caltran S, Staffieri A. Facial nerve paralysis secondary to acute otitis media in infants and children. J Paediatr Child Health. 2004; 40(8): 483–6. doi: 10.1111/j.1440-1754.2004.00436.x.

16. Wohrer D, Moulding T, Titomanlio L, Lenglart L. Acute facial nerve palsy in children: gold standard management. Children (Basel). 2022; 9(2): 273. doi: 10.3390/children9020273.

17. Malik M, Cubitt JJ. Paediatric facial paralysis: An overview and insights into management. J Paediatr Child Health. 2021; 57(6): 786–90. doi: 10.1111/jpc.15310.

18. Helms D, Roberge RJ, Kovalick M. Otomastoiditis related facial nerve palsy. J Emerg Med. 2003; 25(1): 45–9. doi: 10.1016/S07364679(03)00132X.

19. Peitersen E. Bell's palsy: the spontaneous course of 2,500 peripheral facial nerve palsies of different etiologies. Acta Otolaryngol Suppl. 2002; (549): 4–30. doi: 10.1080/000164802320401694.

20. White N, McCans KM. Facial paralysis secondary to acute otitis media. Pediatr Emerg Care. 2000; 16(5): 343–5. doi: 10.1097/00006565200010000000010.

21. Biebl A, Lechner E, Hroncek K, Preisinger A, Eisenkölbl A, Schmitt K, et al. Facial nerve paralysis in children: is it as benign as supposed. Pediatr Neurol. 2013; 49(3): 178–81. doi: 10.1016/j.pediatrneurol.2013.03.013.

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