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Case Report

Fulminant Meningococcemia

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Introduction

Meningococcemia is a life threatening medical emergency that can progress quickly to disseminated intravascular coagulation and multi-organ failure, Meningitis may or may not co-exit. Usually presents with apurpuric rash especially notable on extremities, if adrenal hemorrhage occurs with the rash, the process is called Waterhouse-Frederickson syndrome ***(Wu, Chen et al. 2020).***

Neisseria meningitidis is an aerobic gram-negative coccus that appears typically in pairs (diplococci) with the adjacent sides flattened. The organism is enclosed by a cell envelope containing outer membrane proteins and lipopolysaccharide (endotoxin) and by a polysaccharide capsule. Thirteen serogroups have been identified based on the antigenic structure of the capsular polysaccharide. Six of these serogroups are responsible for most human disease: serogroups A, B, C, Y, W-135, and X ***(Rosenstein, Perkins et al. 2001)***.

Disseminated intravascular coagulation (DIC) is of major pathogenic significance in cases of fulminant meningococcemia rapidly leading to death. This syndrome can be detected clinically with a simple and available battery of coagulation tests, prothrombin time, and activated partial thromboplastin time and the platelet count, These tests are sufficiently sensitive in detection of the earliest phases of DIC and providing a rational means for selecting patients requiring anticoagulant therapy ***(Winkelstein, Songster et al. 1969)***.

Purpura fulminans is a cutaneous manifestation of disseminated intravascular coagulation, begins with dermal microvessels thrombosis that rapidly leads to hemorrhagic skin necrosis, and gangrene that often necessitates amputation ***(Agarwal and Sharma 2010)***.

Sporadic disease occurs more commonly during winter and early spring months and children are predominantly affected. The highest rate of infection is in infants 6 months to 1 year, with a decline in infection rate with age. This explained by passive maternal immunity providing protection in the first 6 months and gradual onset of acquired immunity with age ***(Rivard, David et al. 1995)***.

Neisseria meningitidis is the leading cause of infectious Purpura fulminans, followed by infection with other capsulated bacteria such as Haemophilus influenza and Streptococcus pneumoniae ***(Lécuyer, Borgel et al. 2017)***.

Case Presentation

We report here a case study of Fulminant meningococcemia caused by Neisseria meningitides at Gurayat General Hospital.

A previously healthy 1 year old girl, presented at emergency department with a history of high grade fever (40°c), left Ear discharge and cough, 2 days prior to admission , At the day of admission in the morning pt. had continuous fever ,confusion and hypoactivity, but no rash, no seizure. 3 hours later purpuric rash started to appear, covering the face, trunk and extremities with black discoloration of fingers and toes of all limbs, (figure1), Patient isolated in pediatric intensive care unit, physical examination revealed stiff neck, purpuric rash covering all the body with black discoloration of fingers and toes of all limbs, Vital sign: RR: 60, HR: 192 Temp: 40c, B.P:96/54. O2 sat 98% at NC 2lL/minute, Rbs. 45 mg/ dl, Patient diagnosed clinically as Meningococcemia, lumbar puncture done later because family initially refused, so treatment started promptly with ceftriaxone and vancomycin after initial investigation and septic work up.

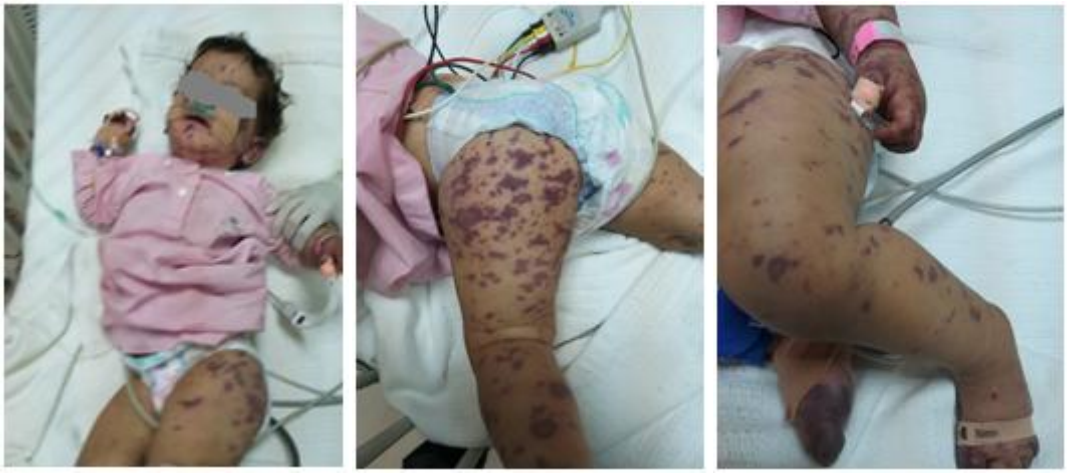

Figure (1): Purpuric rash at admission with black discoloration of fingers and toes of all limbs

Patient developed disseminated intravascular coagulation (DIC) and unfavorable prognostic factors were established (leukopenia WBC $2.5*10^3$/UL thrombocytopenia platelet count <20 000/µl, and plasma fibrinogen concentration <100 mg/dl prolonged PT, PTT, hypoglycemia and metabolic acidosis).

On the second day necrotic changes noticed in the face, left ear ,both hands and fingers, dorsal aspect of both feet, involving all toes and more intense in the first and second toes of the feet (figure 2). Fluid resuscitation, albumin, fresh frozen plasma, platelet transfusion, packed red blood cells, hydrocortisone, vitamin k, and IVIG were given.

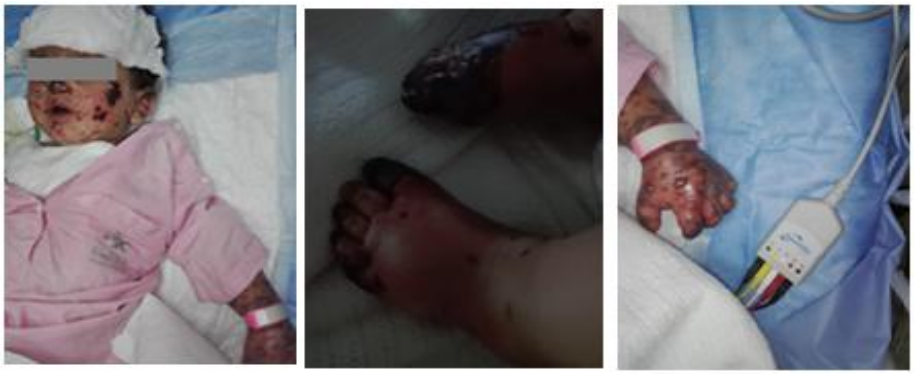

Figure (2): Showed Development of gangrenous discoloration with dry gangrene tip toes.

On day 3 Patient Stabilized after initial management, level of consciousness recovered, Hg, platelet and PT PTT INR back to normal level, no more metabolic acidosis, but the necrotic tissue of the upper and lower limps got worse, despite normal values of coagulation profile, and preserved pulses in both feet.

Blood culture: showed Neisseria meningitides, lumbar puncture showed WBCS: 300/ul, 40% neutrophils, 59% lymphocyte a protein level of 873 mg/dl, a glucose level of 4.04 mg/dl and no growth.

After 12 days of admission patient developed high grade fever with generalized tonic colonic convulsion septic work up showed E.coli-ESPL from the central femoral line, despite no growth from prick samples, CSF culture repeated -no growth, urine culture-no growth. CT-Scan of brain done no abnormality were detected, abdominal computed tomography (CT) done to rule out adrenal hemorrhage.

Line of separation started to appear on the feet for gangrenous toes with sloughed off the gangrenous tissue at different parts of the body (figure 3).

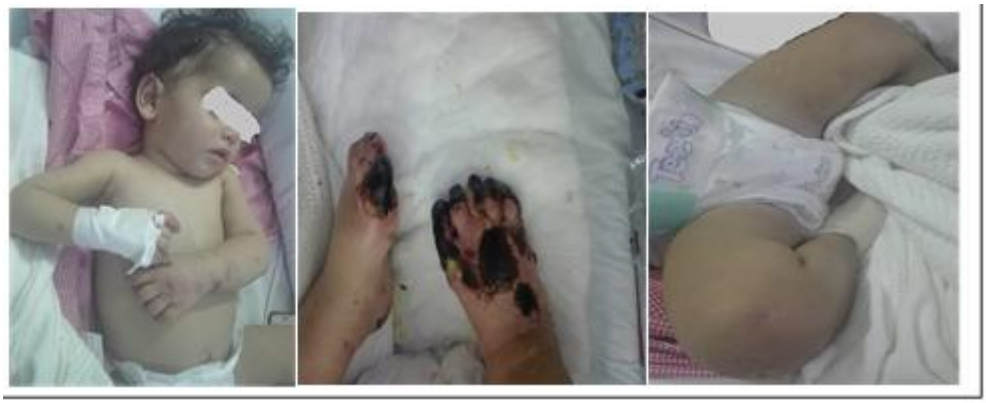

Figure (3): Falling off of the gangrenous tissue at different parts of the body

Summary of the patient's investigation during the hospital stay; complete blood count which showed initial leukopenia, thrombocytopenia and frequent drop of Hg level (table1) and (figure 4), coagulation profile which was prolonged then back to normal (table 2) and (figure 5), blood cultures which showed Neisseria meningitides then no growth after 7days, then become + ve for E.coli-ESPL from the central femoral line in (table 3), CSF results; initial CSF showed, elevated WBC count, elevated CSF protein and hypoglycemia (table 4), urine cultures (table 5) and biochemistry (table 6).

Table (1): CBC finding

Sample	Hg (g/dL)	HCT (%)	WBCs ($*10^3$/UL)	Platelet ($*10^9$/L)	Neutrophil (%)	Neutrophils absolute ($*10^3$/UL)	Lymphocyte (%)
1	9.6	32	2.58	106	2.58	0.66	71.3
2	8.5	27.1	13.5	30	31.2	4.2	46.8
3	6.2	20	8.5	17	64.1	6.1	29.5
4	7	19.9	15.98	13	75.6	12.8	20.5
5	10.2	29	24.5	11	81.6	20.08	15.3
6	10.9	29	28	20	78.2	23.9	16.7
7	10.8	29	35.7	49	68.2	24.3	25.6
8	9.5	28	37.9	108	79.1	26.9	22.9
9	9	28.6	32.4	162	43.5	14.2	50
10	8.3	26	35	445	45.5	25.5	41.2
11	7.7	25	45.3	561	62.3	28.2	33.4
12	8.4	27	32.7	631	67	21.5	26.8
13	11.3	35	25.9	764	68.6	17.7	24.2
14	11.8	38	25.1	951	64.8	16.2	30.7

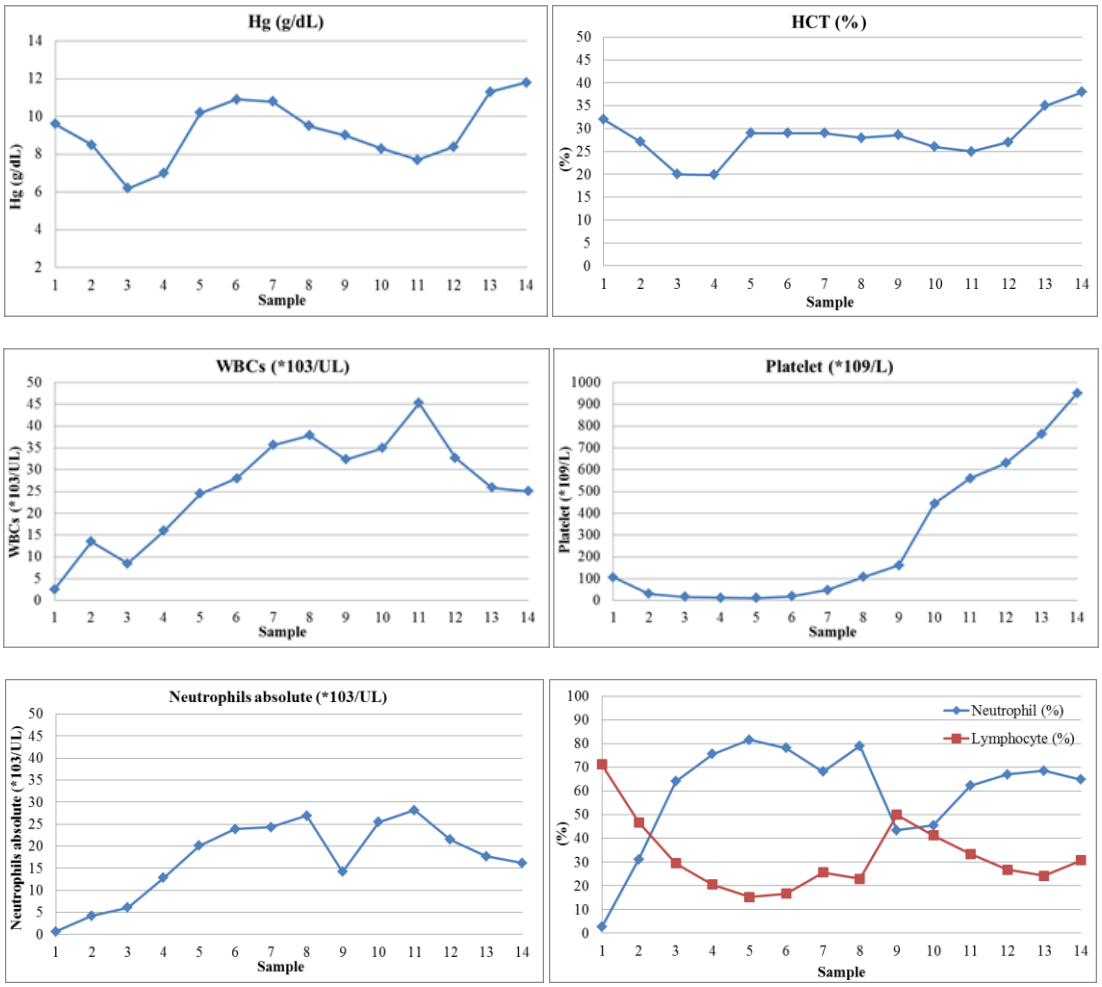

Figure (4): CBC finding

Table (2): Coagulation Profile

Sample	PT (SEC)	PTT (SEC)	INR (SEC)
1	20.4	41.5	1.6
2	17.2	92.4	1.35
3	16.2	59.8	1.26
4	15	30.9	1.14
5	15.6	38	1.3
6	11.5	35.1	0.96

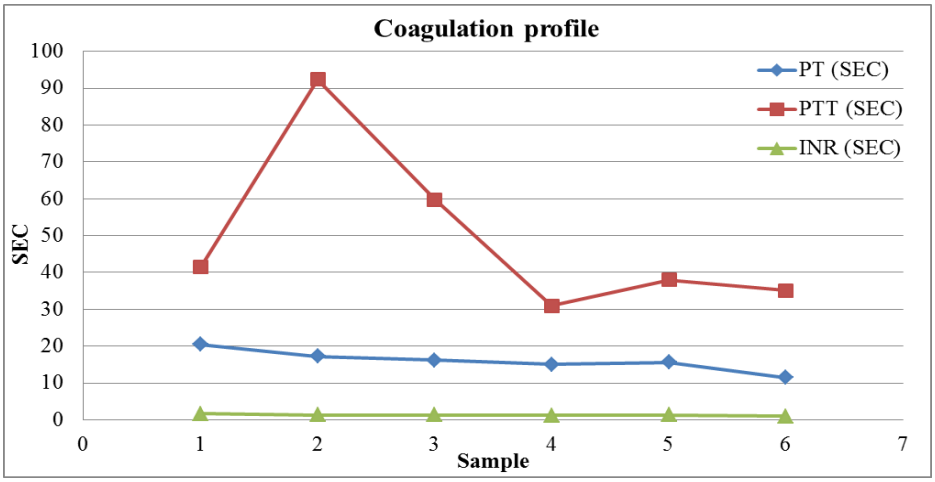

Figure (5): Coagulation profile

Table (3): Blood Culture

Sample	Blood culture
1	Neisseria meningitidis
2	No growth
3	No growth
4	E.coli (From central line)
5	No growth

Table (4): Csf result

Sample	Csf profile	Csf chemistry		Csf hematology			
	Csf culture	Csf glucose (mg/dl)	Protein (mg/dl)	WBCS (/ul)	RBCS	Neutrophil (%)	Lymphocyte (%)
1	No growth	4.04	873	300	0	40	59
2	No growth	3.6	362	29	0	34	66

Table (5): Urine culture

Sample	Urine culture
1	Candida
2	No growth
3	No growth
4	No growth

Table (6): Biochemistry results

Sample	ALP (U/L)	AST (U/L)	ALT (U/L)	Bilirubin (umol/L)	Direct bilirubin (umol/L)	Glucose random (mmol/L)	Serum Urea (mmol/L)	Serum creatinine (umol/L)	NA (mmol/L)	K (mmol/L)	Ca (mmol/L)
1	183	42.3	36.7	5.9	3.7	6.25	7	41	137	4.3	2.1
2	76	43.8	29.4	3.9	1.8	4.2	6.8	26	136	3.4	1.9
3	74	29.8	24.6	8.1	3.8	6.4	2.9	26	141	2.3	2.1
4	100	19.1	21.2	11.2	4.8	5.1	2.5	23	143	2.25	2
5	87	8.5	12	11.2	4.8	2.55	2.5	23	146	1.79	1.71
6	123	8.8	17.5	11.1	5.5	5.85	3.2	28	143	1.82	2.09
7	127	15.3	11.3	7	2	3.8	2.1	20	147	2.78	2.17
8	120	19	18	6.4	2	4.2	2.2	24	134	5.6	2.1
9	125	20.2	18.6	4	2.2	3.08	4.4	26	135	6.9	2.3

Conclusion

Purpura fulminans is a frequent complication of Neisseria meningitidis invasive infection, although relatively uncommon, and is associated with a high mortality rate (up to 50%).

The case presentation highlights life-threatening case of meningococcemia complicated with purpura fulminans, immediate empirical treatment in suspected cases of meningococcemia immediately after blood cultures are obtained, because prognosis is dependent upon early intervention with IV third-generation cephalosporin (ceftriaxone), hydrocortisone, intravenous immunoglobulin, Fluid resuscitation and proper management of DIC improve the outcome, our patient markedly improved.

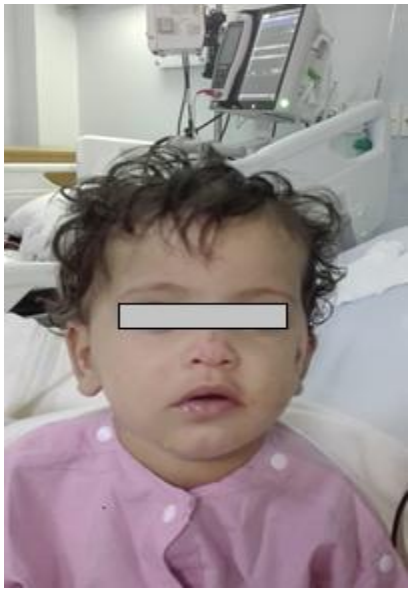

Figure (6): Patient markedly improved

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