

Deep cervical cellulitis with mediastinal and retroperitoneal extension aggravated by non-steroidal anti-inflammatory drugs: A challenging case report

Habib Bellamlih *, Soufiane Belabbes, Brahim Zinoun and Taoufik Africha

Radiology Department, Moulay Ismail Military Hospital, Meknes, Sidi Mohamed Ben Abdellah University, Fez, Morocco.

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Abstract

Background: Cervicofacial and deep cervical cellulitis represent severe, life-threatening infectious emergencies of the head and neck. The inappropriate use of non-steroidal anti-inflammatory drugs (NSAIDs) for pain management is widely recognized as a major risk factor that masks symptoms, promotes rapid tissue destruction, and accelerates deep fascial, mediastinal, and retroperitoneal extension.

Case Presentation: We report the case of a 45-year-old female with no significant prior medical history who presented with an acute ankle sprain treated medically with analgesics and NSAIDs. Three days later, she developed a painful, warm, and inflammatory left laterocervical swelling with no obvious ear, nose, and throat (ENT) or dental portal of entry. Biological evaluations revealed a prominent inflammatory syndrome. Contrast-enhanced cervico-thoracic computed tomography (CT) demonstrated left laterocervical cellulitis exerting mass effect on the upper aero-digestive tract, extending inferiorly into the mediastinum and downwards into the retroperitoneum, accompanied by a small venous thrombosis involving the initial portions of the left internal jugular vein and the left subclavian vein. The patient was successfully managed with broad-spectrum intravenous antibiotic therapy and corticosteroids, resulting in significant clinical, biological, and radiological improvement.

Conclusion: Deep cervical cellulitis extending beyond the cervicothoracic junction into the mediastinum and retroperitoneum is an exceptionally rare and critical condition. This case underscores the detrimental role of NSAIDs in exacerbating deep neck infections and highlights the efficacy of prompt medical therapy and close monitoring in achieving favorable outcomes.

Keywords: Deep Cervical Cellulitis; Non-Steroidal Anti-Inflammatory Drugs (NSAIDs); Mediastinitis; Venous Thrombosis; Retroperitoneal Extension.

1. Introduction

Deep cervical cellulitis (DCC) is a severe, rapidly progressive infection involving the fascial spaces of the head and neck [1]. Despite modern antibiotic therapy and advanced intensive care management, it remains a major medical and surgical emergency associated with high morbidity and mortality rates [2, 3]. Most cases originate from odontogenic, pharyngeal, or salivary gland infections [4, 5]. However, the precipitating and aggravating role of non-steroidal anti-inflammatory drugs (NSAIDs) prescribed or self-administered for unrelated minor inflammatory conditions has been increasingly highlighted in recent literature [1, 6]. NSAIDs inhibit the cyclooxygenase pathway, thereby impairing leukocyte chemotaxis, masking early clinical signs of infection, and facilitating aggressive fascial destruction and descending spread [7, 8].

* Corresponding author: Habib Bellamlih

We report a rare and atypical case of deep left laterocervical cellulitis in a 45-year-old immunocompetent female occurring after NSAID intake for an ankle sprain, characterized by unusual downward extension into the mediastinum and retroperitoneum, complicated by localized venous thrombosis, and successfully treated with conservative medical management.

2. Case Presentation

A 45-year-old female, with no notable past medical history (specifically no diabetes, immunosuppression, or chronic renal disease), presented to the emergency department with a one-week history of an acute left ankle sprain, for which she had received symptomatic treatment consisting of analgesics and NSAIDs. Three days following the initiation of NSAID therapy, she developed an acute, painful, warm, and highly inflammatory left laterocervical swelling accompanied by progressive dysphagia and neck stiffness.

A thorough clinical examination revealed no obvious dental caries, periodontal disease, or primary otorhinolaryngological (ENT) portal of entry. Physical examination of the neck showed a tender, indurated, and warm swelling on the left side of the neck extending from the submandibular region down to the supraclavicular fossa, causing mild mass effect on the upper aero-digestive tract, though without acute upper airway obstruction or respiratory distress.

Biological workup demonstrated a significant inflammatory syndrome, showing severe hyperleukocytosis at $18,500/\mu\text{L}$ (predominantly neutrophils at $15,200/\mu\text{L}$) and a markedly elevated C-reactive protein (CRP) level of 245 mg/L. Renal and hepatic function panels were within normal limits.

Contrast-enhanced cervico-thoracic computed tomography (CT) scan revealed extensive left laterocervical cellulitis with fat stranding, tracking deeply within the fascial planes and exerting a significant mass effect on the upper aero-digestive tract. Strikingly, the CT scan showed inferior tracking of the infectious process extending past the cervicothoracic junction into the mediastinum, and further down into the retroperitoneum. Additionally, vascular opacification revealed a small venous thrombosis involving the initial portions of the left internal jugular vein and the left subclavian vein, without signs of septic pulmonary embolism (Fig 1).

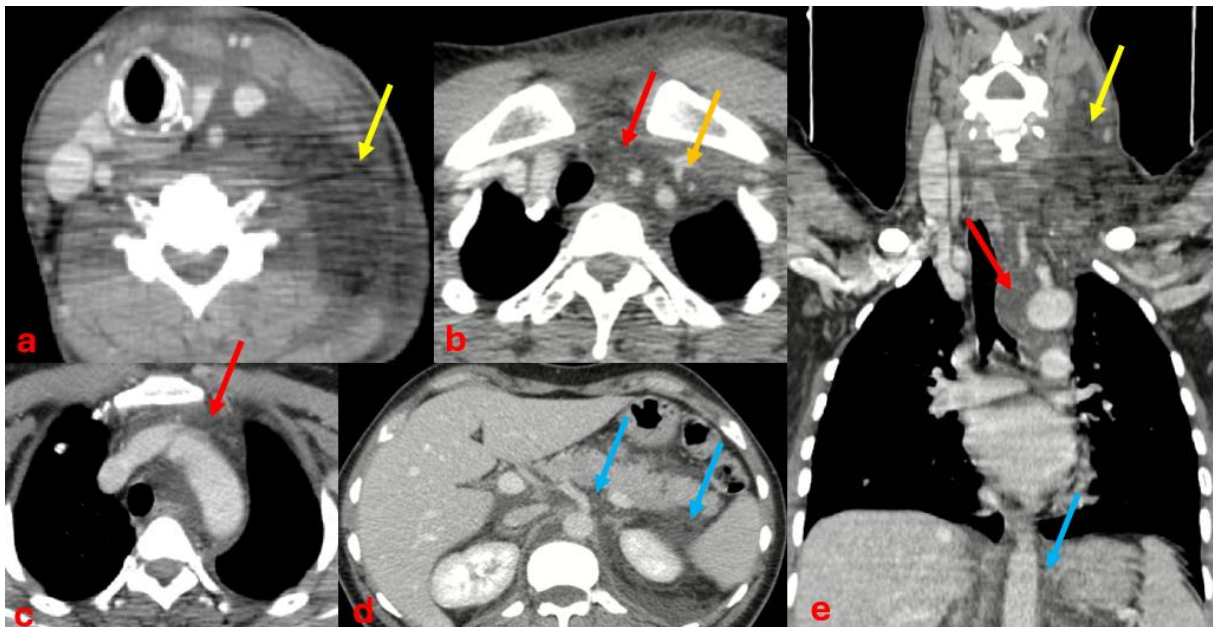


Figure 1 Axial contrast-enhanced CT images of the cervicothoracic region and upper abdomen (a–d), with coronal reconstruction (e), using intravenous iodinated contrast, demonstrate extensive left laterocervical cellulitis associated with marked inflammatory fat stranding (yellow arrows). Notably, the infectious process extends inferiorly beyond the cervicothoracic junction into the mediastinum (red arrows) and further tracks into the retroperitoneum (blue arrows). Additionally, a small venous thrombosis is identified involving the proximal left internal jugular vein and the left subclavian vein (orange arrow).

Given the absence of localized, discrete fluid collections or abscess pockets requiring immediate surgical drainage, and considering the favorable early clinical response, management was based on strict intravenous broad-spectrum antibiotic therapy (combining a third-generation cephalosporin, an aminoglycoside, and metronidazole) combined with targeted corticosteroid therapy to reduce severe inflammatory edema and mucosal compromise. Anticoagulation therapy was initiated to manage the localized venous thrombosis.

The patient showed a rapid and favorable clinical evolution over the first 72 hours. Clinical signs of inflammation and pain regressed progressively, biological inflammatory markers normalized (CRP dropping below 15 mg/L by day 10, and white blood cell count stabilizing at 7,200/ μ L), and control cervico-thoracic CT imaging, after 02 weeks, demonstrated a marked regression of the cervico-mediastinal and retroperitoneal inflammatory infiltration, as well as recanalization of the venous thrombosis (Fig 2).



Figure 2 Axial cervico thoracic and upper abdomen CT scan (a-d) with coronal reconstruction (e), using intravenous iodinated contrast, demonstrate a marked regression of the cervico-mediastinal and retroperitoneal inflammatory infiltration (yellow arrows), as well as recanalization of the venous thrombosis (blue arrow).

3. Discussion

Cervicofacial and deep cervical cellulitis are characterized by their ability to spread rapidly via the cervical fascial planes [9, 10]. The anatomy of the neck compartments provides potential conduits allowing infections to descend easily from the submandibular and parapharyngeal spaces into the mediastinum (descending necrotizing mediastinitis) [2, 11]. However, an extension reaching as far as the retroperitoneum, as observed in this patient, is exceptionally rare and highlights the severity of multi-compartmental fascial involvement [12].

A likely aggravating factor in the aggressive evolution of this patient's condition was the prior administration of NSAIDs [1]. Multiple clinical studies have established a strong correlation between NSAID use and the progression of common localized infections toward severe necrotizing or extensive cellulitis [1, 7]. Pharmacologically, NSAIDs inhibit the conversion of arachidonic acid via the cyclooxygenase pathway, suppressing key inflammatory mediators such as prostaglandins and thromboxane A₂, which are crucial for cellular recruitment, phagocytosis, and early immune defense [8]. Consequently, patients experience a false sense of pain relief while the underlying bacterial proliferation spreads unchecked beneath the fascial layers [6, 13].

Furthermore, deep neck infections frequently induce perivascular inflammation, leading to internal jugular vein thrombophlebitis or localized venous thrombosis, as demonstrated in our patient involving the initial segments of the internal jugular and subclavian veins [14]. While surgical drainage is traditionally the cornerstone of treatment for suppurative collections, selected cases with diffuse phlegmonous infiltration and no discrete abscess pockets can be

managed effectively with intensive medical therapy combining high-dose broad-spectrum intravenous antibiotics and corticosteroids, provided that airway security and clinical parameters are meticulously monitored [15, 16].

4. Conclusion

Deep cervical cellulitis with multi-compartmental extension (mediastinal and retroperitoneal) is a critical condition where early diagnosis and prompt therapeutic intervention are vital. This case highlights the severe hazard associated with the use of non-steroidal anti-inflammatory drugs in masking and accelerating deep-seated bacterial infections. Strict medical vigilance, avoidance of unnecessary NSAIDs in acute inflammatory or infectious contexts, and timely tailored medical-surgical management are essential to improve patient survival and prognosis.

Compliance with ethical standards

Disclosure of conflict of interest

No conflict of interest to be disclosed.

Statement of informed consent

Informed consent was obtained from all individual participants included in the study.

References

- [1] Bennani-Baïti, A.A., Benbouzid, A., Essakalli-Hossyni, L. (2015). Cervicofacial cellulitis: The impact of non-steroidal anti-inflammatory drugs. A study of 70 cases. *European Annals of Otorhinolaryngology, Head and Neck Diseases*, 132(6): 304–309.
- [2] Makeieff, M., Gresillon, N., Berthet, J.P., Garrel, R., Crampette, L., Marty-Ane, C., & Guerrier, B. (2004). Management of descending necrotizing mediastinitis. *The Laryngoscope*, 114(4): 772–775.
- [3] Righini, C.A., Motto, E., Ferriti, G., Boubagra, K., Soriano, E., Reyet, E. (2007). Cellulites extensive et médiastinite descendante nécrosante. *Ann Otolaryngol Chir Cervicofac*, 124(6): 292–300.
- [4] Ngoua Essininguele, M.L., Kabbaj, H., Aziz, Z., et al. (2021). Mediastinitis Complicating Odontogenic Cervicofacial Cellulitis. *Sch J Med Case Rep*, 9(4): 336–339.
- [5] El Messaoudi, L., Khairi, A., Oued Med, A., et al. (2021). Deep Cervical Cellulitis of Unusual Origin: About 2 Cases. *Saudi J Med Pharm Sci*, 7(10): 493–495.
- [6] Chaplain, A., Gouello, J.P., & Dubin, J. (1996). Cellulites cervicales nécrosantes aiguës à porte d'entrée pharyngée: rôle possible des anti-inflammatoires stéroïdiens et non stéroïdiens. A propos de 5 observations. *Revue de laryngologie, d'otologie et de rhinologie*, 117(5): 377–380.
- [7] Rimailho, A., Riou, B., Richard, C., Auzey, P. (1987). Fulminant necrotizing fasciitis and non-steroidal anti-inflammatory drugs. *J Infect Dis*, 155: 143.
- [8] Freytes, N., and Rankin, A.P. (2001). Necrotizing Fasciitis and Non Steroidal Anti-Inflammatory Drugs: A Case Series and Review of the Literature. *New Zealand Medical Journal*, 114: 3–6.
- [9] Gyébré, Y.M.C., Gouéta, A., Zaghré, N., et al. (2016). Complications of Cervicofacial Cellulitis Supported in University Hospital Yalgado Ouedraogo. *International Journal of Otolaryngology and Head & Neck Surgery*, 5: 115–120.
- [10] Benariba, F., Ammar, H., & Alouane, M. (2007). Cellulite cervico-faciale: à propos de 5 cas. *Médecine et Armées*, 35: 171–174.
- [11] Ho, M.W., Dhariwal, D.K., Chandrasekhar, J., et al. (2006). Use of interventional radiology in the management of mediastinitis of odontogenic origin. *British Journal of Oral and Maxillofacial Surgery*, 44(6): 538–542.
- [12] Gallo, S., Karligiotis, A., Lenzi, R., et al. (2012). Necrotizing Craniocervical Soft Tissue Infections: Clinical Experience and Personal Considerations. *Case Reports in Otolaryngology*, 2012: Article ID 489638.
- [13] McHenry, C.R., Piotrowski, J.J., Petrinic, D., & Malangoni, M.A. (1995). Determinants of Mortality for Necrotizing Soft-Tissue Infections. *Annals of Surgery*, 221: 558–565.

- [14] Lee, J.W., Immerman, S.B., & Morris, L.G. (2010). Techniques for Early Diagnosis and Management of Cervicofacial Necrotising Fasciitis. *The Journal of Laryngology & Otology*, 124(11): 1195–1200.
- [15] Hasegawa, J., Hidaka, H., Tateda, M., et al. (2011). An analysis of clinical risk factors of deep neck infection. *Auris Nasus Larynx*, 38: 101–107.
- [16] Maria, A., & Rajnikanth, K. (2013). Cervical necrotizing fasciitis caused by dental infection: a review and case report. *Natl J Maxillofac Surg*, 4(1): 88–91.