



Deep Neck Abscess with Concurrent Orbital and Subgaleal Extension Secondary to Odontogenic Infection: A Case Report

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ABSTRACT

Deep neck infections (DNIs) originating from odontogenic sources are common, but extensive spread involving concurrent orbital and subgaleal spaces is rare. DNIs can lead to life-threatening complications, particularly in patients with comorbidities like diabetes mellitus (DM) and chronic kidney disease (CKD), which impair immune function. This report details a complex case of DNI with unusual superior extension. A 44-year-old male with poorly controlled type 2 DM and CKD presented with a two-day history of rapidly progressing left facial, submandibular, parotid, and orbital swelling, associated with fever, trismus, and severe pain, originating from a carious mandibular molar. CT imaging confirmed an extensive abscess involving the left masticator, submandibular, sublingual, parotid, and parapharyngeal spaces, with contiguous spread to the left preseptal orbital space and the fronto-temporo-parietal subgaleal space. Multidisciplinary management involved urgent surgical drainage of the submandibular and subgaleal abscesses, odontectomy of the offending molar, broad-spectrum intravenous antibiotics (*Citrobacter amalonaticus* and *Proteus hauseri* identified on culture), intensive glycemic control, hemodialysis, and supportive care. In conclusion, this case highlights the potential for aggressive craniofacial spread of odontogenic DNIs, particularly in immunocompromised individuals. Concurrent orbital and subgaleal extension represents a rare and serious complication. Prompt diagnosis with imaging, aggressive multidisciplinary surgical and medical management, including addressing underlying comorbidities, were crucial for a successful outcome.

1. Introduction

Deep neck infections (DNIs) are a significant clinical entity, involving the potential spaces delineated by the layers of the cervical fascia. This encompasses a spectrum of conditions, ranging from cellulitis, an inflammation of soft tissues, to the more severe abscess formation, which is a localized collection of pus. While the incidence of DNIs has decreased notably with the widespread availability and use of antibiotics, they remain a serious health concern. This is due to their capacity for rapid

progression and the potential to give rise to life-threatening complications. These complications can include airway obstruction, a critical condition where the flow of air into the lungs is blocked; mediastinitis, an inflammation of the mediastinum (the space in the chest between the lungs); sepsis, a life-threatening condition caused by the body's overwhelming response to an infection; and jugular vein thrombosis, the formation of a blood clot in the jugular vein. The etiology of DNIs is multifactorial. In adults, the most common cause is infection originating from the teeth



(odontogenic infection), accounting for 50-70% of cases. These infections typically arise from infected mandibular molars. Other causes include tonsillitis, an inflammation of the tonsils; sialadenitis, an inflammation of the salivary glands; trauma; or other infections occurring in the head and neck region. The submandibular space is frequently involved in DNIs. This is often a consequence of infections originating from the second or third mandibular molars, whose roots are located below the attachment of the mylohyoid muscle. From this initial site, the infection can spread along the fascial planes to adjacent anatomical spaces. These spaces include the parotid space, housing the parotid gland; the parapharyngeal space, located beside the pharynx; the masticator space, associated with the muscles of mastication; and the sublingual space, situated below the tongue.¹⁻³

While the spread of DNIs typically occurs in a contiguous manner along these fascial planes, superior extension, or spread towards the upper regions of the head and neck, is less common. However, when it does occur, it can lead to severe and complex complications. Orbital involvement, manifesting as cellulitis or abscess within the eye socket, is one such complication. This can arise through several mechanisms: direct spread from adjacent sinuses such as the maxillary or ethmoid sinuses, hematogenous spread (through the bloodstream) via valveless facial veins, or indirectly via the pterygopalatine and infratemporal fossae from masticator space infections. Another rare but serious complication is the formation of a subgaleal abscess. This is a collection of pus in the potential space between the periosteum of the cranium (the membrane covering the skull bones) and the galea aponeurotica (a tough, fibrous layer of the scalp). Subgaleal abscesses typically result from trauma, infections of the scalp, neurosurgical procedures, or hematogenous seeding. However, they can also occasionally occur due to the superior spread of DNIs or as a consequence of otogenic infections (infections of the ear). The

severity and the risk of complications from DNIs are significantly influenced by the presence of underlying systemic comorbidities, particularly diabetes mellitus (DM) and chronic kidney disease (CKD). These conditions are known to impair the body's immune function, thereby predisposing individuals to more severe infections and poorer outcomes. Diabetes mellitus, characterized by hyperglycemia (elevated blood sugar levels), impairs various aspects of neutrophil function. Neutrophils are a type of white blood cell crucial for the immune response. The impairment includes reduced adhesion, chemotaxis (movement towards a chemical stimulus), and phagocytosis (the engulfment and destruction of pathogens), all of which are essential for fighting off infections. This dysfunction increases the susceptibility to more aggressive infections, more extensive spread, and poorer overall outcomes.⁴⁻⁷

Chronic kidney disease also contributes to a state of immunocompromise. The management of DNIs in patients with these comorbidities often necessitates a more aggressive and multidisciplinary approach. This highlights the importance of considering the patient's overall health status in the treatment strategy.⁸⁻¹⁰ This case report presents a unique and complex instance of an extensive DNI. The infection originated from an infected mandibular molar in a 44-year-old male patient with type 2 DM and CKD. What makes this case particularly noteworthy is the concurrent spread of the infection to involve several anatomical spaces: the parotid, parapharyngeal, masticator, preseptal orbital, and subgaleal spaces.

2. Case Presentation

This case report details the presentation, clinical findings, and diagnostic evaluation of a 44-year-old male patient who presented with a complex and extensive deep neck infection (DNI) originating from an odontogenic source. The patient's clinical picture was further complicated by significant comorbidities, contributing to the severity and extent of the infectious



process. A thorough analysis of the patient's demographics, anamnesis (history), physical examination, laboratory findings, and imaging studies provides a comprehensive understanding of the case. The patient was a 44-year-old male. This age is relevant as DNIs can occur across a broad age spectrum, but certain age groups may be more predisposed to specific etiologies or complications. The patient's gender is male. While DNIs can affect both genders, some studies suggest a slight male predominance, potentially related to occupational exposures or lifestyle factors influencing oral hygiene. His ethnicity/origin was identified as a Pyruvate sector employee. While ethnicity itself may not be a direct risk factor for DNIs, socioeconomic factors, access to healthcare, and cultural practices related to oral hygiene within specific populations can indirectly influence susceptibility to odontogenic infections. The patient's occupation as a pyruvate sector employee is not directly linked to DNIs, but it is important to note for the overall patient context. The patient's chief complaint was rapidly progressing swelling involving the left face, jaw, and eye, accompanied by severe pain. This symptom complex is highly suggestive of a rapidly evolving infectious process in the deep tissues of the head and neck. The acute onset and rapid progression are critical indicators of the potential for serious complications, such as airway compromise or spread to adjacent anatomical spaces. The history of present illness revealed that the patient experienced the onset of symptoms approximately 2 days prior to presentation. The symptoms were characterized by significant swelling of the left mandible, left cheek, and periorbital region. The rapid development of swelling in these interconnected anatomical areas suggests a spreading infection along fascial planes. The patient also reported worsening of left cheek and periorbital region swelling for 1 day. This progression highlights the aggressive nature of the infection. He described the pain as biting and severe, underscoring the intensity of the inflammatory response. The patient also

reported difficulty opening his mouth (trismus). Trismus, or limitation of jaw opening, is a common finding in DNIs, particularly those involving the masticator space. It results from inflammation or infection of the muscles of mastication. The patient also reported dysphagia, noting difficulty swallowing both solids and liquids. Dysphagia indicates involvement of structures in the oropharynx or the spread of infection to adjacent spaces, potentially affecting the swallowing mechanism. A detailed dental history elicited that the patient had a history of caries in the left second mandibular molar approximately 1 week prior to presentation. This finding is crucial as odontogenic infections are the most common cause of DNIs. Carious teeth, especially mandibular molars, provide a portal of entry for bacteria to invade the deeper tissues of the neck. The patient's past medical history was significant for Type 2 Diabetes Mellitus (poorly controlled) and Chronic Kidney Disease (end-stage, on maintenance hemodialysis). These comorbidities are highly relevant in the context of DNIs. Diabetes mellitus, particularly when poorly controlled, impairs the immune system, predisposing individuals to more severe and rapidly spreading infections. Hyperglycemia affects neutrophil function, including chemotaxis, phagocytosis, and intracellular killing of bacteria. Chronic kidney disease also contributes to an immunocompromised state. End-stage renal disease requiring hemodialysis further increases the risk of infection due to uremia-associated immune dysfunction and the need for invasive procedures (e.g., hemodialysis catheter placement). A review of systems revealed that the patient was alert and oriented with a Glasgow Coma Scale (GCS) score of 15/15, indicating no acute neurological compromise. Moderate general condition was noted. The GCS is a standardized neurological scale used to assess level of consciousness. A score of 15 indicates full alertness. The patient's general condition provides context for the overall impact of the infection on his systemic health. The patient's vital



signs revealed a blood pressure of 130/90 mmHg, a heart rate of 80 bpm, a respiratory rate of 20 breaths/minute, and a temperature of 37.0°C. The elevated heart rate (tachycardia) is a common finding in infections and reflects the body's systemic inflammatory response. The temperature of 37.0°C is within the normal range, however, the patient had a fever history. Fever is a common symptom of infection, but its absence does not rule out a significant infectious process, especially in immunocompromised individuals. Examination of the head and neck revealed significant edema, erythema, warmth, marked tenderness, and fluctuance involving the left submandibular, submental, parotid, cheek, temporal, and periorbital regions, extending to the left scalp. These findings are consistent with a severe and extensive soft tissue infection. Edema (swelling) and erythema (redness) are classic signs of inflammation. Warmth and tenderness indicate an active inflammatory process. Fluctuance, a wave-like sensation upon palpation, strongly suggests the presence of a fluid collection, likely an abscess. The involvement of multiple anatomical spaces (submandibular, submental, parotid, cheek, temporal, and periorbital) highlights the aggressive spread of the infection. Examination of the oral cavity/oropharynx revealed severe trismus (mouth opening ~2 cm), limiting evaluation. Poor oral hygiene was also noted. Severe trismus, as mentioned earlier, is a significant finding that can hinder oral examination and potentially compromise airway management. Poor oral hygiene is a predisposing factor for odontogenic infections. Examination of the eyes revealed left periorbital edema and erythema (preseptal cellulitis). Periorbital edema and erythema indicate inflammation and infection of the tissues surrounding the eye. Preseptal cellulitis, an infection of the soft tissues anterior to the orbital septum, is a serious condition that can potentially spread to the orbit itself, leading to orbital cellulitis and more severe complications. Other systems examination revealed the lungs were

clear on auscultation (consistent with normal X-ray). No signs of dehydration were noted. Clear lung sounds are important to rule out concurrent pulmonary infections. The absence of dehydration is relevant for fluid management, especially considering the patient's renal disease. An initial procedure was performed, involving needle aspiration of the fluctuant submandibular/parotid area, which yielded pus. The aspiration of pus confirms the presence of an abscess, a localized collection of purulent material. This procedure also provides material for microbiological analysis. Complete blood count (CBC) revealed a white blood cell (WBC) count of $31.65 \times 10^3/\mu\text{L}$ (marked leukocytosis) and an absolute neutrophil count of $29.47 \times 10^3/\mu\text{L}$. Leukocytosis, an elevated WBC count, is a hallmark of infection. Neutrophilia, an elevated neutrophil count, indicates the body's acute response to a bacterial infection. These findings are consistent with a severe bacterial infection. Hemoglobin was 7.50 g/dL (anemia). Anemia can be multifactorial in this setting, potentially related to chronic kidney disease, inflammation, or infection. Renal function tests (RFT) showed a BUN of 66.1 mg/dL and serum creatinine of 7.03 mg/dL (elevated). These results confirm the patient's underlying chronic kidney disease and indicate significant renal impairment. Liver function tests (LFT) showed an SGPT of 56 U/L (within normal range). While slightly elevated, this finding could be related to the systemic inflammatory response or medication effects. Blood glucose revealed a glucose random (GDS) of 243 mg/dL (hyperglycemia). Hyperglycemia confirms the patient's poorly controlled diabetes mellitus. Elevated blood glucose levels impair immune function and contribute to the severity of infections. Electrolytes showed sodium of 135 mmol/L and potassium of 4.03 mmol/L (within normal limits). Electrolyte imbalances can occur in patients with renal disease, but in this case, they were within the normal range. Microbiology (Pus Culture) revealed *Citrobacter amalonaticus* and *Proteus hauseri* (sensitivity: sensitive to Cefoperazone). The



identification of specific bacterial pathogens is crucial for guiding antibiotic therapy. *Citrobacter* and *Proteus* are Gram-negative bacteria that can cause opportunistic infections, particularly in immunocompromised individuals. A chest X-ray showed no abnormalities. This is important to rule out any concurrent chest infections or complications such as mediastinitis. A contrast-enhanced CT (Head & Neck) revealed extensive inflammatory changes and multiple rim-enhancing fluid collections (abscesses) involving multiple deep neck spaces. CT imaging is the gold standard for evaluating DNIs. Contrast enhancement helps to delineate the extent of the infection and identify abscess formation. The specific spaces involved included the masticator, sublingual, submental, parotid, parapharyngeal, and left preseptal orbital space. The involvement of these spaces demonstrates the widespread nature of the infection. Superior extension was noted to the left fronto-temporo-parietal subgaleal space. This extension to the subgaleal space is a rare but serious complication. The primary diagnosis was an extensive left deep neck abscess involving the masticator, sublingual, submental, parotid, parapharyngeal, and left preseptal orbital space. The etiology was secondary to odontogenic infection (left mandibular second molar). Complications included left preseptal orbital cellulitis/abscess and extensive left subgaleal abscess. Comorbidities identified were Type 2 Diabetes Mellitus (poorly controlled) and Chronic Kidney Disease (End-Stage Renal Disease on Hemodialysis) (Table 1).

The management of this patient with a complex and extensive deep neck infection, further complicated by significant comorbidities, required a comprehensive and multidisciplinary approach. The treatment strategy encompassed initial management in the emergency department and during admission, surgical intervention to address the source and extent of the infection, postoperative care during the inpatient stay, and subsequent discharge and outpatient follow-up. Upon presentation to the emergency department, the

patient's condition necessitated prompt and coordinated action. The initial phase of management was characterized by a multidisciplinary approach, involving consultations from various medical specialties. These consultations were crucial for formulating a holistic treatment plan that addressed both the infectious process and the patient's underlying health conditions. The specialties involved included Otorhinolaryngology-Head & Neck Surgery (ENT), Neurosurgery, Oral & Maxillofacial Surgery (OMFS), Internal Medicine (specifically Nephrology and Endocrinology), and Ophthalmology. The collaboration of these teams ensured that all aspects of the patient's complex presentation were thoroughly evaluated and managed. The ENT team focused on the primary infection in the deep neck spaces, while Neurosurgery was consulted due to the subgaleal extension. OMFS addressed the odontogenic source of the infection. Internal Medicine specialists managed the patient's diabetes mellitus and chronic kidney disease, and Ophthalmology addressed the orbital complications. In addition to the multidisciplinary consultations, supportive care was initiated immediately. This included intravenous hydration with 0.9% sodium chloride solution administered at a rate of 20 drops per minute. Intravenous fluids are essential in patients with severe infections to maintain adequate hydration, support circulatory volume, and facilitate the delivery of medications. Empiric antibiotic therapy was commenced promptly. Given the severity and potential for rapid progression of deep neck infections, broad-spectrum antibiotics are crucial to cover a wide range of potential pathogens. The initial antibiotic regimen consisted of Cefoperazone 1 gram intravenously every 12 hours and Metronidazole 500 milligrams intravenously every 8 hours. Cefoperazone is a broad-spectrum cephalosporin antibiotic effective against many Gram-positive and Gram-negative bacteria, while Metronidazole is particularly effective against anaerobic bacteria, which are frequently involved in deep neck infections. To mitigate the



inflammatory response associated with the infection, anti-inflammatory medication was administered. Methylprednisolone 62.5 milligrams was given intravenously every 12 hours. Corticosteroids like Methylprednisolone can help reduce swelling, pain, and other inflammatory manifestations. Analgesic medication was provided to manage the patient's severe pain. Ketorolac 30 milligrams was administered intravenously every 8 hours. Ketorolac is a nonsteroidal anti-inflammatory drug (NSAID) with potent analgesic properties. Gastroprotection was also included in the initial management strategy. Omeprazole 40 milligrams was given intravenously every 12 hours. Proton pump inhibitors like Omeprazole help to reduce gastric acid production, which is important in patients receiving multiple medications, especially corticosteroids, to prevent stress ulcers and other gastrointestinal complications. Given the presence of extensive abscesses and the potential for serious complications, urgent surgical intervention was deemed necessary. The surgical procedure involved a joint operation performed by the Neurosurgery, ENT, and OMFS teams under general anesthesia. General anesthesia ensured patient comfort and immobility during the complex and lengthy procedure. The surgical intervention comprised three key components. First, incision and drainage of the subgaleal abscess were performed via a scalp incision. This involved making an incision in the scalp to access the subgaleal space and evacuate the purulent collection. Second, incision and drainage of the submandibular abscess were carried out via a cervical incision. A standard external cervical incision was made in the neck to access and drain the abscess in the submandibular space. Third, odontectomy of the left mandibular second molar was performed. Removal of the infected tooth, the source of the odontogenic infection, is a critical step to prevent recurrence. During the surgical procedure, approximately 300 cubic centimeters of purulent fluid were evacuated from the subgaleal and

submandibular spaces combined. This volume of pus underscores the extent of the infectious process. Following the drainage of the abscesses, specific measures were taken to facilitate continued drainage and healing. Penrose drains were placed in the subgaleal space. Penrose drains are passive drains that allow for the egress of any residual fluid. Cutimed Sorbact packing was used for the submandibular wound. Cutimed Sorbact is an antimicrobial dressing that helps to prevent infection and promote wound healing. Pus samples were collected and sent for culture and sensitivity testing to identify the causative organisms and guide antibiotic therapy. Following the surgical intervention, the patient was transferred to an inpatient setting for intensive postoperative care. Monitoring was a crucial aspect of this phase, with intensive care monitoring conducted initially. Daily wound assessment was performed to monitor for signs of infection or complications. Clinical monitoring for signs of improvement, such as decreased swelling, pain, trismus, and drainage, was conducted regularly. Laboratory monitoring, including serial white blood cell (WBC) counts, was performed to assess the patient's response to treatment and monitor for systemic signs of infection. Antibiotic therapy was adjusted based on the results of the pus culture and sensitivity testing, as well as the patient's clinical response. The culture results identified *Citrobacter amalonaticus* and *Proteus hauseri*. Both organisms were sensitive to Cefoperazone. However, based on the patient's clinical response, specifically a decrease in the WBC count to $13.00 \times 10^3/\mu\text{L}$, the antibiotic regimen was changed to Cefepime 1 gram intravenously every 12 hours. Cefepime is a fourth-generation cephalosporin antibiotic with a broad spectrum of activity. This adjustment may have been made to provide more targeted coverage or to minimize potential side effects, considering the patient's renal function. Given the patient's significant comorbidities, careful comorbidity management was essential. Diabetes management involved intensive glycemic



control. Insulin Aspart 12 units subcutaneously with meals and Insulin Glargine 12 units subcutaneously daily were administered to achieve optimal blood glucose levels. Chronic kidney disease management included a specific CKD diet, providing 35 kilocalories per kilogram of body weight per day and 1.2 grams of protein per kilogram of body weight per day. Routine hemodialysis was performed every 4 days. Anemia, with a hemoglobin level of 7.5 grams per deciliter, was treated with a packed red blood cell (PRC) transfusion. Ophthalmologic care was provided to manage the left preseptal cellulitis. This included the use of Lyteers eye drops, Xitrol eye ointment, Levofloxacin eye drops, warm compresses, and eyelid hygiene. These measures aimed to reduce inflammation and infection in the periorbital region. Wound care was an integral part of the postoperative management. Daily wound irrigation, drain management or removal as indicated, and dressing changes were performed to promote healing and prevent further infection. The patient demonstrated gradual clinical improvement during the inpatient stay. Edema and trismus resolved on postoperative day 8. Pain and pus drainage also decreased significantly. Upon achieving clinical stability and demonstrating significant improvement, the patient was deemed suitable for discharge. Discharge medications were prescribed to ensure continued recovery and prevent recurrence of infection. These included an oral antibiotic, Levofloxacin 500 milligrams once daily, an oral corticosteroid, Methylprednisolone 8 milligrams twice daily, and an oral analgesic, Paracetamol 500 milligrams every 8 hours. Levofloxacin provided continued antibiotic coverage, Methylprednisolone helped to further reduce any residual inflammation, and Paracetamol addressed any remaining pain. Follow-up care was arranged to monitor the patient's progress and ensure complete resolution of the infection. At the first follow-up visit to the ENT clinic, the patient reported minimal residual pain. Trismus was completely resolved, and there was no further pus

or drainage from the incision sites. Examination confirmed resolution of the abscesses. Wound drainage and care were performed as needed during this follow-up visit to ensure continued healing and prevent any complications (Table 2).

3. Discussion

The primary source of the infection in this case was identified as the left second mandibular molar. Odontogenic infections are a well-established etiology for DNIs. The anatomical relationship of the mandibular molars to the deep neck spaces predisposes them to be a frequent origin of these infections. The roots of these teeth, particularly the second and third molars, often extend below the mylohyoid muscle, a key anatomical landmark. This location facilitates the direct spread of infection from the tooth apex into the submandibular space. The submandibular space is one of the most commonly involved spaces in DNIs of odontogenic origin. Once an infection is established in this space, it can readily spread to adjacent deep neck spaces. This spread occurs along the fascial planes, which are layers of connective tissue that separate and compartmentalize the structures of the neck. These planes provide pathways for the dissemination of infection, often following the path of least resistance. In this particular case, the infection extended beyond the submandibular space to involve a multitude of other deep neck spaces. These included the sublingual space, located below the tongue, the parotid space, housing the parotid gland, the masticator space, encompassing the muscles of mastication and the parapharyngeal space, situated adjacent to the pharynx. This widespread involvement underscores the aggressive nature of the infection and its ability to rapidly traverse anatomical boundaries. The sublingual space, situated in the floor of the mouth, can become involved through direct extension from the submandibular space or from infections of the anterior mandibular teeth.



Table 1. Summary of patient's clinical findings.

Category	Finding	Details
Demographics	Age	44 years
	Gender	Male
	Ethnicity/Origin	Indonesian (Bali)
	Occupation	Private sector employee
Anamnesis (History)	Chief Complaint	Rapidly progressing swelling (left face, jaw, eye) and severe pain
	History of Present Illness	Onset 2 days prior to admission. Swelling started under left mandible, enlarged significantly, became painful. Worsening swelling of left cheek and periorbital region. Fever onset ~3 days prior to admission. Difficulty opening mouth (trismus). Dysphagia (solids > liquids).
	Dental History	History of caries in the left second mandibular molar (~1 week prior). Poor oral hygiene noted.
	Past Medical History	Significant for Type 2 Diabetes Mellitus (poorly controlled) and Chronic Kidney Disease (end-stage, on maintenance hemodialysis).
	Review of Systems	Denied chest pain. No signs of dehydration reported initially.
Physical examination	General Appearance	Alert, oriented (GCS 15/15), moderate general condition.
	Vital Signs (Initial)	BP: 130/90 mmHg, HR: 80/min, RR: 20/min, Temp: 37.0°C.
	Head & Neck	Significant edema, erythema, warmth, marked tenderness, and fluctuance involving left submandibular, submental, parotid, cheek, temporal, and periorbital regions, extending to the left scalp.
	Oral Cavity/Oropharynx	Severe trismus (mouth opening ~2 cm), limiting examination. Poor oral hygiene.
	Eyes	Left periorbital edema and erythema (preseptal cellulitis).
	Other Systems	Ears, Nose: Unremarkable. Chest: Clear on auscultation (consistent with normal X-ray). No signs of dehydration.
	Initial Procedure	Needle aspiration of fluctuant submandibular/parotid area yielded pus.
Laboratory findings	Complete Blood Count (CBC)	WBC: $31.65 \times 10^3/\mu\text{L}$ (Marked Leukocytosis) Absolute Neutrophils: $29.47 \times 10^3/\mu\text{L}$ (Neutrophilia) Hemoglobin (Hb): 7.50 g/dL (Anemia)
	Renal Function Test (RFT)	BUN: 66.1 mg/dL (Elevated) Serum Creatinine: 7.03 mg/dL (Elevated)
	Liver Function Test (LFT)	SGPT: 56 U/L (Elevated) SGOT: 13 U/L (Within normal range)
	Blood Glucose	Glucose Random (GDS): 243 mg/dL (Hyperglycemia)
	Electrolytes	Sodium: 135 mmol/L Potassium: 4.03 mmol/L (Within normal limits)
	Microbiology (Pus Culture)	<i>Citrobacter amalonaticus</i> <i>Proteus hauseri</i> (Sensitivity: Sensitive to Cefoperazone/Sulbactam)
Imaging findings	Chest X-Ray	Normal; no acute cardiopulmonary abnormalities.
	Contrast-Enhanced CT (Head & Neck)	Extensive inflammatory changes and multiple rim-enhancing fluid collections (abscesses) involving: - Deep Neck Spaces: Left masticator, submandibular, sublingual, parotid, parapharyngeal. - Superior Extension: Left maxillofacial subcutaneous tissue, left preseptal orbital space, left fronto-temporo-parietal subgaleal space. Associated with diffuse edema and air foci. No thoracic inlet extension.
Clinical diagnosis	Primary Diagnosis	Extensive Left Deep Neck Abscess (Submandibular, Sublingual, Parotid, Masticator, Parapharyngeal)
	Etiology	Secondary to Odontogenic Infection (Left Mandibular Second Molar)
	Complications / Extension	Concurrent Left Preseptal Orbital Cellulitis/Abscess and Extensive Left Subgaleal Abscess
	Comorbidities	Type 2 Diabetes Mellitus (Poorly Controlled) Chronic Kidney Disease (End-Stage Renal Disease on Hemodialysis)



Table 2. Summary of patient's treatment and follow-up.

Phase	Intervention/Procedure	Details
Initial management (Emergency Department & Admission)	Consultations	Multidisciplinary: Otorhinolaryngology-Head & Neck Surgery (ENT), Neurosurgery, Oral & Maxillofacial Surgery (OMFS), Internal Medicine (Nephrology, Endocrinology), Ophthalmology
	Supportive Care	Intravenous hydration (0.9% NaCl @ 20 drops/min)
	Initial Medications	Antibiotics (Empiric): Cefoperazone 1g IV q12h, Metronidazole 500mg IV q8h Anti-inflammatory: Methylprednisolone 62.5mg IV q12h Analgesic: Ketorolac 30mg IV q8h Gastroprotection: Omeprazole 40mg IV q12h
Surgical intervention	Procedure	Urgent joint operation (Neurosurgery, ENT, OMFS) under general anesthesia: 1. Incision & Drainage of Subgaleal Abscess (via scalp incision) 2. Incision & Drainage of Submandibular Abscess (via cervical incision) 3. Odontectomy of left Mandibular Second Molar.
	Intraoperative Findings	Approx. 300 cc total purulent fluid evacuated from subgaleal and submandibular spaces.
	Post-Procedure	Penrose drains placed in subgaleal space. Cutimed Sorbact packing used for submandibular wound. Pus sent for culture & sensitivity.
Postoperative care (Inpatient)	Monitoring	Intensive care monitoring initially. Daily wound assessment. Clinical monitoring for signs of improvement (swelling, pain, trismus, drainage). Laboratory monitoring (WBC count).
	Antibiotic Adjustment	Based on culture results (<i>Citrobacter amalonaticus</i> , <i>Proteus hauseri</i>) and clinical response (WBC decreased to $13.00 \times 10^3/\mu\text{L}$), changed to Cefepime 1g IV q12h.
	Comorbidity Management	Diabetes: Intensive glycemic control (Insulin Aspart 12U SC with meals, Insulin Glargine 12U SC daily). CKD: Specific CKD diet (35 kcal/kg/d, 1.2 g protein/kg/d), routine Hemodialysis (HD) every 4 days. Anemia: Packed Red Blood Cell (PRC) transfusion (for Hb 7.5 g/dL).
	Ophthalmologic Care	Management of left preseptal cellulitis: Lyteers eye drops, Xitrol eye ointment, Levofloxacin eye drops, warm compresses, eyelid hygiene.
	Wound Care	Daily wound irrigation, drain management/removal as indicated, dressing changes.
	Clinical Progression	Gradual improvement. Edema and trismus resolved on post operation day 8). Pain and pus decreased.
Discharge	Discharge Medications	Oral Antibiotic: Levofloxacin 500mg once daily; Oral Corticosteroid: Methylprednisolone 8mg twice daily; Oral Analgesic: Paracetamol 500mg every 8 hours
Outpatient Follow-up	First Follow-up (ENT Clinic)	Patient reported minimal residual pain. Trismus completely resolved. No further pus or drainage from incision sites. Examination confirmed resolution of abscesses. Wound drainage/care performed as needed.



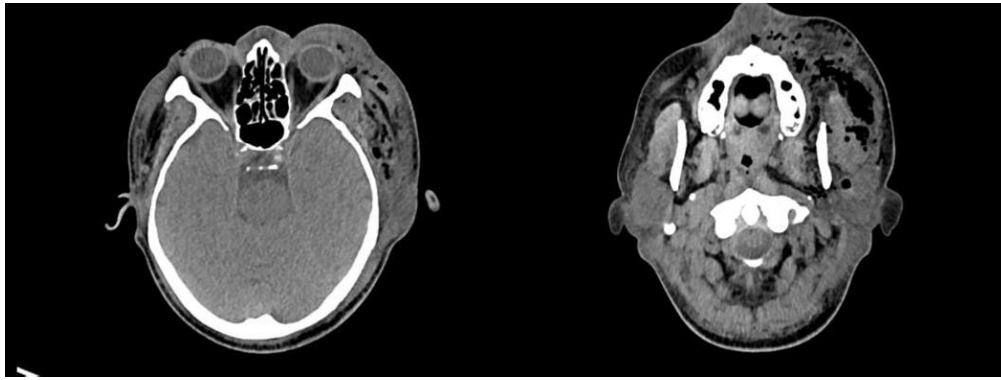


Figure 1. CT scan of the patient's head and neck.

The parotid space, while primarily associated with salivary gland infections, can also be affected by the spread of infection from adjacent spaces. The masticator space, containing the muscles responsible for chewing, is often involved in infections originating from the posterior mandibular teeth. The parapharyngeal space is of particular concern due to its proximity to vital structures, including the carotid artery, jugular vein, and cranial nerves. Infections in this space can lead to serious complications. The pattern of spread observed in this case highlights the interconnectedness of the deep neck spaces and the potential for seemingly localized infections to rapidly evolve into extensive and complex conditions. Understanding these anatomical relationships and the typical pathways of infection spread is crucial for accurate diagnosis, effective treatment planning, and the prevention of potentially life-threatening complications.^{11,12}

A particularly noteworthy aspect of this case is the unusual superior extension of the infection, involving both the preseptal orbital space and the subgaleal space. While DNIs commonly spread along the deep neck spaces, extension beyond these spaces into the orbit and subgaleal region is relatively rare. This atypical spread pattern makes this case particularly unique and clinically significant. Orbital involvement in DNIs can manifest as either preseptal cellulitis or orbital cellulitis. Preseptal cellulitis, as seen in this case, is an infection of the soft tissues anterior to the

orbital septum, the fibrous membrane that separates the eyelids and anterior structures from the deeper orbital tissues. While usually less severe than orbital cellulitis, preseptal cellulitis can progress to orbital cellulitis if not promptly and effectively treated. The mechanisms by which DNIs can extend to involve the orbit are multifactorial. Direct spread from adjacent structures, such as the sinuses, is one potential route. Sinusitis, particularly of the ethmoid or maxillary sinuses, can lead to the development of orbital cellulitis due to the close proximity of these structures to the orbit. Another possible mechanism is hematogenous spread, where bacteria from the primary infection site enter the bloodstream and disseminate to the orbit. The facial veins, which lack valves, can facilitate the retrograde spread of infection to the orbit. Additionally, infection can spread indirectly from the deep neck spaces via the pterygopalatine and infratemporal fossae. These anatomical spaces provide pathways for the transmission of infection from the masticator and parapharyngeal spaces to the orbit. Subgaleal abscess, the other unusual extension observed in this case, is a collection of pus in the potential space between the periosteum of the cranium and the galea aponeurotica. The galea aponeurotica is a tough, fibrous sheet that covers the calvarium (skullcap). Subgaleal abscesses are relatively rare and are more commonly associated with other etiologies than DNIs. The typical causes of subgaleal abscesses include

trauma to the scalp, such as lacerations or hematomas infections of the scalp, such as folliculitis or cellulitis neurosurgical procedures, which can introduce bacteria into the subgaleal space and hematogenous spread from distant sites of infection. In some cases, subgaleal abscesses can also arise from the superior spread of infections from the deep neck or as a complication of otogenic infections (ear infections). The concurrent presence of both preseptal orbital cellulitis and subgaleal abscess as a result of a DNI, as seen in this case, is an extremely rare occurrence. This unusual pattern of spread highlights the aggressive potential of these infections, particularly when host defenses are compromised, and underscores the importance of considering atypical presentations in the diagnosis and management of DNIs.^{13,14}

The patient in this case had two significant comorbidities poorly controlled type 2 diabetes mellitus and chronic kidney disease requiring maintenance hemodialysis. These comorbidities played a crucial role in the development, progression, and severity of the DNI. Both diabetes mellitus and chronic kidney disease are known to impair the immune system, predisposing individuals to infections and altering their response to them. Diabetes mellitus, characterized by hyperglycemia, has a profound impact on various aspects of the immune response. Hyperglycemia impairs neutrophil function, which is critical for the body's defense against bacterial infections. Neutrophils are a type of white blood cell that plays a key role in phagocytosis, the process by which bacteria and other pathogens are engulfed and destroyed. In individuals with diabetes, neutrophils exhibit reduced chemotaxis (the ability to migrate towards a site of infection), impaired adhesion to endothelial cells (the cells lining blood vessels), and decreased intracellular killing of bacteria. These functional deficits increase the susceptibility to infections and contribute to their severity. Furthermore, hyperglycemia can also affect other

components of the immune system, including T-cell function and cytokine production. Cytokines are signaling molecules that play a crucial role in regulating the immune response. Dysregulation of cytokine production in diabetes can lead to a prolonged and exaggerated inflammatory response, which can contribute to tissue damage and complications. Chronic kidney disease also results in a state of immunocompromise. Uremia, the accumulation of waste products in the blood due to impaired kidney function, has a toxic effect on immune cells. Patients with CKD often exhibit impaired neutrophil function, similar to that seen in diabetes. They may also have decreased lymphocyte function and impaired cell-mediated immunity. Maintenance hemodialysis, while essential for managing end-stage renal disease, can further increase the risk of infection. Hemodialysis involves the use of vascular access, such as catheters or arteriovenous fistulas, which can serve as a portal of entry for bacteria. Patients undergoing hemodialysis are also at increased risk of infection due to factors such as malnutrition, anemia, and iron overload. In the context of DNIs, the presence of diabetes mellitus and chronic kidney disease is associated with an increased risk of complications, more extensive infections, longer hospital stays, and potentially higher mortality. The altered immune response in these patients can lead to a more rapid spread of infection, a greater propensity for abscess formation, and a reduced ability to clear the infection effectively. The management of DNIs in patients with these comorbidities requires a multidisciplinary approach that addresses both the infection and the underlying medical conditions. This includes aggressive antibiotic therapy, surgical drainage of abscesses, and meticulous control of blood glucose levels and renal function.^{15,16}

The microbiological analysis of the pus aspirated from the abscess in this case revealed the presence of *Citrobacter amalonaticus* and *Proteus hauseri*. These



are Gram-negative bacteria. The identification of the causative organisms is crucial for guiding antibiotic therapy and understanding the pathogenesis of the infection. *Citrobacter* species are generally considered to be opportunistic pathogens. They are commonly found in the environment and as part of the normal gut flora. While they are often of low virulence, they can cause infections, particularly in individuals with compromised immune systems. *Citrobacter* infections are frequently associated with healthcare-associated infections, such as urinary tract infections, bloodstream infections, and wound infections. In some cases, they can also be involved in head and neck infections. *Proteus* species are also Gram-negative bacteria that are commonly found in the environment and the gastrointestinal tract. They are known for their ability to produce urease, an enzyme that breaks down urea into ammonia. This can alkalize the urine and contribute to the formation of kidney stones in urinary tract infections. *Proteus* species are also opportunistic pathogens and can cause a variety of infections, including urinary tract infections, wound infections, and pneumonia. They can also be found in DNIs, often as part of a polymicrobial infection. DNIs are typically polymicrobial infections, meaning that they involve a mixture of different types of bacteria. These can include both aerobic bacteria, which require oxygen to survive, and anaerobic bacteria, which can grow in the absence of oxygen. Common aerobic bacteria found in DNIs include *Streptococci* and *Staphylococci*, which are Gram-positive bacteria, and *Klebsiella*, which is a Gram-negative bacterium. Anaerobic bacteria commonly involved in DNIs include *Prevotella*, *Peptostreptococcus*, *Bacteroides*, and *Fusobacterium*. These anaerobic bacteria often play a significant role in the formation of abscesses. The isolation of *Citrobacter* and *Proteus* in this case may reflect the patient's immunocompromised status. These organisms are more likely to cause infections in individuals with weakened immune systems. It is also possible that the patient's prior hospitalization or

exposure to the healthcare environment contributed to the acquisition of these organisms. While these organisms can be part of the complex microbial flora of head and neck infections, their presence highlights the importance of considering the patient's overall health and potential risk factors. The polymicrobial nature of DNIs underscores the need for broad-spectrum antibiotic therapy that covers both aerobic and anaerobic bacteria. This is particularly important in the initial stages of treatment, before the results of culture and sensitivity testing are available.^{17,18}

The diagnosis of DNIs relies on a combination of clinical evaluation and imaging studies. A thorough history and physical examination are essential for identifying the signs and symptoms suggestive of a deep neck infection. These can include neck pain, swelling, redness, warmth, tenderness, trismus, and dysphagia. Fever and chills may also be present. In this case, the patient presented with a constellation of symptoms consistent with a DNI, including rapidly progressing swelling, pain, fever, and trismus. The physical examination revealed significant edema, erythema, warmth, tenderness, and fluctuance in the affected areas, further supporting the diagnosis of an abscess. While clinical evaluation is crucial, imaging studies are essential for confirming the diagnosis, determining the extent of the infection, and identifying any complications. Contrast-enhanced computed tomography (CT) is considered the gold standard imaging modality for evaluating DNIs. CT scans provide detailed anatomical information and can accurately delineate the location and extent of abscesses, as well as any involvement of adjacent structures. In this case, the CT scan revealed extensive inflammatory changes and multiple rim-enhancing fluid collections consistent with abscess formation involving multiple deep neck spaces. The CT scan also demonstrated the unusual superior extension of the infection into the preseptal orbital space and the subgaleal space. This information was critical for surgical planning and guiding the management of the



infection. Other imaging modalities, such as ultrasound and magnetic resonance imaging (MRI), may also be used in the evaluation of DNIs, but CT is generally preferred due to its superior spatial resolution and ability to visualize bony structures. The prompt and accurate diagnosis of DNIs is crucial for initiating timely and appropriate treatment, which can significantly reduce the risk of complications and improve patient outcomes.^{19,20}

4. Conclusion

This case report illustrates a rare and severe presentation of a deep neck infection (DNI) originating from an odontogenic source, specifically a carious mandibular molar, in a 44-year-old male with poorly controlled type 2 diabetes mellitus and chronic kidney disease. The infection demonstrated an unusual pattern of spread, extending beyond the typical deep neck spaces to involve the left preseptal orbital space and the subgaleal space. The patient's underlying comorbidities of diabetes mellitus and chronic kidney disease significantly contributed to the aggressive nature and the extent of the infection. These conditions are known to impair immune function, thereby increasing the susceptibility to severe infections and complicating their management. The successful management of this complex case required a multidisciplinary approach, involving prompt surgical intervention for drainage of the abscesses and removal of the infected tooth, broad-spectrum antibiotic therapy to address the identified pathogens (*Citrobacter amalonaticus* and *Proteus hauseri*), and meticulous management of the patient's diabetes and renal disease. This case underscores the importance of considering the potential for atypical and aggressive spread of odontogenic DNIs, particularly in immunocompromised patients. It highlights the critical role of prompt diagnosis with appropriate imaging, a multidisciplinary treatment strategy, and careful management of underlying comorbidities to achieve a favorable outcome in such challenging

clinical scenarios.

5. References

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