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RESEARCH ARTICLE

INTRAFAMILIAL CLUSTER OF MENINGOCOCCAL PURPURA FULMINANS: THREE FATAL CASES IN MOROCCO

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Abstract

Meningococcal purpura fulminans is the most severe manifestation of invasive Neisseria meningitidis, characterized by septic shock, disseminated intravascular coagulation (DIC), and extensive necrotic skin lesions. Despite aggressive modern intensive care, its rapid progression often renders treatment ineffective. We describe three consecutive cases involving previously healthy women (ages 39, 41, and 46) admitted to the Moulay Abdellah Hospital in Salé. All patients presented with acute infectious syndromes and rapidly spreading purpura. Despite immediate management—including meningeal-dose ceftriaxone, corticosteroids, and vasopressor support—the clinical course was fulminant, with mortality occurring between 30 minutes and 12 hours post-admission. Notably, two patients were sisters, suggesting intrafamilial transmission. Diagnosis was confirmed via multiplex PCR in one case,while the extreme speed of death precluded microbiological confirmation in the others. These cases underscore the devastating virulence of N. meningitidis and the critical limitations of reactive therapy. The findings emphasize that survival depends on hyper-early recognition and immediate empirical antibiotic administration. Further more, this cluster highlights the vital role of preventive strategies, specifically vaccination and urgent chemoprophylaxis for close contacts, to manage transmission risks and reduce mortality in high-risk environments.

Introduction:-

Invasive meningococcal disease (IMD) remains a major public health problem on a global scale, responsible for considerable morbidity and mortality (1). Although the incidence has decreased in high-income countries thanks to vaccination, the disease persists in limited-resource regions, notably in sub-Saharan Africa and the Maghreb, where it occurs in the form of sporadic cases or epidemic outbreaks.

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In Morocco, the estimated incidence of IMD varies between 0.1 and 3.75 per 100,000 inhabitants, with lethality rates oscillating between 13% and 31% (2,3). Among its clinical presentations, purpura fulminans represents the most dramatic form. It associates a massive systemic inflammatory response with endothelial failure and uncontrolled activation of coagulation, leading to DIC, extensive skin necrosis, and multi-organ failure (4,5). The pathophysiology of purpura fulminans is based on a massive release of endotoxins inducing a cytokine storm and a major alteration of the microcirculation. Even when the diagnosis is made early and antibiotic therapy is established without delay, the progression often remains fatal due to the rapidity of the process. We present here three cases of fatal meningococcal purpura fulminans observed in a Moroccan intensive care unit over a short period. A rare and notable fact, two of the patients were sisters, which illustrates the possibility of intrafamilial transmission, highlights the importance of clinical and epidemiological vigilance, and recalls the need to strengthen prevention and surveillance measures.

Clinical Observations:-

Case 1:-

A 39-year-old woman, with no notable medical history, presented with intense headaches, vomiting, and fatigue three days prior to her admission. On June 15, 2025, she was admitted to the emergency department of Moulay Abdellah Hospital in Salé due to persistent fever and worsening fatigue. At admission, the patient was confused (GCS 14/15), tachycardic at 120 beats per minute, with a blood pressure of 110/60 mmHg. Her extremities were cold, with a capillary refill time greater than 3 seconds. Skin examination showed purpuric spots of necrotic appearance on the trunk and upper and lower limbs, without meningeal rigidity or photophobia. The initial SOFA score was 9. The laboratory workup revealed severe metabolic acidosis, a very high procalcitonin level (100 ng/mL), acute kidney injury stage KDIGO 3, thrombocytopenia at 50,000/µL, anemia at 10 g/dL, a decreased PT at 15%, and hepatic cytolysis (ASAT and ALAT 4x normal).

Antibiotic therapy with meningeal-dose ceftriaxone (4 g/day) and dexamethasone (0.15 mg/kg every 6 h) was initiated as an emergency, accompanied by volume expansion with isotonic saline. Rapidly, the patient developed hemodynamic instability, requiring the administration of norepinephrine in continuous infusion. The extension of the purpura and the worsening of hypoperfusion, with marked cyanosis of the extremities, led to intubation for hemodynamic and neurological criteria before her admission to the ICU. In the ICU, fluid resuscitation and antibiotic therapy were continued. A lumbar puncture was performed, yielding clear fluid, as well as blood cultures. The progression was rapidly unfavorable with refractory shock, an elevation of lactates to 7 mmol/L, multi-organ failure, and the death of the patient eight hours after her admission to the hospital. Post-mortem, the lumbar puncture and blood cultures were sterile. Multiplex PCR was not available.

Case 2:-

A 41-year-old woman, with no particular pathological history, was admitted on August 24, 2025, to the emergency department of Moulay Abdellah Hospital in Salé for a febrile syndrome evolving for 36 hours, associated with chills, vomiting, and the progressive appearance of necrotic purpura on the face, neck, and anterior surface of the thorax. At admission, the patient was confused (GCS 13/15), tachycardic at 110 beats/min, hypotensive at 60/30 mmHg, with cold extremities and a capillary refill time > 3 seconds. Skin examination showed a confluent petechial purpura with areas of early necrosis, involving the lower limbs and the trunk.

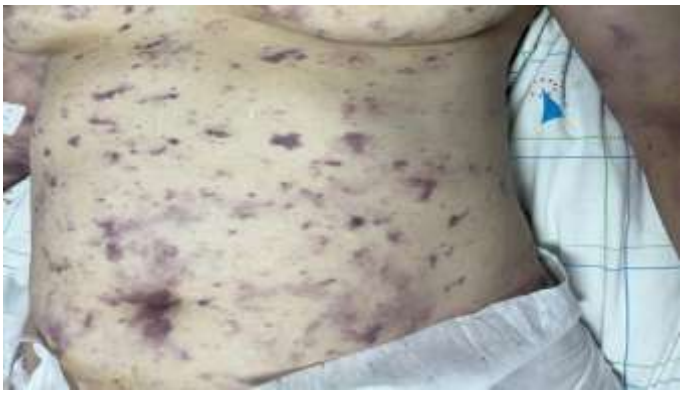

Figure 1 Purpura on the trunk of the patient in case 2

The initial biological workup revealed severe metabolic acidosis with a lactate level of 8 mmol/L, thrombocytopenia at 61,000/µL, anemia at 9 g/dL, a collapsed PT at 20%, and anuric acute kidney injury. The SOFA score at admission was 11.

Antibiotic therapy with meningeal-dose ceftriaxone (4 g/day IV) and dexamethasone (0.15 mg/kg every 6 hours) was initiated immediately, associated with initial fluid resuscitation of 30 mL/kg of isotonic saline and vasopressor support with titrated norepinephrine to maintain a mean arterial pressure ≥ 65 mmHg. A lumbar puncture was performed, yielding clear fluid, without cytobacteriological or biochemical abnormalities. Blood cultures were also taken. The progression was rapidly unfavorable, with worsening of septic shock, multi-organ failure, and irreversible cardiopulmonary arrest six hours after admission to the ICU, despite optimal management. Multiplex PCR of the cerebrospinal fluid confirmed the presence of Neisseria meningitidis, the type not being specified. Blood cultures remained sterile.

Case 3:-

A 46-year-old woman, the sister of the patient in Case 2, presented with headaches, vomiting, and a fever of 39°C on the day of her sister's death. She presented to the emergency department the following day. At admission, she already had generalized necrotic purpura, hemodynamic instability, and frank meningeal syndrome. An antibiotic treatment with ceftriaxone (4 g), fluid resuscitation, corticosteroids (dexamethasone 0.15 mg/kg), and vasopressor support with norepinephrine were initiated without delay. The patient was also intubated for hemodynamic and neurological criteria. Despite this management, she rapidly progressed toward refractory shock and died 30 minutes after her admission. No bacteriological or blood biological examination could be performed. However, the temporal and familial proximity to the previous case strongly suggests intrafamilial transmission of Neisseria meningitidis. Chemoprophylaxis was administered to all close contacts, and no secondary cases were reported.

Table 1: Clinical and biological characteristics of the three patients with Neisseria meningitidis purpura fulminans

PARAMETRE	CAS 1	CAS 2	CAS 3
AGE (YEARS)	39	41	46
FAMILIAL LINK	–	Sister of case 3	Sisterof case 2
TIME FROM SYMPTOM ONSET TO ADMISSION.	72 h	36 h	24 h
GCS AT ADMISSION	14	13	10
INITIAL BP (MMHG	110/60	60/30	70/40
SOFA SCORE	9	11	12
THROMBOCYTOPENIA (/µL)	50 000	61 000	ND
PT (%)	15	20	ND
LACTATES (MMOL/L)	7	6.8	ND
MULTIPLEX PCR	Unavailable	+ N. meningitidis	Unavailable
DEATH POST-ADMISSION	8 h	6 h	0.5 h

Public Health Notification:-

For each of the three cases, the provincial health delegation was immediately notified, in accordance with current regulations relative to mandatory reportable diseases. Respiratory isolation measures were established upon the patients' admission, and an epidemiological investigation was conducted to identify contacts at risk. Preventive chemoprophylaxis with rifampicin was administered to all family and professional contacts, accompanied by a awareness campaign and meningococcal vaccination in the close entourage. No secondary infections were reported after the implementation of these measures.

Discussion:-

Purpura fulminans (PF) constitutes the most severe and lethal clinical expression of invasive Neisseria meningitidis infection. Its pathophysiology is based on a massive release of bacterial endotoxins inducing an uncontrolled systemic inflammatory response, responsible for diffuse endothelial damage and exacerbated activation of coagulation (6,7). This cascade leads to disseminated intravascular coagulation (DIC), extensive microvascular thromboses, rapidly progressive skin necrosis, and, ultimately, severe multiple organ failure (8,9). The three reported observations strikingly illustrate the fulminant kinetics of meningococcal sepsis. Despite relatively short consultation delays for two patients (24 to 36 hours) and management consistent with international recommendations—including immediate empiric antibiotic therapy with ceftriaxone at meningitis dose, adjuvant corticosteroid therapy, and early vasopressor support—the outcome was rapidly fatal. These data confirm that, once the phase of refractory septic shock and multiple organ failure is established, conventional resuscitation therapies may prove insufficient to reverse the course of the disease (10).

The major difficulty of PF lies in its early diagnosis. Initial signs are often non-specific—fever, headaches, vomiting, deterioration of general condition—and may delay the recognition of the picture before the appearance of necrotic purpura, which nevertheless constitutes a major warning sign. In this context, clinical vigilance remains essential, and the slightest suspicion of meningococcemia must lead to the immediate initiation of empiric antibiotic therapy, without waiting for microbiological confirmation. Adjuvant therapeutic strategies aiming to modulate the inflammatory response or coagulation, such as activated protein C or recombinant thrombomodulin, have been evaluated in severe sepsis. However, they have not demonstrated a consistent benefit in terms of mortality and remain of limited access, particularly in resource-restricted countries (11,12). This reality highlights the current limits of the therapeutic arsenal against fulminant forms of the disease.

Beyond individual management, these observations shed light on the paramount importance of public health measures. In Morocco, the prevention of invasive meningococcal infections continues to face several challenges, notably heterogeneous vaccination coverage and the emergence of new serogroups, particularly W and Y, adding to the historically prevalent serogroups B and C (13). The introduction and reinforcement of tetravalent conjugate vaccines (ACWY), as well as the vaccine against serogroup B, constitute essential levers to reduce the incidence of severe forms. International recommendations, including catch-up vaccination for adolescents, aim to mitigate the waning of immunity over time (14,15). Nevertheless, vaccine adherence remains conditioned by socio-economic and cultural factors and access to care (16).

The particularly notable aspect of this series lies in the occurrence of an intrafamilial cluster, involving two sisters who died a few hours apart. This situation illustrates the high contagiousness of N. meningitidis and the high risk of transmission within the close circle. In such contexts, immediate chemoprophylaxis of contacts with rifampicin, associated with targeted vaccination measures, remains the only effective means to break the chain of transmission and prevent potentially fatal secondary cases (17). The absence of new cases after the implementation of these measures in our series highlights their effectiveness. This series nevertheless presents certain limits, notably the small number of cases and the absence of complete microbiological confirmation for two patients, linked to the extreme fulminance of the clinical picture, which does not allow for the generalization of results but detracts nothing from the descriptive value and the public health message of these observations.

Conclusion:-

These three fatal cases of meningococcal purpura fulminans in previously healthy women illustrate the rapidity of progression and the extreme severity of this infection. Despite rapid management consistent with recommendations, all patients progressed to a state of refractory shock and death, highlighting the limits of available therapeutic interventions once the fulminant phase has begun. The early recognition of warning signs and the immediate administration of appropriate antibiotic therapy remain crucial. Preventive measures, particularly vaccination and chemoprophylaxis of contacts, play a decisive role in limiting transmission and reducing mortality. These observations underscore the necessity to sensitize clinicians, reinforce healthcare infrastructures, and ensure active epidemiological surveillance. In the Moroccan context, where vaccination coverage is partial and access to care is unequal, these measures remain essential to prevent fulminant forms of meningococcal disease and improve the prognosis.

References:-

1. Harrison LH, Trotter CL, Ramsay ME. Global epidemiology of meningococcal disease. Vaccine. 2009;27(Suppl 2):B51–B63.
2. World Health Organization. Meningococcal disease in the African meningitis belt: situation report. Geneva: WHO; 2018.
3. Thompson MJ, Ninis N, Perera R, et al. Clinical recognition of meningococcal disease in children and adolescents. Lancet. 2006;367:397–403.
4. Spina S, Icardi G, Mandell GL, et al. Fulminant meningococcal sepsis: pathophysiology and clinical management. Curr Opin Infect Dis. 2018;31:306–314.
5. Stephens DS. Biology and pathogenesis of Neisseria meningitidis. Curr Opin Infect Dis. 2009;22:301–306.
6. van Deuren M, Brandtzaeg P, van der Meer JW. Update on meningococcal disease with emphasis on pathogenesis and clinical management. Clin Microbiol Rev. 2000;13:144–166.
7. Levi M, ten Cate H. Disseminated intravascular coagulation. N Engl J Med. 1999;341:586–592.
8. Esmon CT. The protein C pathway. Chest. 2003;124:26S–32S.
9. Read RC, et al. Genetic susceptibility to meningococcal disease: the role of complement and innate immunity. Lancet Infect Dis. 2009;9:522–530.
10. MacCormick AD, et al. Early ICU intervention in fulminant meningococcal sepsis. Crit Care Med. 2013;41:1760–1766.
11. Boutsikou T, et al. Toll-like receptor polymorphisms and susceptibility to severe meningococcal infection. J Infect Dis. 2010;202:243–251.
12. Van Deuren M, et al. Meningococcal sepsis: clinical and therapeutic aspects. Crit Care. 2010;14:236.
13. Bernard GR, et al. Activated protein C for severe sepsis. N Engl J Med. 2001;344:699–709.
14. Haute Autorité de Santé. Recrudescence des infections invasives à méningocoques : de nouvelles recommandations sur le rattrapage vaccinal. HAS; 2025.
15. Griskaitis M, Thielemann I, Schönfeld V, Falman A, Scholz S, Reinacher U, Haas L, Wichmann O, Harder T. Effectiveness and duration of protection of primary and booster immunisation against meningococcal serogroup C disease with meningococcal conjugate C and ACWY vaccines: Systematic review. J Infect. 2024 Sep;89(3):106228. doi: 10.1016/j.jinf.2024.106228. Epub 2024 Jul 10. PMID: 38996818.
16. Gusmão JD, da Silva TPR, Velasquez-Melendez G, et al. Association between contextual factors and coverage of the ACWY meningococcal vaccine after overdue inclusion in adolescent vaccination schedules: global space regressions. BMC Infect Dis. 2023;23:615.
17. Levi M, et al. Recombinant thrombomodulin in sepsis-induced DIC. J Thromb Haemost. 2018;16:150–161.