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# The Silent Sequelae: Irreversible Profound Sensorineural Hearing Loss Following Mumps Parotitis in a Pediatric Patient

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**A B S T R A C T**

Mumps-associated Sudden Sensorineural Hearing Loss (SSNHL) is a rare but catastrophic complication of epidemic parotitis, historically estimated to occur in 1 per 20,000 cases. Despite global vaccination efforts, the re-emergence of mumps in adolescent populations due to waning immunity poses significant otologic risks. This report documents a case of unilateral, profound SSNHL in a vaccinated pediatric patient, highlighting the diagnostic challenges when auditory symptoms manifest after the resolution of systemic parotitis. An 11-year-old Balinese male presented with acute-onset tinnitus and hearing loss in the left ear, occurring one week after the clinical resolution of bilateral parotid swelling. The patient had a history of basic immunization. Audiometric evaluation revealed profound sensorineural hearing loss in the left ear with a Pure Tone Average of 110 dB, while the right ear remained normal. Serological analysis confirmed acute mumps infection with a highly positive IgM titer (Index 11.0). Hematological profiling demonstrated leukopenia with a neutrophil shift. Despite a prompt and aggressive multimodal treatment protocol including high-dose intravenous methylprednisolone, oxygen therapy, and neurotrophic support, follow-up audiometry at 14 days revealed no functional improvement (Pure Tone Average 91.25 dB). In conclusion, mumps-induced SSNHL is characterized by rapid, irreversible cochlear destruction that frequently exhibits resistance to corticosteroid therapy. The dissociation between the resolution of systemic parotitis and the onset of otologic sequelae requires high clinical vigilance. This case emphasizes the critical need for serological confirmation in pediatric SSNHL and underscores the poor prognosis associated with this specific viral etiology.

## 1. Introduction

The auditory system serves as the primary conduit for human communication and environmental awareness; consequently, the acute loss of this sensory modality represents a profound functional and psychological crisis.[1] Sudden Sensorineural Hearing Loss (SSNHL) is clinically defined as a rapid, often catastrophic decline in auditory acuity of at least 30 decibels (dB) across three contiguous frequencies, manifesting within a narrow temporal window of 72 hours. This condition constitutes a true otologic emergency, with an estimated annual incidence ranging from 5 to 27 per 100,000 individuals in the United States alone, translating to tens of thousands of new cases annually.[2]

The etiology of SSNHL is remarkably heterogeneous, encompassing vascular occlusion, autoimmune mechanisms, traumatic insults, and neoplastic processes such as vestibular schwannoma.[3] However, despite rigorous diagnostic protocols, the vast majority of cases—approximately 80% to 90%—remain classified as idiopathic at the time of initial presentation, leaving clinicians to rely on empirical treatment algorithms. Within the subset of identified causes, viral etiologies occupy a prominent position, particularly in pediatric and young adult populations. Viral pathogens are estimated to account for approximately 10% of bilateral SSNHL cases and a significant proportion of unilateral presentations.[4] Among the diverse array of neurotropic viruses capable of inflicting auditory damage—including Cytomegalovirus (CMV), Herpes Simplex Virus (HSV), and Rubella—the Mumps virus (family Paramyxoviridae) remains a historically significant and clinically devastating pathogen.[5]

Mumps, an acute viral illness caused by a single-stranded RNA virus of the genus Rubulavirus, is classically characterized by painful swelling of the parotid glands (epidemic parotitis), fever, and malaise.[6] While the systemic manifestations of mumps are generally self-limiting, its capacity for extra-salivary complications poses serious health risks. These complications include orchitis, meningitis, pancreatitis, and, most notably for the otologist, sensorineural hearing loss. Historically referred to as mumps deafness, this complication is rare but carries a disproportionately high morbidity. Recent epidemiological analyses, such as those conducted on Japanese insurance claims databases, indicate that mumps-associated hearing loss occurs in approximately 1 out of every 668 mumps patients (an incidence of roughly 15 per 10,000 cases). Crucially, the risk profile appears to be age-dependent; the incidence of auditory complications is significantly higher in adolescents and adults (73.6 per 10,000) compared to pediatric populations (8.8 per 10,000). This statistical variance underscores that while children are the primary reservoir for infection, the otologic consequences may be more severe or more frequently recognized in older cohorts, or perhaps underreported in younger children unable to articulate unilateral hearing loss.[7]

The pathophysiology of mumps-associated hearing loss is distinct from other forms of viral labyrinthitis, characterized by its rapidity and destructive severity. Unlike bacterial labyrinthitis, which may spread via tympanogenic or meningogenic routes, the mumps virus typically accesses the inner ear via the hematogenous route. Following viremia, the virus penetrates the blood-labyrinth barrier, targeting the highly metabolic tissues of the cochlea. Experimental models and histopathological studies of temporal bones have elucidated the specific mechanisms of injury.[8] The virus exhibits a tropism for the stria vascularis and the organ of Corti. Upon invasion, the virus induces an acute endolymphatic labyrinthitis. This inflammatory cascade leads to the degeneration of the stria vascularis—the metabolic powerhouse of the cochlea responsible for maintaining the endocochlear potential—and the subsequent collapse of Reissner's membrane and the tectorial membrane.

The cellular damage is profound. Direct viral cytolysis results in the destruction of the outer hair cells and the supporting pillar cells within the organ of Corti. Furthermore, the virus may induce demyelination of the cochlear nerve, compounding the sensorineural deficit with a neural component. This multi-level assault on the peripheral auditory system explains the clinical presentation: a hearing loss that is typically rapid in onset, severe to profound in degree, and predominantly unilateral. The specific targeting of the basal turn of the cochlea initially may result in high-frequency loss, which rapidly progresses to involve all frequencies, resulting in a flat or corner audiogram indicative of total cochlear failure.

One of the most defining and distressing features of mumps-associated SSNHL is its prognosis. In the

broader context of idiopathic SSNHL, spontaneous recovery rates are relatively favorable, occurring in 32% to 65% of cases.[9] With the timely administration of systemic or intratympanic corticosteroids—the current gold standard of treatment—recovery rates can be bolstered further. In stark contrast, mumps-induced deafness is notoriously resistant to therapy. The damage to the organ of Corti is widely considered permanent. Prognostic studies have shown that the rate of complete recovery in mumps-associated hearing loss is approximately 1.3%, with over 96% of cases showing absolutely no response to therapeutic interventions. This resistance likely stems from the mechanism of cell death; whereas idiopathic SSNHL may involve reversible metabolic strain or edema, mumps infection leads to irreversible necrosis of the sensory neuroepithelium. Consequently, the clinical trajectory is almost invariably one of permanent, profound unilateral deafness (single-sided deafness), which poses significant rehabilitative challenges and impacts auditory localization and speech perception in noise.

The introduction of the Measles-Mumps-Rubella (MMR) vaccine has been one of the great triumphs of public health, dramatically reducing the global incidence of mumps and its complications. In nations with high vaccination coverage, mumps deafness has become an exceptionally rare clinical entity. However, the elimination of the disease has proven elusive. We are currently witnessing a shifting epidemiology characterized by sporadic outbreaks in vaccinated populations, particularly among adolescents and young adults. This phenomenon is attributed to several factors, including waning immunity (secondary vaccine failure) where antibody titers decline 10 to 15 years post-vaccination, and genotype mismatch between the vaccine strain and circulating wild-type viruses. This resurgence creates a dangerous diagnostic blind spot. Clinicians accustomed to the post-vaccination landscape may not immediately include mumps in the differential diagnosis of SSNHL, especially in patients with a documented immunization history. Furthermore, the classic sign of parotitis is absent in 30-40% of mumps infections (subclinical cases). Consequently, a vaccinated child presenting with sudden hearing loss without obvious facial swelling may be misdiagnosed as having idiopathic SSNHL, delaying the crucial serological confirmation required to understand the etiology and prognosis. The silent interval—a period ranging from days to weeks between the resolution of systemic symptoms and the onset of hearing loss—further obscures the causal link, potentially leading to diagnostic delays.[10]

In light of these diagnostic and therapeutic complexities, this study aims to report a case of profound, irreversible unilateral SSNHL in a vaccinated 11-year-old male following a mild course of parotitis. The novelty of this report lies in the detailed documentation of the silent interval between the resolution of parotitis and the onset of deafness, the serological confirmation in a vaccinated individual suggesting genotype mismatch or waning immunity, and the detailed analysis of the failure of high-dose corticosteroid therapy. This report serves as a critical alert for otolaryngologists to maintain a high index of suspicion for mumps in pediatric SSNHL and underscores the limitations of current therapeutic protocols for this specific viral etiology.

## 2. Case Presentation

Written informed consent was obtained from the parents/legal guardians of the patient for the publication of this case report and any accompanying clinical data, including audiograms and laboratory results. The nature and purpose of the publication were fully explained to the guardians, who granted permission for the material to be used for educational and scientific dissemination in a peer-reviewed journal. The authors certify that strict anonymity has been maintained; all direct personal identifiers have been removed or masked to protect patient

confidentiality. This report adheres to the ethical principles outlined in the Declaration of Helsinki and complies with the institutional ethical guidelines of Prof. Dr. I.G.N.G Ngoerah General Hospital.

An 11-year-old male student of Balinese ethnicity was admitted to the Emergency Department of Prof. Dr. I.G.N.G Ngoerah General Hospital, a tertiary referral center. The patient presented with a distressing and sudden-onset neurotologic profile, characterized primarily by unilateral tinnitus and a profound loss of auditory acuity in the left ear. The onset of these symptoms was acute and functionally disruptive; the patient explicitly recalled the moment of onset occurring three days prior to admission while he was engaged in daily activities at school. The subjective description of the auditory insult provided by the patient was clinically significant. He described the initial sensation as a high-pitched ringing (tinnitus), which rapidly evolved into a persistent sensation of aural fullness or blockage. In the local vernacular, the patient described this specific sensation as grebeg-grebeg. This term, while colloquial, provides critical semiological insight, often correlating with the acute perception of sensorineural drop or sudden pressure changes within the inner ear, distinct from the pain of otitis media. Importantly, the patient reported these symptoms solely in the left ear, with no subjective alteration in hearing sensitivity on the right side.

A reconstruction of the clinical timeline revealed a distinct biphasic pattern of illness, characteristic of mumps-associated complications. The acute otologic catastrophe was preceded by a systemic prodrome. Approximately one week prior to the onset of the auditory symptoms, the patient had developed the classic clinical stigmata of epidemic parotitis: bilateral swelling of the cheeks localized to the parotid region, accompanied by febrile episodes and odynophagia (pain on swallowing). Crucially, the parents reported that the systemic phase of the illness—the facial swelling and the fever—had largely resolved by the time the hearing loss manifested. This temporal dissociation creates a silent interval between the viremic phase and the neurotologic sequelae, a phenomenon that often provides a false sense of security to patients and guardians. The patient had returned to school, assuming a full recovery, only to be struck by the sudden deafness days later.

At the time of admission to the Emergency Department, the patient denied any accompanying vestibular symptoms. There were no reports of true vertigo (spinning sensation), dysequilibrium, nausea, or vomiting. This absence of vestibular involvement is diagnostically relevant, suggesting a pathology that selectively targets the cochlear division of the vestibulocochlear nerve (Cranial Nerve VIII) or the cochlear labyrinth, sparing the vestibular apparatus. Furthermore, the anamnesis effectively ruled out external insults; there was no history of recent head trauma, exposure to ototoxic medications, or exposure to high-intensity impulse noise that could account for the sudden acoustic failure.

The patient's medical background was non-contributory regarding prior otologic dysfunction; he had no history of chronic otitis media, congenital hearing deficits, or previous sudden hearing loss events. Of particular interest to the epidemiological context of this case was the patient's immunization history. The parents provided records indicating that the patient had completed the basic national immunization schedule up to the age of 9 months. However, the documentation regarding the booster dose of the MMR (Measles, Mumps, Rubella) vaccine—typically administered at school age (elementary school screening programs)—was ambiguous. This ambiguity raises a critical immunological hypothesis: despite the initial vaccination series, the patient may have experienced secondary vaccine failure due to waning immunity over the ensuing decade, or primary vaccine failure where seroconversion was never fully achieved. This gap in demonstrable long-term immunity renders the adolescent susceptible to

breakthrough infection, a phenomenon increasingly documented in recent mumps outbreaks.

Upon physical assessment, the patient appeared well-nourished and alert, demonstrating appropriate cognitive function with a Glasgow Coma Scale (GCS) of E4V5M6. The hemodynamic and respiratory parameters were stable, indicating that the acute systemic viremia had subsided. The recorded vital signs were: blood pressure 110/80 mmHg, heart rate 94 beats per minute, respiratory rate 20 breaths per minute, and a temperature of 36.4°C. Oxygen saturation was preserved at 98% on room air. The normothermic status further corroborated the history that the acute febrile phase of the parotitis had resolved.

A focused head and neck examination was conducted to rule out conductive causes of hearing loss and to assess the current status of the salivary glands. Examination of the external auditory canals revealed no abnormalities. The tympanic membranes bilaterally were intact, pearly grey in color, and mobile, with a preserved cone of light reflex. There were no signs of acute inflammation, such as hyperemia or bulging, nor was there any evidence of middle ear effusion, tympanic perforation, or trauma. This deceptively normal otoscopic appearance is a hallmark of sudden sensorineural hearing loss (SSNHL), shifting the diagnostic focus immediately to the inner ear or retrocochlear pathways. The oropharyngeal examination demonstrated tonsils of size T2/T2 with normal mucosa and no evidence of acute hyperemia. Inspection of the buccal mucosa specifically targeted the orifices of Stenson's ducts (the parotid duct openings); these appeared normal, with no expression of purulent discharge upon massage, ruling out active suppurative parotitis. Careful palpation of the parotid and submandibular regions was performed. Consistent with the history of resolved illness, the examination revealed no residual mass, induration, fluctuation, tenderness, or warmth. The resolution of the parotid swelling confirmed that the patient was in the post-acute or convalescent phase of the viral infection.

Bedside audiological assessment using 512 Hz tuning forks provided the first objective confirmation of the sensorineural nature of the hearing loss. The Rinne test was positive (+) bilaterally, meaning the patient perceived the sound of the tuning fork louder via air conduction (AC) than bone conduction (BC). In the context of hearing loss, a positive Rinne in the affected ear excludes a significant conductive block (such as cerumen impaction or ossicular fixation), implying that the deficit lies in the sensorineural pathway. In the Weber test, the sound was lateralized distinctly to the right side (the healthy ear). In patients with unilateral sensorineural hearing loss, the Weber test lateralizes to the better-hearing ear because the cochlea on the affected side has a higher threshold for neural excitation. Bone conduction duration was shortened on the left side compared to the examiner's normal ear. Collectively, this triad of tuning fork findings—Rinne positive, Weber lateralization to the healthy ear, and shortened Schwabach—constituted a strong clinical diagnosis of left-sided sensorineural hearing loss (SNHL).

To quantify the severity and configuration of the hearing loss, pure tone audiometry (PTA) and tympanometry were performed on the day of admission. The results confirmed the catastrophic nature of the auditory insult. The right ear (auricula dextra-AD) functioned as a healthy internal control, demonstrating normal hearing thresholds across the frequency range (250–8000 Hz) with a pure tone average of 12.5 dB. The audiogram for the left ear (auricula sinistra- AS) revealed a profound deficit. The patient exhibited a corner audiogram pattern—a term used to describe a remnant of hearing only at the lowest frequencies with no response at higher frequencies, or in this case, a near-total loss across the board. The pure tone average (PTA), calculated across the speech frequencies of 500 Hz, 1000 Hz, 2000 Hz, and 4000 Hz, was 110 dB. This level of hearing loss is

classified as profound (anacusis), effectively rendering the ear non-functional for speech communication. The drop was consistent across frequencies from 250 Hz to 8000 Hz. Bilateral Type A tympanograms were obtained. A Type A tracing indicates normal middle ear pressure and normal tympanic membrane compliance. This finding was pivotal as it objectively ruled out conductive pathologies like otitis media with effusion or negative middle ear pressure, firmly localizing the lesion to the cochlea or auditory nerve.

A comprehensive laboratory workup was initiated to assess the systemic impact of the infection and identify potential risk factors. The complete blood count (CBC) provided evidence consistent with a viral etiology. The patient exhibited leukopenia, with a white blood cell (WBC) count of. This suppression of the leukocyte count is a frequent hematological manifestation of acute viral viremia, reflecting temporary bone marrow suppression by the mumps virus. The differential count revealed a relative neutrophilia (73.5%) and lymphopenia (22.4%). Consequently, the Neutrophil-to-Lymphocyte Ratio (NLR) was elevated at 3.28. In the context of SSNHL, an elevated NLR is increasingly recognized as a prognostic biomarker of systemic stress and inflammatory intensity, often correlating with poorer recovery outcomes. The red blood cell indices, including Hemoglobin (14.3 g/dL) and Hematocrit (45.8%), were within normal limits, ruling out anemia as a contributing factor to cochlear ischemia. Investigation of metabolic risk factors revealed a Total Cholesterol of 167 mg/dL (<200 mg/dL) and HDL of 52 mg/dL. However, the Low-Density Lipoprotein (LDL) was notably elevated at 119 mg/dL (>100 mg/dL). While not the primary etiology, elevated LDL is a known risk factor for microvascular compromise, potentially rendering the cochlear microcirculation more susceptible to the ischemic insults caused by viral inflammation. To definitively establish the etiology, specific IgM antibody testing for the Mumps virus was performed. The assay returned a strongly positive result with an Index of 11.0. This high titer is diagnostic of a recent, acute infection. The presence of specific IgM antibodies confirms that the clinical parotitis observed one week prior was indeed caused by the mumps virus, validating the diagnosis of mumps-associated sudden sensorineural hearing loss. This serological evidence was crucial in moving the diagnosis from idiopathic SSNHL to a specific viral etiology, thereby explaining the severity and potential irreversibility of the condition.

Upon admission, recognizing the catastrophic nature of the auditory insult and the narrow therapeutic window inherent to sudden sensorineural hearing loss (SSNHL), the clinical team initiated an aggressive, multimodal salvage protocol. The treatment strategy was designed to target the multiple potential pathophysiological pathways of cochlear injury—specifically inflammation, hypoxia, and neurodegeneration—while simultaneously managing the systemic physiology of the patient. The cornerstone of the pharmacological intervention was high-dose systemic corticosteroid therapy, widely regarded as the gold standard for acute cochlear rescue. The rationale for this approach lies in the potent ability of glucocorticoids to stabilize the blood-labyrinth barrier, downregulate the expression of pro-inflammatory cytokines within the cochlear microenvironment, and reduce edema in the stria vascularis. Immediately upon admission, the patient was started on intravenous Methylprednisolone at a dosage of 60 mg per day. This loading dose was selected to achieve rapid therapeutic plasma concentrations, essential for penetrating the blood-perilymph barrier, which is often compromised during the acute viremic phase. This high-dose intravenous regimen was maintained for the first 72 hours (3 days) to maximize the anti-inflammatory effect during the critical acute phase.

**Table 1. Summary of Clinical Findings on Admission**

| PARAMETER | FINDING / VALUE |
|---|---|
| **PATIENT DEMOGRAPHICS & HISTORY** | |
| Patient Profile | 11-year-old Male, Balinese Ethnicity |
| Chief Complaint | Sudden onset tinnitus and hearing loss (Left Ear) |
| Onset Duration | 3 days prior to admission |
| Prodromal History | Bilateral parotitis (resolved), fever, odynophagia (1 week prior) |
| Vaccination Status | Basic immunization complete (9 months); Booster status ambiguous |
| **PHYSICAL EXAMINATION** | |
| Vital Signs | BP 110/80 mmHg, HR 94 bpm, RR 20 bpm, Temp 36.4°C |
| Parotid Region | No residual mass, no tenderness, no warmth (Resolved phase) |
| Otoscopy (Bilateral) | Normal; Intact tympanic membrane, pearly grey, cone of light (+) |
| Tuning Fork Tests | Rinne (+/+), Weber (Lateralized to Right), Schwabach (Shortened Left) |
| **AUDIOLOGICAL ASSESSMENT** | |
| Pure Tone Average (Right Ear) | 12.5 dB (Normal Hearing) |
| Pure Tone Average (Left Ear) | 110 dB (Profound Sensorineural Hearing Loss) |
| Audiogram Pattern (Left) | Corner Audiogram (Profound loss across all frequencies 250-8000 Hz) |
| Tympanometry | Type A (Bilateral) - Normal middle ear compliance |
| **LABORATORY PROFILING** | |
| Leukocytes (WBC) | 1.96 × $10^3$/µL (Leukopenia) |
| Neutrophil / Lymphocyte Ratio | 3.28 (Elevated) |
| Mumps Serology (IgM) | Positive (Index 11.0) |
| Lipid Profile (LDL) | 119 mg/dL (Elevated) |

Following the initial three-day intensive phase, the protocol transitioned to a tapering strategy to mitigate potential adrenal suppression and systemic side effects while maintaining anti-inflammatory pressure. The dosage was reduced to 30 mg/day intravenously for the subsequent three days. Upon completion of the intravenous course, the patient was switched to an oral maintenance regimen consisting of Methylprednisolone 8 mg administered every 12 hours. This graduated step-down approach ensured that the cochlea was not subjected to a rebound inflammatory effect, a phenomenon that can exacerbate cellular apoptosis in the organ of Corti.

Recognizing that the stria vascularis is one of the most metabolically active tissues in the human body, maintaining adequate oxygenation is critical for hair cell survival, particularly when microvascular perfusion is compromised by viral endotheliitis. To address cochlear hypoxia, the patient was placed on a regimen of normobaric oxygen inhalation. High-flow oxygen was administered at a rate of 15 liters per minute (LPM) for 15-minute intervals every 6 hours. The objective of this intermittent high-flow therapy was to increase the partial pressure of oxygen ($pO_2$) in the perilymph via diffusion from the round window and systemic circulation. By enhancing the oxygen

gradient, the clinical team aimed to salvage cochlear hair cells that were metabolically stunned but not yet necrotic (the ischemic penumbra), thereby potentially reversing functional loss in areas where cellular architecture remained intact.

To complement the anti-inflammatory and oxygenation strategies, the protocol included specific neuroprotective measures. The vestibulocochlear nerve (CN VIII) is highly susceptible to demyelination during viral labyrinthitis. Consequently, Mecobalamin (Vitamin B12) at a dose of 500 mcg was administered, initially via the intravenous route and subsequently as oral maintenance. Mecobalamin serves as an essential cofactor in the synthesis of methionine and the maintenance of neural myelin sheaths, theoretically promoting nerve regeneration and optimizing neural transmission in the surviving auditory pathways. Furthermore, general supportive measures were strictly enforced to optimize the patient's systemic physiology. The patient was placed on bed rest to minimize total body metabolic demand, redirecting energy substrates to the healing process. Concurrently, a low-salt diet was prescribed. Sodium restriction is a standard otologic precaution intended to prevent or manage secondary endolymphatic hydrops—a condition of fluid imbalance in the inner ear that can fluctuate hearing thresholds and exacerbate tinnitus.

The clinical trajectory of the patient revealed a dissociation between subjective symptom relief and objective functional recovery, a pattern frequently observed in severe viral labyrinthitis. By the second day of hospitalization, the patient reported a distinct subjective reduction in the intensity of tinnitus. The initially distressing high-pitched ringing had dampened, and the patient perceived a slight improvement in the fullness sensation. Furthermore, the systemic sequelae of the prodromal mumps infection—fever, malaise, nausea, and vomiting—remained absent, confirming that the systemic viremia had resolved. This symptomatic improvement provided initial optimism regarding the efficacy of the anti-inflammatory regimen. However, the final audiological assessment performed on Day 10 presented a starkly different reality, underscoring the destructive potency of the mumps virus. Despite the aggressive, multi-pronged therapeutic intervention, repeat pure tone audiometry demonstrated persistent severe-to-profound hearing loss in the affected ear. On the left ear (affected), the functional gain was negligible. The pure tone average (PTA) measured 91.25 dB via air conduction and 77.5 dB via bone conduction. While technically an improvement from the admission threshold of 110 dB, clinically, this remains within the very severe to profound range. The persistence of a pronounced air-bone gap or lack of response at high frequencies indicated that the damage to the organ of Corti was extensive and irreversible. The minimal improvement likely reflected a reduction in transient edema rather than the regeneration of sensory hair cells. The contralateral ear remained stable and within normal limits, with an air conduction threshold of 21 dB, confirming the strictly unilateral nature of the pathology.

The patient was discharged with a final diagnosis of irreversible post-mumps sudden sensorineural hearing loss. The resistance to high-dose steroids confirmed the necrotic, rather than merely inflammatory, mechanism of the viral injury. Recognizing the long-term implications of single-sided deafness (SSD) on auditory localization and speech understanding in noise, a rehabilitation plan was initiated. A hearing aid trial was conducted one month later. During the fitting, the patient reported subjective improvement in sound awareness and environmental orientation. However, the benefit was limited to sound detection rather than speech discrimination, a common limitation when amplifying a dead cochlea. Tragically, despite the potential benefit of amplification or advanced solutions like CROS (Contralateral Routing of Signal) aids, the family was forced to defer procurement of the device due to

financial constraints. This conclusion highlights not only the medical severity of mumps deafness but also the socioeconomic barriers that can prevent pediatric patients from accessing essential auditory rehabilitation.

**Table 2. Diagnosis, Treatment Protocol, Follow-up, and Final Outcome**

| CLINICAL PHASE | DESCRIPTION & PROTOCOL |
|---|---|
| **I. DIAGNOSIS** | |
| **Primary Diagnosis** | **Sudden Sensorineural Hearing Loss (SSNHL), Left Ear**<br>*Etiology: Post-Mumps Parotitis Complication* |
| **Diagnostic Criteria Met** | ▪ **Audiometric:** >30 dB loss across 3 contiguous frequencies within 72 hours.<br>▪ **Serological:** Positive IgM Mumps Titer (Index 11.0).<br>▪ **Temporal:** Onset 7 days post-parotitis resolution. |
| **II. THERAPEUTIC INTERVENTION (INPATIENT)** | |
| **Corticosteroid Protocol**<br>(Systemic Anti-inflammatory) | ▪ **Day 1-3:** Inj. Methylprednisolone 60 mg/24h (IV)<br>▪ **Day 4-6:** Inj. Methylprednisolone 30 mg/24h (IV) [Tapering]<br>▪ **Discharge:** Oral Methylprednisolone 8 mg q12h |
| **Adjuvant Therapies** | ▪ **Oxygenation:** Normobaric O2 inhalation (15 LPM, 15 mins q6h)<br>▪ **Neurotrophic:** Mecobalamin 500 mcg (IV/Oral)<br>▪ **Supportive:** Bed rest, Low-salt diet |
| **III. FOLLOW-UP & EVALUATION** | |
| **Subjective Response**<br>**(Day 2 - Day 7)** | Patient reported reduction in tinnitus intensity.<br>**No subjective improvement** in hearing acuity. |
| **Audiometric Evaluation**<br>**(Day 10 - March 24)** | **Left Ear (AS):** Persistent Profound SNHL<br>**PTA: 91.25 dB** (vs. 110 dB at admission)<br>**Bone Conduction: 77.5 dB**<br>*Interpretation: Minimal/Non-significant recovery.* |
| **IV. FINAL OUTCOME & PROGNOSIS** | |
| **Final Status** | **Irreversible Profound Sensorineural Hearing Loss (Left)** |
| **Rehabilitation Plan** | Hearing Aid trial conducted<br>**Result:** Marginal benefit in sound awareness; speech discrimination remained poor.<br>**Recommendation:** CROS (Contralateral Routing of Signal) Hearing Aid or Cochlear Implantation (Deferred by family). |

## 3. Discussion

This study serves as a stark illustration of the destructive potency of the mumps virus (*Rubulavirus* family) within the delicate microenvironment of the inner ear. While the clinical presentation—rapid, unilateral, and profound sensorineural hearing loss—is phenotypically similar to idiopathic sudden sensorineural hearing loss (SSNHL), the underlying pathophysiology of mumps deafness involves a distinct and far more aggressive cellular mechanism.[11] The pathogenesis begins with hematogenous dissemination (Figure 1). The mumps

virus, while classically associated with glandular tropism (parotitis, orchitis), is intrinsically neurotropic. During the viremic phase, the virus breaches the blood-labyrinth barrier, likely gaining entry via the capillary network of the stria vascularis. The stria vascularis acts as the metabolic powerhouse of the cochlea, responsible for maintaining the endocochlear potential essential for hair cell transduction. Once inside the cochlear duct, the virus initiates a fulminant endolymphatic labyrinthitis. Historical temporal bone histopathology studies derived from patients with mumps deafness have provided a post-mortem window into this devastation. These studies consistently demonstrate a triad of destruction: severe atrophy of the stria vascularis, collapse of the vestibular (Reissner's) membrane, and the total degeneration of the organ of Corti.[12]

A critical mechanistic hypothesis for the suddenness of the hearing loss is the concept of metabolic strangulation.[13] The viral invasion triggers an intense inflammatory edema within the stria vascularis. Given the confined bony architecture of the cochlea, this edema likely compresses the microvasculature, leading to secondary ischemic necrosis. Unlike apoptosis (programmed cell death), which can sometimes be halted, necrosis results in the immediate and chaotic death of the outer and inner hair cells. Since mammalian cochlear hair cells lack the capacity for spontaneous regeneration, this loss is permanent. In this patient, the hematological finding of leukopenia (WBC) serves as a systemic corollary to this local destruction. It indicates a profound viral suppression of the bone marrow, reflecting the intensity of the viremic phase that coincided with the cochlear insult. This systemic footprint reinforces the conclusion that the hearing loss was not merely a coincidental idiopathic event, but the direct sequela of a potent systemic viral assault.[14]

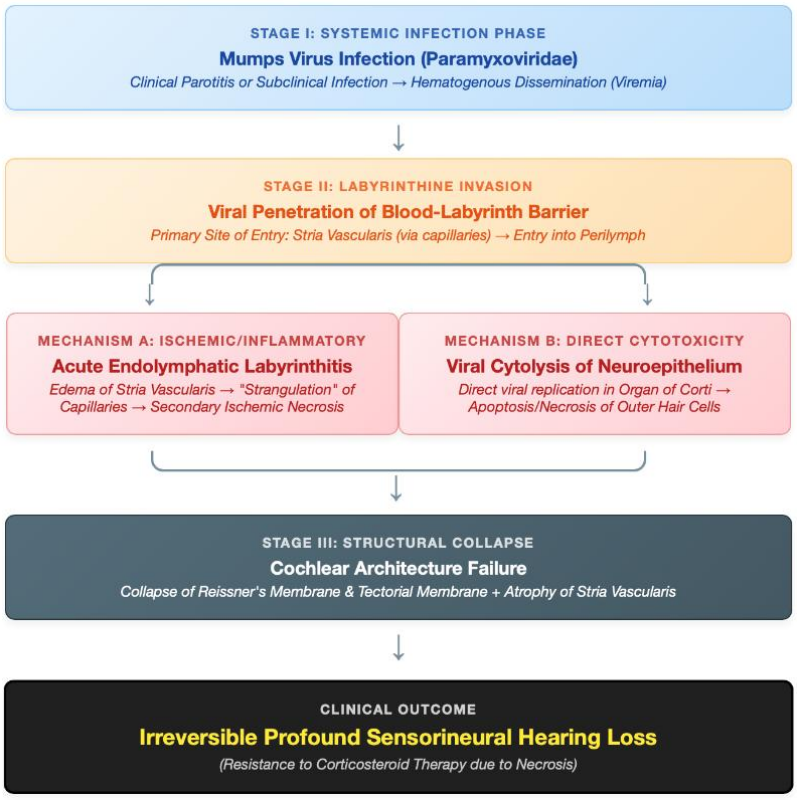


Figure 1. Pathophysiology of mumps-induced cochlear destruction.

One of the most clinically instructive aspects of this case is the temporal dissociation between the systemic illness and the otologic catastrophe. The patient experienced the onset of tinnitus and hearing loss approximately one week after the clinical resolution of the parotid swelling. This silent interval—the latency period between the glandular phase and the neuropathic phase—represents a significant diagnostic pitfall. Literature indicates that mumps-associated hearing loss can manifest anywhere from 4 days prior to the onset of parotitis to 18 days after its resolution. This wide temporal window creates a cognitive disconnect for the clinician and the patient. In this case, the parents and the child believed the illness had passed, only to be struck by a devastating sequela during the convalescent period.[15]

Furthermore, the diagnostic challenge is compounded by the phenomenon of subclinical infection. Epidemiological data suggest that 30% to 40% of mumps infections occur without the pathognomonic sign of parotitis. In such silent systemic cases, sudden hearing loss may be the *only* presenting symptom. If a clinician relies solely on a history of facial swelling, the diagnosis will be missed. This underscores the indispensable role of serological testing. In our case, the physical examination of the parotid region at the time of admission was unremarkable. Without the specific investigation for mumps IgM, this case would almost certainly have been misclassified as idiopathic SSNHL. The highly positive IgM titer (Index 11.0) served as the cornerstone of the etiological diagnosis. This finding advocates for a paradigm shift in the workup of pediatric SSNHL: serological screening for mumps should be mandatory, regardless of the presence or absence of active parotitis, to ensure accurate etiological classification and counseling.[16]

Perhaps the most disturbing element of this case is that it occurred in a child with a history of basic immunization. The assumption that vaccination confers absolute immunity often leads to the premature exclusion of mumps from the differential diagnosis.[17] However, this case highlights the growing public health reality of secondary vaccine failure. Current immunological models suggest that vaccine-derived immunity to mumps is not lifelong. Neutralizing antibody titers are known to wane significantly over a period of 10 to 15 years post-vaccination. This waning immunity creates a vulnerability gap specifically in the adolescent and young adult population—the exact demographic of our 11-year-old patient. If the primary series was completed in infancy (such as at 9 months and 18 months), the protective titers may have fallen below the threshold for sterility by the time the child reached pre-adolescence, particularly in the absence of a recent booster or natural exposure.

Additionally, the issue of genotype mismatch cannot be ignored. The wild-type mumps virus circulating in the community may have evolved distinct antigenic properties compared to the vaccine strain (typically the Jeryl Lynn or Urabe strains). This antigenic drift allows the wild-type virus to evade the host's immune memory, leading to breakthrough infections. This case serves as a critical signal to the pediatric and otolaryngology communities: a documented vaccination history does not rule out mumps. The exclusion of mumps based on immunization status alone is a clinical error. Consequently, there is a pressing need to re-evaluate vaccination schedules, potentially advocating for a routine adolescent booster dose (a third MMR) to close the immunity gap and protect this vulnerable age group from debilitating neurological complications.[18]

The prognosis for mumps-associated SSNHL is historically and consistently poor. While idiopathic SSNHL has a spontaneous recovery rate of up to 65%—often bolstered by corticosteroid therapy—mumps deafness is characterized by its resistance to treatment.[19] In the case of Patient PPJ, despite the prompt initiation of a comprehensive, high-dose intravenous corticosteroid protocol combined with

oxygen therapy and neurotrophic support, there was no functional recovery of hearing. This therapeutic failure (steroid resistance) provides insight into the mechanism of injury. Corticosteroids act primarily by reducing inflammation and edema. In idiopathic SSNHL, where the pathology may involve reversible metabolic stress or synaptic blockade, reducing inflammation allows the hair cells to recover function. However, in mumps deafness, the virus causes direct cytolysis. Once the hair cell membrane is ruptured and the cell undergoes necrosis, the damage is structural and absolute. No amount of anti-inflammatory medication can resurrect a necrotic cell. The persistence of the profound hearing loss (PTA >90 dB) at the follow-up evaluation confirms that the cochlear reserve was depleted. This outcome aligns with literature citing a recovery rate of less than 1.3% for this specific etiology. Clinicians must be transparent with parents regarding this prognosis to manage expectations realistically, moving the conversation quickly from cure to rehabilitation.

The primary limitation of this case report is the absence of high-resolution Magnetic Resonance Imaging (MRI) of the internal auditory canal. While the diagnosis was firmly established via serology and audiometry, an MRI would have provided valuable visual confirmation of cochlear inflammation (e.g., fluid-attenuated inversion recovery [FLAIR] hyperintensity) and definitively ruled out retrocochlear pathologies like vestibular schwannoma or cochlear nerve aplasia. Additionally, the follow-up period was limited to one month; a longer surveillance period would be beneficial to monitor for *labyrinthitis ossificans*, a common sequela of suppurative or viral labyrinthitis that can complicate future cochlear implantation.[20]

The absolute failure of current standard-of-care protocols (steroids) mandates a search for better interventions. Future research should investigate the utility of combining high-dose systemic steroids with immediate intratympanic antiviral administration, although the window for preventing necrosis is likely extremely narrow. Furthermore, the role of Cochlear Implantation (CI) in Single-Sided Deafness (SSD) for children warrants aggressive exploration. Currently, SSD is often managed expectantly, but the consequences of auditory deprivation include deficits in sound localization and speech development. Studies focusing on the quality-of-life improvements in children with mumps-induced SSD who undergo early implantation could change the management paradigm from acceptance to auditory restoration.

**4. Conclusion**

This case report documents a catastrophic clinical event: the onset of irreversible, profound sensorineural hearing loss in a vaccinated 11-year-old male, caused by a breakthrough mumps infection. The case challenges several prevailing clinical assumptions. It demonstrates that mumps can strike the inner ear even after the systemic symptoms have resolved (the silent interval), that vaccination does not guarantee immunity against neurological complications (secondary vaccine failure), and that the resulting hearing loss is largely impervious to standard corticosteroid rescue therapies. The defining clinical lesson of this report is the necessity for diagnostic vigilance. Otolaryngologists must maintain a high index of suspicion for mumps in any pediatric or adolescent patient presenting with sudden hearing loss, regardless of vaccination status or the presence of active parotitis. The inclusion of IgM serology in the standard workup is not merely academic; it is essential for accurate diagnosis and prognosis. Ultimately, because the hearing loss is permanent and treatment is largely futile, the emphasis must shift back to prevention. This case underscores the fragility of herd immunity and suggests that public health policies may need to prioritize adolescent booster vaccinations to eliminate the immunity gaps that allow this virus to continue claiming the hearing of children. Until such preventative measures are universal, early

identification and rapid rehabilitation with hearing technologies remain our only recourse for these silent victims of a preventable pathogen.

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