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Fulminant Meningococcemia Presenting with Bilateral Adrenal Hemorrhage: A Case of Acute Endocrine Collapse.

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Abstract

Background: Waterhouse–Friderichsen syndrome (WFS) is a rare, life-threatening endocrine emergency characterized by bilateral adrenal hemorrhage, acute adrenal insufficiency, septic shock, and disseminated intravascular coagulation (DIC), most commonly caused by Neisseria meningitidis. Early recognition and prompt treatment are essential to reduce mortality.

Case Presentation: A 21-year-old male presented with a 24-hour history of high-grade fever, vomiting, generalized weakness, altered sensorium, hypotension, and rapidly progressive purpuric rash. Laboratory evaluation demonstrated leukocytosis, severe thrombocytopenia, hyponatremia, hyperkalemia, hypoglycemia, prolonged PT/INR and aPTT, and markedly reduced serum cortisol, consistent with acute adrenal insufficiency and DIC. The patient was diagnosed with fulminant meningococcemia complicated by Waterhouse–Friderichsen syndrome. Management included aggressive intravenous fluid resuscitation, ceftriaxone, stress-dose intravenous hydrocortisone, vasopressor support, correction of hypoglycemia, and blood component therapy for DIC. The patient required mechanical ventilation for 48 hours and subsequently showed gradual clinical improvement.

Conclusion: This case highlights the importance of maintaining a high index of suspicion for Waterhouse–Friderichsen syndrome in patients presenting with septic shock, purpuric rash, and biochemical evidence of adrenal insufficiency. Early diagnosis, prompt initiation of broad-spectrum antibiotics, corticosteroid replacement, and intensive supportive care are crucial to improve survival and reduce the high mortality associated with this condition.

Keywords: Waterhouse–Friderichsen syndrome; Fulminant meningococcemia; Bilateral adrenal hemorrhage; Acute adrenal insufficiency; Septic shock; Disseminated intravascular coagulation (DIC); Neisseria meningitidis; Purpuric rash; Hydrocortisone; Ceftriaxone.

1. Introduction: Waterhouse–Friderichsen Syndrome (WFS)

Acute adrenal gland failure brought on by bilateral adrenal hemorrhage is the hallmark of Waterhouse-Friderichsen Syndrome (WFS), an uncommon and quickly lethal medical emergency that typically arises in the setting of overwhelming bacterial sepsis. Although other bacterial infections may potentially be present, Neisseria meningitidis, the bacterium that causes meningococcal disease, is traditionally linked to severe infection in this condition.WFS occurs when a severe systemic inflammatory response brought on by a fulminant infection results in disseminated intravascular coagulation (DIC), septic shock, and significant bleeding into both adrenal glands. The abrupt loss of adrenal function causes severe hypotension, circulatory collapse, and multi-organ failure because the adrenal glands produce vital chemicals like cortisol and aldosterone, which are necessary for regulating blood pressure, fluid balance, and stress response.

The condition is named for its creators, Carl Friderichsen and Rupert Waterhouse, who originally described it in the early 20th century. Because of its high fatality rate and quick progression, WFS is still a crucial diagnosis in emergency and critical care medicine, despite its rarity. To increase survival rates, early detection and prompt administration of supportive care, corticosteroids, and antibiotics are crucial.

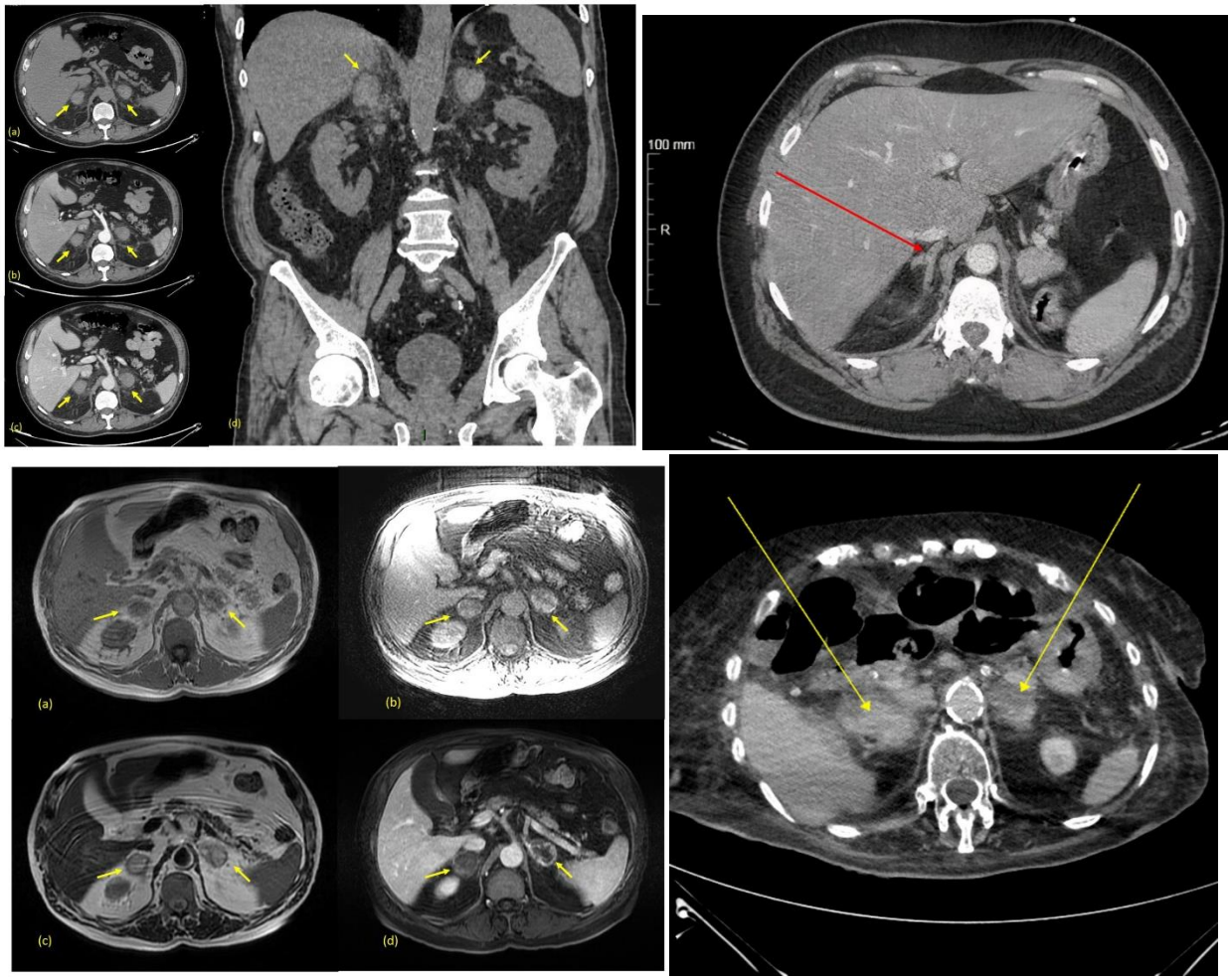
Case Presentation:

A 21-year-old man with a 24-hour history of high-grade fever, frequent vomiting, and widespread weakness was brought to the emergency room without any known comorbidities. He had dizziness, altered sensorium, and a quickly growing purpuric rash throughout his trunk and lower limbs six hours before being admitted. Upon assessment, the patient looked confused and poisonous. Temperature was 39.8°C, blood pressure was 78/46 mmHg, pulse rate was 128 beats per minute, respiration rate was 28 breaths per minute, and oxygen saturation was 90% on room air, according to vital signs. On the belly and limbs, non-blanching purpuric lesions were observed. There was a longer capillary refill time (>3 seconds). There were no indications of meningeal inflammation. Considering the severe decline accompanied by purpura and septic shock, a tentative diagnosis of fulminant meningococcemia exacerbated by disseminated intravascular coagulation (DIC).

Table: Laboratory Investigations

Parameter	Patient Value	Reference Range	Interpretation
Hemoglobin	11.2 g/dl	13-17 g/dl	Mild anemia
Platelet count	52,000/mm^3	150,000-400,000/mm^3	Severe thrombocytopenia
Total Leucocyte count	18,500/mm^3	4000-11000/mm^3	Leukocytosis
Serum sodium	126mEq/L	135-145 mEq/L	Hyponatremia
Serum potassium	5.9 mEq/L	3.5-5.0 mEq/L	Hyperkalemia
Random blood glucose	48 mg/dl	70-140 mg/dl	Hypoglycemia
Serum cortisol	Marked low	___	Adrenal insufficiencies
PT/INR	Prolonged	___	coagulopathy

aPTT	Prolonged	___	Coagulation pathway defect



Treatment Advised:

The patient was taken right away to the intensive care unit, where they were treated using: Fluid Resuscitation with Aggression boluses of isotonic saline given intravenously to replenish blood volume. Ceftriaxone 2 g IV twice daily is an example of an empirical intravenous antibiotic (subsequently continued dependent on culture sensitivity). Stress-Dose Corticosteroids: To treat acute adrenal insufficiency, inject 100 mg of hydrocortisone intravenously every eight hours. Vasopressor Support: In order to keep mean arterial pressure at or above 65 mmHg, noradrenaline is infused. Hypoglycemia correction using intravenous infusion of dextrose. Handling DIC Transfusions of platelets and fresh frozen plasma when needed. Hemodynamic parameters, urine output, blood glucose, and electrolytes were all regularly observed. Despite needing artificial ventilation for 48 hours, the patient's condition gradually improved.

2. Discussion:

A rare but deadly side effect of severe bacterial septicemia, Waterhouse-Friderichsen Syndrome is almost always linked to meningococcal infection. The pathophysiology includes broad microvascular thrombosis, endothelial damage, activation of coagulation falls, and systemic inflammation brought on by endotoxins. Abrupt adrenal failure is the result of these events, which culminate in bilateral adrenal hemorrhage.

Clinically, hypoglycemia, purpuric rash, electrolyte imbalances, and septic shock should all raise suspicions of WFS. Laboratory validation of DIC and low serum cortisol further supports the opinion. Adrenal bleeding can be confirmed by imaging tests, however treatment shouldn't be postponed until radiological confirmation is obtained.

In order to stabilize hemodynamics and reverse adrenal insufficiency, early corticosteroid treatment is essential. Direct administration of broad-spectrum antibiotics is required when meningococcemia is suspected. Mortality is still high even with aggressive operation, especially when intervention is delayed. This instance emphasizes the value of quick decisions and interdisciplinary teamwork, including prompt pharmaceutical treatment to alleviate survival concern.

Conclusion**:**
Traditionally linked to Meningococcal illness produced by Neisseria meningitidis, Waterhouse-Friderichsen Syndrome (WFS) is a severe and potentially fatal consequence of excessive septicemia. Supported by biochemical and coagulation abnormalities, the current case exhibits the traditional triad of acute adrenal insufficiency, purpuric rash, and septic shock.

A severe systemic inflammatory response is shown in the noticeably raised total leukocyte count. Prolonged PT/INR, prolonged aPTT, and severe thrombocytopenia all point to the activation of the coagulation cascade with clotting factor consumption, which is consistent with disseminated intravascular coagulation (DIC). Endotoxin-induced endothelial damage in meningococcemia causes cytokine release, extensive microvascular thrombosis, and consequent bleeding tendencies. Hemorrhagic infarction, thrombosis of the adrenal veins, and vascular congestion are thought to be the causes of bilateral adrenal bleeding.

Acute adrenal insufficiency brought on by a mineralocorticoid shortage is characterized by electrolyte abnormalities, specifically hypoonatremia and hyperkalemia. Cortisol shortage, which affects gluconeogenesis and the physiological stress response, is reflected in concurrent hypoglycemia. This patient's significantly low serum cortisol level offers biochemical evidence of adrenal insufficiency. Circulatory collapse and refractory hypotension are further exacerbated
The diagnosis is further supported by radiological evidence of bilateral adrenal hypertrophy with bleeding. WFS is still essentially a clinical diagnosis, though, thus waiting for imaging confirmation shouldn't postpone therapy. To restore vascular reactivity to catecholamines, stress-dose corticosteroids (intravenous hydrocortisone) must be administered as soon as possible. When meningococcemia is detected, it is imperative to start broad-spectrum antibiotics such ceftriaxone as soon as possible because waiting is directly linked to a higher death rate.

ETHICS APPROVAL AND CONSENT TO PARTICIPATE
The santhiram medical college and general hospital provided IEC approval.

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