Peruvian Journal of
Health Care and Global Health

Case Report

Ramsay Hunt Syndrome in Sub-Saharan Africa: A Case Report from Benguela, Angola

Síndrome de Ramsay Hunt en África subsahariana: Reporte de caso de Benguela, Angola

Antonino Sambundo Benjamin Kapitão[1,2,a], **Raúl Fernandes Tchitali**[1,2,b], **José Domingos Paiva**[1,2,b], **Eladio Geraldo Palanga**[1,2,b]

[1] Katiavala Bwila University. Benguela, Angola.
[2] Internal Medicine Department, General Hospital of Benguela. Benguela, Angola.
[a] Physician and Doctor in Biomolecule`s Sciences
[b] Medical Student

Article information

Cite as: Benjamin Kapitão AS, Fernandes Tchitali R, Domingos Paiva J, Geraldo Palanga E. Ramsay Hunt Syndrome in Sub-Saharan Africa: A Case Report from Benguela, Angola. Health Care & Global Health.2025;9(2):90-94.

DOI: 10.22258/hgh.2025.92.226

Corresponding author:
Antonino Sambundo Benjamin Kapitão
Address: Estrada Nacional Nº 100. Benguela, Angola.
Email: antoninocapitao66@gmail.com
Telephone: 940827155

History
Received: 08/07/2025
Revised: 13/07/2025
Accepted: 23/07/2025
Published Online : 15/08/2025
Internal peer review: Yes
External peer review: No

OPEN ACCESS
CC BY

Abstract

Ramsay Hunt syndrome (RHS) is a rare neurological complication of varicella-zoster virus infection, characterized by peripheral facial paralysis, otalgia, and auricular vesicular lesions. We report the case of a 31-year-old woman presenting with typical symptoms of the syndrome, clinically diagnosed at the General Hospital of Benguela, Angola, in a resource-limited setting. Treatment with acyclovir, prednisone, and supportive measures led to good functional recovery. This case highlights the importance of clinical recognition of RHS in regions with limited access to complementary tests, reinforcing the need for improved diagnostic surveillance and medical training.

Keywords: Hunt's syndrome; Herpes Zoster Oticus; Facial Paralysis, Peripheral; Varicella Zoster Virus Infection (Source: MeSH, NLM).

Resumen

El síndrome de Ramsay Hunt (SRH) es una complicación neurológica poco frecuente de la infección por el virus varicela-zóster, caracterizada por parálisis facial periférica, otalgia y lesiones vesiculares en el pabellón auricular. Describimos el caso de una mujer de 31 años que presentó las manifestaciones clásicas del SRH y fue diagnosticada clínicamente en el Hospital General de Benguela, Angola, en un entorno con recursos limitados. El tratamiento combinado con aciclovir, prednisona y medidas de soporte resultó en una recuperación funcional favorable. Este caso resalta la importancia del reconocimiento clínico del SRH en áreas con acceso limitado a pruebas complementarias y subraya la necesidad de fortalecer la vigilancia diagnóstica y la formación médica.

Palabras clave: Síndrome de Ramsay Hunt; Herpes Zóster Ótico; Parálisis Facial; Infección por el Virus de la Varicela-Zóster (Fuente: DeCS, BIREME).

Introduction

Ramsay Hunt syndrome (RHS), initially described by James Ramsay Hunt in 1907, is a rare neurological complication caused by the reactivation of the varicella-zoster virus (VZV) in the geniculate ganglion of the facial nerve[1]. Clinically, it manifests with the classic triad of peripheral facial paralysis, intense otalgia, and vesicular eruptions in the auricle or external auditory canal[2]. In addition to these hallmark features, complications such as sensorineural hearing loss, vertigo, dysgeusia, and postherpetic neuralgia are frequently reported, contributing to significant morbidity and impact on quality of life[3]. Despite its well-established viral etiology, RHS remains underdiagnosed in resource-constrained settings, where limited access to advanced diagnostic techniques such as PCR for VZV and the shortage of neurologists or otolaryngologists hinder early detection[4].

Viral reactivation is closely linked to immunosuppression due to conditions such as HIV/AIDS, diabetes mellitus, or the use of immunosuppressive therapies[5]. In Sub-Saharan Africa, where infectious diseases like HIV and tuberculosis are endemic, the prevalence of RHS may be underestimated due to symptom overlap with other neuropathies and prioritization of diseases with a higher epidemiological burden[6].

Globally, RHS accounts for approximately 12% of all peripheral facial palsies, with an estimated annual incidence of 5 cases per 100,000 individuals in high-income countries[7]. In contrast, Bell's palsy, its main differential diagnosis, has a significantly higher incidence (15–30 per 100,000/year), often with idiopathic etiology[8]. However, robust epidemiological data on RHS are scarce in Sub-Saharan Africa, reflecting a significant underreporting of cases. The World Health Organization (WHO) estimates that 3.9 million cases of herpes zoster (a precursor of RHS) occur annually in low- and middle-income countries, with increased risk among immunocompromised populations[9].

In Angola, where HIV prevalence is 1.8%[10], the coexistence of risk factors for VZV reactivation is substantial. However, the lack of specific surveillance systems for infectious neuropathies and limited access to diagnostic resources (such as viral serology and neuroimaging) hampers timely identification and management of RHS. Studies from neighboring countries like Mozambique and Zambia suggest that up to 40% of facial palsies attributed to Bell's palsy may actually be undiagnosed RHS, highlighting a critical gap in regional clinical management[11].

Documenting RHS cases in Angola is imperative for several reasons. First, the scarcity of reports in African scientific literature limits understanding of its local epidemiology and clinical patterns, perpetuating diagnostic gaps[11]. Second, treatment delays, often due to misdiagnosis as Bell's palsy, raise the risk of irreversible sequelae, such as permanent facial paralysis and hearing loss, with profound socioeconomic consequences in communities reliant on manual labor. Third, the absence of standardized therapeutic protocols in fragile healthcare systems worsens clinical outcomes. Robust evidence shows that combination therapy with antivirals (e.g., acyclovir) and corticosteroids, initiated within 72 hours of symptom onset, reduces complications by up to 70%[12]. However, in Angola, where access to essential medications is inconsistent and continuous medical education is limited, implementing these protocols faces structural barriers.

Moreover, the high prevalence of HIV in the country warrants special attention to syndromes associated with immunosuppression. Case reports like this not only raise awareness among healthcare providers regarding differential diagnoses but also reinforce the need for integrated public health policies linking the management of opportunistic infections to national HIV control programs.

Case Description

We report the case of a 31-year-old Black woman, married, born and residing in Benguela, Angola, who presented to the emergency department of the General Hospital of Benguela (HGB) with complaints of facial asymmetry (labial commissure deviation), severe right-sided ear pain, and vesicular lesions on the right auricle, with a duration of approximately four days.

The patient also reported intermittent episodes of vomiting and vertigo, worsening ear pain, and progressive hypoacusis. She denied changes in speech, visual acuity, or motor/sensory deficits in the limbs. Two weeks earlier, she had consulted a physician for right ear pain without facial asymmetry. Otoscopy was not documented, and empirical treatment was initiated with amoxicillin/clavulanate (500 mg/125 mg) and paracetamol (500 mg), administered every 8 hours for 10 days, with a working diagnosis of otitis media. There was no clinical improvement.

At admission, she was afebrile, hemodynamically stable, and had the following vital signs: blood pressure of 134/82 mmHg, heart rate of 86 bpm, oxygen saturation of 98%, and respiratory rate of 17 breaths per minute. On general examination, she was conscious, oriented, non-icteric, and non-cyanotic, with no relevant systemic findings. Examination of the right ear revealed otalgia, vesicular and pustular lesions with crusting on the auricle, and imbalance-type vertigo (Figure 1). Neurological assessment showed preserved peripheral reflexes and a peripheral facial palsy on the right side, classified as grade III on the House-Brackmann scale (Figure 2).

Due to lack of imaging and laboratory resources at HGB (e.g., CT, MRI, and herpes serology), the diagnosis was established clinically. The combination of peripheral facial

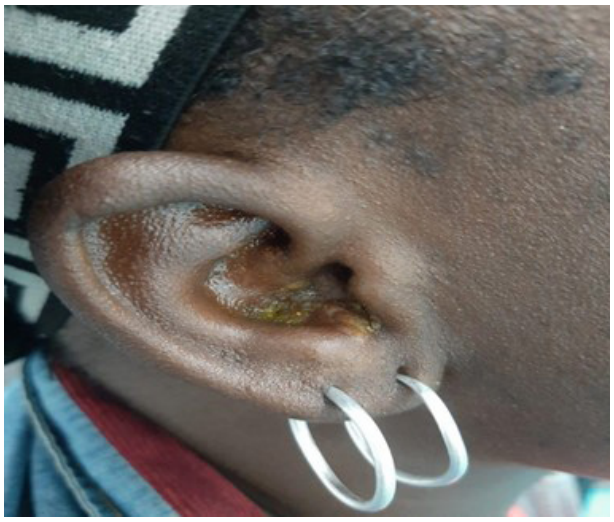

Figure 1. Vesicular and crusted lesions on the right auricle, typical of Ramsay Hunt syndrome (herpes zoster oticus).

palsy, otalgia, and characteristic auricular lesions was sufficient to confirm RHS. Differential diagnoses such as Bell's palsy and otitis media with facial palsy were ruled out due to the absence of signs typical of those conditions.

Treatment was immediately initiated and included:

1. Oral acyclovir 200 mg every 12 hours for 15 days;
2. Oral prednisone 5 mg every 12 hours for 5 days;
3. Oral diclofenac 50 mg every 8 hours for 10 days;
4. Adjunctive therapy with chewing gum to stimulate facial muscles and prevent atrophy.

The patient was followed up for 15 days, during which she showed progressive improvement in facial function and complete resolution of otalgia. At the end of the treatment, she presented with mild residual facial weakness, without

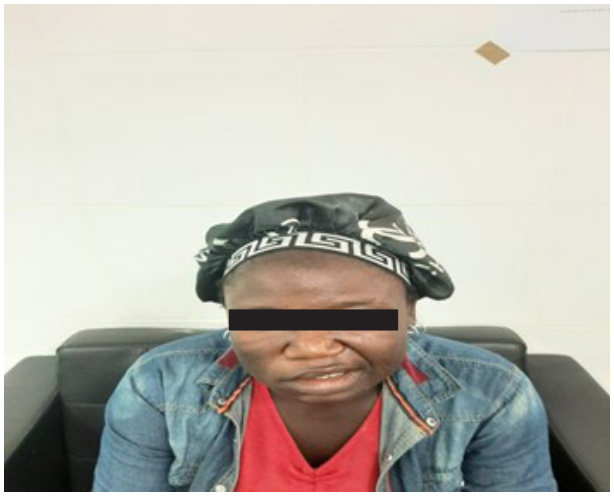

Figure 2. Right-sided peripheral facial paralysis (House-Brackmann grade III), with visible asymmetry of the labial commissure and incomplete eyelid closure.

Figure 3. Clinical improvement after treatment showing resolution of a mild residual facial weakness.

complications such as postherpetic neuralgia (Figure 3) (Figure 4).

The patient provided written consent for the anonymous publication of this case.

Discussion

RHS, also known as herpes zoster oticus, is a rare but clinically significant neurological condition caused by reactivation of VZV in the geniculate ganglion of the facial nerve (cranial nerve VII). This reactivation can result in a complex clinical picture of peripheral facial paralysis and vesicular lesions in the distribution of the facial nerve, frequently accompanied by auditory and vestibular symptoms. This case represents a textbook presentation of RHS and exemplifies the challenges of diagnosis and management in resource-limited settings.

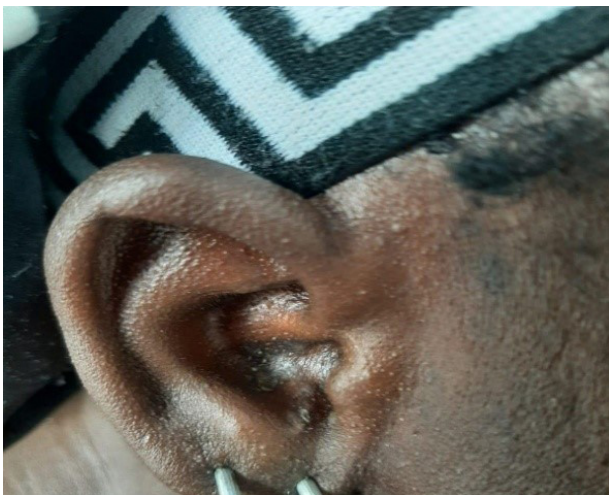

Figure 4. Clinical improvement after treatment showing resolution of auricular lesions.

Following primary infection (chickenpox), VZV remains latent in sensory ganglia for decades. Reactivation may be triggered by factors such as immunosuppression, stress, advanced age, and comorbidities. In RHS, reactivation in the geniculate ganglion, which contains motor and sensory fibers of the facial nerve, as well as connections to the vestibulocochlear nerve (cranial nerve VIII)—can lead to inflammation extending to the cochlea, vestibule, and middle ear[13].

Although more common in individuals over 60, this case highlights an atypical presentation in a previously healthy young adult, suggesting under-recognized prevalence in less-studied populations. The lack of robust epidemiological data in Sub-Saharan Africa further complicates prevalence estimates, making this report particularly valuable for regional medical literature[14].

Clinical Manifestations

Peripheral Facial Paralysis: Caused by facial nerve involvement, resulting in facial asymmetry, inability to close the ipsilateral eye, and loss of facial expression. In this case, the patient exhibited grade III facial paralysis on the House-Brackmann scale, indicating moderate dysfunction[15].

Auricular Vesicular Lesions: Vesicular eruptions in the auricle and external auditory canal are pathognomonic of RHS and reflect viral replication in the sensory distribution of the facial nerve[16].

Auditory and Vestibular Symptoms: Sensorineural hearing loss and vertigo, as seen in this patient, are indicative of vestibulocochlear nerve involvement, more common in severe RHS type II presentations[17].

Additional symptoms may include dysgeusia (taste alteration in the anterior two-thirds of the tongue), xerostomia (dry mouth), and xerophthalmia (dry eyes), due to autonomic involvement of the facial nerve[1].

Diagnostic Rationale

The diagnosis of RHS is primarily clinical, based on correlating neurological findings with dermatological signs. In this case, the combination of unilateral peripheral facial paralysis, severe otalgia, and ipsilateral vesicular lesions allowed for diagnosis even in the absence of imaging (CT, MRI) or serology[13]. Up to 50% of facial palsy cases due to herpes zoster are misdiagnosed initially, delaying antiviral therapy and worsening outcomes[16]. Early clinical recognition is crucial in low-resource environments.

Differential Diagnosis

Bell's Palsy: Idiopathic facial nerve palsy without vesicular eruptions or significant pain. The presence of vesicular lesions excluded this diagnosis[1].

Otitis Media with Facial Paralysis: Typically associated with middle ear inflammation, more frequent in children. Lack of otoscopic inflammatory signs and presence of vesicular rash made this unlikely[12].

Zoster Sine Herpete: A variant of VZV reactivation without skin lesions. This was excluded due to visible vesicles[14].

Other excluded conditions included cerebellopontine angle tumors and inflammatory or ischemic neuropathies, for which no clinical evidence was present.

Treatment and Management

The therapeutic approach includes antiviral therapy, corticosteroids, analgesics, and physical rehabilitation. The treatment regimen in this case was:

Antiviral (Acyclovir 200 mg BID for 15 days): Inhibits viral replication. Early initiation within 72 hours is linked to better outcomes[18].

Corticosteroids (Prednisone 5 mg BID for 5 days): Reduce inflammation and nerve edema, improving prognosis[19].

Analgesics (Diclofenac 50 mg TID for 10 days): Control of severe pain, often disabling in RHS.

Facial Physiotherapy: Chewing gum was used to stimulate muscle function and prevent atrophy[16].

Although therapy started after 96 hours from symptom onset, persistent active lesions justified intervention, aligning with international guidelines[20].

Prognosis

Complete recovery in RHS ranges from 50% to 70%, even with early treatment. Cochlear and vestibular involvement, as observed in this case, is associated with a worse prognosis[16]. The patient showed substantial recovery after 15 days of treatment, with only mild residual paresis, consistent with the literature.

Epidemiological Significance and Case Relevance

This may be the first documented RHS case in Angola, significantly enriching African and global medical literature. In low-income settings, underreporting and limited diagnostic access obscure the true burden of RHS.

Moreover, this case underscores the value of detailed clinical examination in low-resource contexts. Prompt recognition of vesicular lesions in the auricular region should always prompt thorough evaluation of facial and auditory function.

Conclusion

RHS is a rare yet clinically recognizable condition that relies heavily on accurate physical examination and targeted history-taking for diagnosis. This case reinforces the importance of early intervention, even where diagnostic tools are scarce, and emphasizes the need for broader awareness to reduce diagnostic delays.

The positive therapeutic outcome demonstrates the potential for functional recovery with proper management and structured follow-up. Furthermore, the publication of this case advances medical knowledge in Sub-Saharan Africa and supports the development of locally adapted, evidence-based clinical protocols.

Additional Information

Author Contributions: All authors contributed equally.

Conflicts of Interest: The authors declare no conflict of interest.

Funding: This research received no external funding.

Acknowledgments: We thank the patient for consenting to publication and the medical staff at the General Hospital of Benguela for their clinical support.

Data availability: Not applicable.

References

1. Sweeney CJ, Gilden DH. Ramsay Hunt syndrome. J Neurol Neurosurg Psychiatry 2001;71(2):149–54. DOI: 10.1136/jnnp.71.2.149.
2. Vargas IC, Frazão CQ, Gaboardi KR, et al. Síndrome de Ramsay Hunt: uma abordagem diagnóstica, evolução clínica e revisão. Braz J Health Rev 2023;6(5):21447–55. DOI: 10.34119/bjhrv6n5-173.
3. Gaudêncio M, Bertão MI, Carvalho A, et al. Ramsay Hunt Syndrome in a Patient with Ulcerative Colitis Treated with Infliximab. Eur J Case Rep Intern Med 2021;8(12):003032. DOI: 10.12890/2021_003032.
4. Malhotra R, Mudey A, Agrawal I. Clinical Features and Prognosis of Facial Palsy and Hearing Loss in Patients With Ramsay Hunt Syndrome. Cureus 2022;14(10):e30897. DOI:10.7759/cureus.30897.
5. The path that ends AIDS: UNAIDS Global AIDS Update 2023. Geneva: Joint United Nations Programme on HIV/AIDS; 2023. Licence: CC BY-NC-SA 3.0 IGO. https://thepath.unaids.org/ (accessed June 20, 2025).
6. Guseva AL, Zamergrad MV, Levin OS. [Ramsay Hunt syndrome]. Zh Nevrol Psikhiatr Im S S Korsakova 2020;120(9):151–7. DOI:10.17116/jnevro2020120091151.
7. Goswami Y, Gaurkar SS. Ramsay Hunt Syndrome: An Introduction, Signs and Symptoms, and Treatment. Cureus 2023;15(1):e33688. DOI:10.7759/cureus.33688.
8. Bento RE, Vellutini EA, Pahl FH, et al. [Topodiagnosis in peripheral facial paralysis]. Arq Neuropsiquiatr 1985;43(23):275–80. DOI:10.1590/s0004-282x1985000300006.
9. Wang Q, Yang L, Li L, et al. Willingness to Vaccinate Against Herpes Zoster and Its Associated Factors Across WHO Regions: Global Systematic Review and Meta-Analysis. JMIR Public Health Surveill 2023;9:e43893. DOI:10.2196/43893.
10. Solari PN. Epidemiological and Clinical Profile of HIV-Positive Patients Monitored at the General Hospital of Benguela – Angola, from January to December 2023. Rev Científica Clínica Sagrada Esperança 2025:32–7. DOI:10.70360/rccse..v.157.
11. Chinyathi AD, Chiweza KB, Kapembwa KC. Ramsay Hunt syndrome presenting as “a window” to HIV diagnosis in 48 year old African female: A case report. Med J Zambia 2017;44(1):54–57. https://www.ajol.info/index.php/mjz/article/view/157410.
12. Jeon Y, Lee H. Ramsay Hunt syndrome. J Dent Anesth Pain Med 2018;18(6):333–337. DOI: 10.17245/jdapm.2018.18.6.333.
13. Gilden DH, Kleinschmidt-DeMasters BK, Wellish M, et al. Neurological complications of the reactivation of varicella-zoster virus. N Engl J Med 2000;342(9):635–645. DOI:10.1056/NEJM200003023420906.
14. Yawn BP, Saddier P, Wollan PC, St Sauver JL, Kurland MJ, Sy LS. A population-based study of the incidence and complication rates of herpes zoster before zoster vaccine introduction. Mayo Clin Proc. 2007 Nov;82(11):1341-9. DOI:10.4065/82.11.1341.
15. House JW, Brackmann DE. Facial nerve grading system. Otolaryngol Neck Surg 1985;93(2):146–7. DOI: 10.1177/019459988509300202.
16. Murakami S, Honda N, Mizobuchi M, et al. Prognostic factors for recovery in Ramsay Hunt syndrome. Arch Otolaryngol Neck Surg 1997;123:848–52.
17. Crouch AE, Hohman MH, Moody MP, et al. Ramsay Hunt Syndrome. [Updated 2023 Aug 28]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK557409/
18. Kinishi M, Amatsu M, Mohri M, et al. Acyclovir improves recovery rate of facial nerve palsy in Ramsay Hunt syndrome. Auris Nasus Larynx 2001;28(3):223–6. DOI: 10.1016/S0385-8146(01)00055-4.
19. Monsanto RD, Bittencourt AG, Bobato Neto NJ, Beilke SC, Lorenzetti FT, Salomone R. Treatment and Prognosis of Facial Palsy on Ramsay Hunt Syndrome: Results Based on a Review of the Literature. Int Arch Otorhinolaryngol. 2016 Oct;20(4):394-400. DOI: 10.1055/s-0036-1584267.
20. Waheed D-E-N, Bolio A, Guillaume D, et al. Planning, implementation, and sustaining high coverage of human papillomavirus (HPV) vaccination programs: What works in the context of low-resource countries? Front Public Health 2023;11:1112981. DOI: 10.3389/fpubh.2023.1112981.