## THE PROFILE OF NEONATAL SEPSIS IN DUHOK CITY AND PREDICTORS OF MORTALITY: A PROSPECTIVE CASE SERIES STUDY

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#### ABSTRACT

**Background:** Neonatal sepsis is a major cause of death all over the world. Risk factors represent an interaction between maternal-fetal colonization and each of transplacental immunity and the defense mechanisms of the neonate. This study is to assess the epidemiological, clinical and laboratory profiles of neonates with sepsis in relation to outcome and to determine the predictors of outcome.

**Subject and Methods:** A prospective study included neonates with sepsis admitted to neonatal care unit. 126 neonates with features of sepsis were included with age ranged from (1-30) days. From each patient, neonatal and maternal data were collected and clinical features as well as laboratory test results of hemoglobin, platelets count. total white blood cell and absolute neutrophil count , C-reactive protein and blood culture were collected and statistically analyzed.

**Results:** of 126 neonates, 32 (25.39%) died while others survived. Age < 7 days was in 61.9% of all cases, 69.84% had respiratory distress syndrome, 7.93% had hypoxic ischemic encephalopathy, 60.31% were preterm, 61.9% were born vaginally and male to female ratio was 1.73:1. There is a significant relation of mortality to respiratory distress syndrome and hypoxic ischemic encephalopathy, preterm delivery, low birth weight and male gender. Vomiting, apnea, sclerema, cyanosis and tachypnea were significantly related to the mortality. *Eschericia coli* were the most common followed by *Klebsiella sp.* The highest mortality is with Acenatobacterbaumani followed by *Staphylococcu aureus* with a significant relation. The C reactive protein was>10 mg/dl was in higher number of neonates with sepsis who died by comparison to those who survived, with a significant relation.

**Conclusions:** Neonatal sepsis is still a common cause of mortality in neonates with change in the pattern of causative organisms and this requires more monitoring and periodic surveillance. There is a real need to find out the local antibiotic sensitivities of pathogens to establish an optimal empirical treatment before the results of culture and sensitivity are available.

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 $\mathbf{N}$  eonatal sepsis is a major cause of death all over the world<sup>1</sup>.Up to 4 million neonates die annually in developing countries most commonly due to sepsis, hypoxic ischemic encephalopathy, and consequences of prematurity and low birth weight<sup>2,3</sup>. The

incidence of neonatal sepsis is developing significantly higher in countries than in developed ones 1-4 vs 10-50/1000 live birth<sup>4,5</sup>. Also. this incidence varies from a neonatal nursery to another and even it varies within the same nursery from time to time and depending

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on the predisposing conditions<sup>6</sup>. Risk factors represent an interaction between maternal-fetal colonization and each of transplacental immunity and the defense mechanisms of the neonate, both physical and cellular<sup>7</sup>. Sepsis in neonates manifests as either focal or non-specific signs and symptoms of infection<sup>4</sup>. The virulence of microorganism the and neonate's inflammatory response to that agent determine theclinical manifestations. The term systemic inflammatory response syndrome (SIRS) describes the unique process of infection and the subsequent response<sup>4</sup> while the systemic term systemic inflammatory response(SIR) describes the syndrome that includes two or more of the following: tachycardia, tachypnea, fever or hypothermia, and abnormal white blood cells in immature forms.It is important to evaluate tests for neonatal sepsis because the infection may be a serious threat to the neonate. It is urgently necessary to know if the neonate has sepsis to start treatment as early as possible<sup>8</sup>. There is no enough specificity and sensitivity of any single laboratory test used and therefore lab confirmation must be used in conjunction with risk factors and clinical signs<sup>5</sup>. The lab tests used are : blood, urine and cerebrospinal fluid culture, profile of white blood cells, platelet count, acute phase reactants (ESR, C reactive protein), latex agglutination tests, or counter immune electrophoreses, and Polymerase Chain Reaction (PCR)<sup>4-7,9</sup>. Synthesis of C reactive protein (CRP) increases within (4-6) hours and then doubles every 8 hours after that and peaks at 36-50 hours after the onset of inflammation. With ongoing inflammation and tissue destruction, CRP level remains

high, but declines rapidly with resolution of inflammation because of short half-life (4 to 7 hrs.), so it is parallel to the degree of injury and repair and this supports its value as an acute measure of disease activity. In the serum of normal healthy person CRP is in very low concentration < 0.02 mg/dl and mostly does not exceed 6 mg/dl)<sup>10-13</sup>. Depending on the definition of sepsis, the mortality rate from sepsis varies. When all bacteremic infections are included in the definition, the reported mortality rate in neonatal sepsis is 10- $40\%^5$ . To anticipate from the clinical to suspect from clinical history, presentation and to confirm diagnosis by preliminary laboratory test are essential to maintain intact survival of the neonate with sepsis<sup>7</sup>. To the best of our knowledge, there are no enough studies that cover this very vital subject in our locality. This study was accomplished on neonates with sepsis to assess the epidemiological, clinical and laboratory profiles of neonates with sepsis in relation to outcome (survival and mortality) and to determine the predictors of outcome.

## **METHODS**

A prospective study was accomplished on neonates with sepsis who have been admitted to neonatal nursery at Maternity and Obstetric Hospital in Duhok city from the first of March 2015 to the first of March 2016. A total of 126 neonates with features of sepsis were included(we excluded neonates with previous use of antibiotic and those having congenital anomalies). Their age ranged from (1-30) days. The following data were taken: name, age, sex, mode and place of delivery, date of admission, gestational age (was assessed using Dubowitz criteria)<sup>4</sup>, any history of acute neonatal suffering i.e. any illness during birth or soon after it such as hypoxic ischemic encephalopathy and respiratory distress. Maternal data included: history of prolonged rupture of membrane more than 24 hour, antibiotic use, fever, and urinary tract infection (UTI). Clinical features of neonates included: lethargy, poor feeding, diarrhea, coffee-ground vomiting, temperature instability, convulsion, pallor, jaundice, cyanosis, tachycardia, apnea, respiratory distress, mottled skin, sclerma, omphalitis, hepato-splenomegaly and abdominal distension. The neonates were followed throughout their presence in the hospital and were divided into those who remained alive and those who died. A sample of 0.5 ml of blood was taken from every neonate for estimation of hemoglobin, platelets count, total white blood cell and absolute neutrophil count and before antibiotic use. A sample of at least 2ml of blood per set was taken from peripheral vein from 2 separate sites after adequate disinfection of skin by iodine solution that was left to dry and then wiped off using (70%) alcohol, then the both samples were cultured aerobically and anaerobically. C-reactive protein was measured using 0.5 ml of blood collected in a plain tube without EDTA by latex-agglutination test. The cutoff value for CRP >  $10 \text{mg/dl}^{4,5,10-13}$  was considered positive.

#### STATISTICAL ANALYSIS

Statistical analysis was done using SPSS package 20, data were expressed as mean + SD, Chi-square and exact Fisher's test were used for comparison of proportions, P-value of less than 0.05 was considered as statistically significant, P-value <0.01 as highly significant and P-value <0.001 as extremely significant.

The homogeneity of patients' age, weight, and BMI was examined through the Oneway ANOVA statistical tests. The differences between sensory and motor duration among three study groups were evaluated through the One-Way ANOVA and post-hoc statistical tests and chisquared tests for adverse effects of different doses of dexamethasone. The pvalue less than 0.05 was considered as statistically significant and less than 0.01 as a clinically substantial difference. The Statistical Package for Social Sciences version 23:00 (SPSS: IBM) was used for statistical calculations.

#### RESULTS

Among all participants, 32 (25.39%) died while others survived. Most common age of patient was less than 7 days in 61.9% of all cases, 69.84% had respiratory distress syndrome, 7.93% had hypoxic ischemic encephalopathy, 60.31% were preterm, 61.9% were born vaginally and male to female ratio was 1.73:1. The outcome of in relation neonates' sepsis to characteristics is shown in Table 1. There is a significant relation of mortality to respiratory distress syndrome and hypoxic ischemic encephalopathy, preterm delivery, low birth weight and male gender.

#### THE PROFILE OF NEONATAL SEPSIS IN DUHOK CITY AND PREDICTORS Table 1: The Relation of Neonates' Variables to the Outcome of Neonates with Sepsis P value Variables Outcome Alive 94 Dead 32 Age (days) <7 (78) 56(71.7%) 22(28.3%) 0.256 7-28 (48) 38(79.1%) 10(20.9%) Acute suffering RDS\* (88) 62(70.4%) 26(29.6%) 0.031 HIE\*\* (10) 6 (60%) 4(40%) 2(7.2%) 26(92.8%) None (28) **Gestational Age** Preterm (76) 48(63.1%) 28(36.9%) 0.0001 Term (50) 46(92%) 4 (8%) **Birth weight** 2288+/- 776 1825+/- 588 0.0001 Mean+/- SD (grams) Range 900-3800 800-3000 **Delivery mode** Vaginal (78) 56(71.8%) 22(28.2%) 0.256 38(79.2%) 10(20.8%) Caesarean (48) 0.009 Sex Male (80) 54(67.5%) 26(32.5%) Female (46) 40(87%) 6(13%)

\*Respiratory distress syndrome

\*\* hypoxic ischemic encephalopathy

The maternal characteristics include prolonged rupture of membranes that occurred in 3.17% of cases, the use of antibiotics before delivery in 7.93%, maternal fever in 9.52% and urinary tract infection in 17.46% of all cases. As shown in **Table 2**, none of these variables was significantly related to the outcome of sepsis.

Variables		outc	P value	
		Alive 94	Dead 32	
PROM*	Yes	4(100%)	0(0%)	0.222
	No	90(73.8%)	32(26.2%)	
Antibiotics use	Yes	8(80%)	2(20%)	0.625
	No	86(74.2%)	30(25.8%)	
fever	Yes	6(50%)	6(50%)	0.092
	No	88(77.2%)	26(22.8%)	
UTI**	Yes	16(72.8%)	6(27.2%)	0.955
	No	78(75%)	26(25%)	

\*Respiratory distress syndrome

\*\* hypoxic ischemic encephalopathy

The clinical symptoms of sepsis are presented in **Table 3.** Lethargy and poor feeding are the most frequent symptoms.

Vomiting is significantly related to mortality while the other symptoms are not.

Symptoms		Outcome		Total	P value
		Alive 94	Dead 32		
Lethargy	Present	66(75%)	22(25%)	88	0.967
0.	Absent	28(73.7%)	10(26.3%)	38	
Poor feeding	present	62(73.8%)	22(26.2%)	84	0.622
	Absent	32(76.2%)	10(23.8%)	42	
Diarrhea	present	4(66.7%)	2(33.3%)	6	0.701
	Absent	92(75.4%)	30(24.6%)	120	
Vomiting	present	12(54.6%)	10(45.4%)	22	0.027
_	Absent	82(78.9%)	22(21.1%)	104	
Seizures	present	12(66.7%)	6(33.3%)	18	0.483
	Absent	82(75.93%)	26(24.07%)	108	

\*Respiratory distress syndrome\*\* hypoxic ischemic encephalopathy

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Tachypnea, cyanosis, sclerema and apnea were the most frequent signs in septic neonates followed by jaundice, fever, hypothermia and abdominal distension. As shown in **Table 4**, apnea, sclerema, cyanosis and tachypnea were significantly related to the mortality.

		Outo	come	Total	P value
Sigr	1	Alive 94	Dead 32		
Fever	present	26(86.7%)	4(13.3%)	30	0.061
	absent	68(70.9%)	28(29.1%)	96	
Hypothermia	present	14(58.4%)	10(41.6%)	24	0.063
	absent	80(78.5%)	22(21.5%)	102	
Cyanosis	present	24(57.2%)	18(42.8%)	42	0.004
·	absent	70(83.4%)	14(16.6%)	84	
Apnea	present	22(55%)	18(45%)	40	0.001
-	absent	72(83.73%)	14(16.27%)	86	
Tense fontanel	present	4(50%)	4(50%)	8	0.121
	absent	90(76.3%)	28(23.7%)	108	
Tachypnea	present	38(86.4%)	6(13.6%)	44	0.017
	absent	56(68.3%)	26(31.7%)	82	
Pallor	present	14(77.8%)	4(22.2%)	18	0.653
	absent	80(74.1%)	28(25.9%)	108	
Jaundice	present	28(77.8%)	8(22.2%)	36	0.487
	absent	66(73.4%)	24(26.6%)	90	
Purpura	present	6(75%)	2(25%)	8	0.918
-	absent	88(74.6%)	30(25.4%)	118	
Sclerema	present	24(57.2%)	18(42.8%)	42	0.004
	absent	70(83.4%)	14(16.6%)	84	
Abdominal	present	12(60%)	8(40%)	20	0.139
distension	absent	72(75%)	24(25%)	96	
Hepato-	present	2(50%)	2(50%)	4	0.281
splenomegaly	absent	92(75%)	30(24.6%)	122	

\*Respiratory distress syndrome

\*\* hypoxic ischemic encephalopathy

According to the results of blood culture, the most common isolated bacteria were *Eschericia coli* followed by *Klebsiella sp.* and then *Non coagulase staphyllococci and* non-lactose fermenters. As in **Table 5**, the highest mortality is with Acinatobacter baumannii followed by Staphylococcus aureus and then Escherichia coli and Klebsiella sp. with a significant relation.

<b>Bacteria isolated</b>	Total	Out	come
		Alive 94	Dead 32
Escherichia coli	74	54 (72.98%)	20 (27.02%)
Klebsiella sp.	18	13 (72.23%)	5 (27.77%)
Non coagulase staphyllococci	11	11 (100%)	0 (0%)
Non lactose fermentors	11	11 (100%)	0 (0%)
Acinetobacter baumannii	4	0 (0%)	4 (100%)
Staphylococcus aureus	4	2 (50%)	2 (50%)
Gram positive cocci	4	4 (100%)	0 (0%)

P=0.003

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The hematologic variables are presented in **Table 6.** The mean hemoglobin, platelet count, white blood cell count and platelet count is lower in neonates who died of sepsis as compared to those who survived but no statistical significance was found.

The C reactive protein as shown also in this table is  $\geq 10$ mg/dl in a significantly higher number of neonates with sepsis who died by comparison to those who survived, with a significant relation.

Variables	_	Outcome		Р	
	_	Alive 94	Dead 32		
Hemoglobin (g /dl)		14.6+/- 3.7	13.4+/- 3.9	0.18	
Platelets (cell / mm3)		176+/- 136	174.78+/- 172.15	0.075	
White blood cells (cell / mm3)		15.1+/-9.48	14.4+/-9.47	0.091	
Absolute neutrophil count (cell / mm3)		13.6+/-4.2	5.9+/-4.8	0.077	
C-reactive protein	Positive	59	28	0.003	
-	Negative	35	4		

### **DISCUSSION**

To evaluate the perinatal care in a community it is wise rely on the neonatal mortality rate to establish an effective health care delivery system. It is very essential to have an integrated statistical information about the neonatal mortality in order to develop a sound program for the early diagnosis of the neonatal sepsis and assessment of treatment and outcome<sup>14</sup>. Neonatal sepsis may just manifest as diverse, subtle and nonspecific signs and symptoms. If the diagnosis is not made early and treatment not started immediately, both morbidity and mortality rates rise significantly<sup>12</sup>. Mortality from neonatal sepsis in this study was 25.39%, which is close to the results of other studies where it was in United Arab Emirates  $(26\%)^{15}$ , in USA<sup>16</sup>, and Saudi Arabia it was 28%<sup>17</sup>. It is higher than what was found in Nigeria, where it was 19.3%<sup>18</sup> but the mortality is lower than two Iraqi studies where they were(44.2%)<sup>19</sup> and (43.5%)<sup>20</sup>, a study in Nepal $(36.95\%)^{21}$  a Saudi study  $(44\%)^{22}$  and Mexican study  $(43.9\%)^{23}$ . Many factors

explain the difference in mortality rate among different countries like use of ventilators. different microorganisms, socioeconomic and racial factors, incubators, use of different antibiotics and geographical factors<sup>15</sup>. Although early onset sepsis is more frequent in this study and the mortality is higher than late onset, it is not significant. Other studies have proved similar results significant differences<sup>19,23,24</sup> and with conversely, others found late onset sepsis to be associated with higher mortality<sup>20,25,26</sup>. However, the causative agents in early onset sepsis mainly comes from mother's genitorurinary tract while in late onset sepsis it comes from prolonged antibiotic use, procedures invasive and prolonged hospitalization. Male gender is a predictor of mortality in this study, which suggests the probability of sex related factors in host susceptibility. Similar results were found by other studies<sup>17,27</sup> while others did not find any role of sex in predilection to mortality  $^{15,21,23}$ .

Mostly, because of inherent immunodeficiency in premature neonates

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and the need for prolonged hospitalization in low birth weight neonates, sepsis was more common and mortality was higher in these two groups of neonates in our study. This is similar to what was found in other studies in different parts of the world<sup>15,17,22,23, 27-29</sup>, but different from other studies<sup>21,31</sup> that found them not significant. Neonates who had, in addition to sepsis, other acute illnesses like respiratory distress and hypoxic ischemic encephalopathy did show a significantly higher mortality in agreement with what was found in other studies <sup>17,23</sup> because they need prolonged hospital stay and may be subjected to more invasive procedures. Similar to what was found in a Saudi study<sup>22</sup>, prolonged rupture of membranes was not found frequently in septic neonates and is not predictor of mortality from sepsis, probably because the affected mothers are treated with antibiotics in such cases which seems to be protective for neonate. This is in contrast to other different studies that found it a significant factor<sup>15,19, 24,27, 29-31</sup>.

Among presenting signs and symptoms of sepsis, predictors of mortality were apnea, cvanosis. sclerema and vomiting in accordance with other studies<sup>1,s19,23,27,30</sup>. The causative microorganisms isolated from were most culture blood commonly Escherichia coli followed by Klebsiella sp. with similar mortality rates, while the highest 1. Weber of mortality was found rate with Acinatobacter baumannii followed by Staphylococcus aureus. This is similar to another study<sup>30</sup> but in contrast to an Iraqi study<sup>19</sup> where the mortality rates were P. 2. Meeting aeruginosa (100%), Staphylococcus aureus (100%) followed by klebsiella (71.1%) and E. coli (48.5%) and other different studies showed similar hematological variables including

hemoglobin, white blood cells, absolute neutrophil count and platelets were found lower in septic neonates who died as compared to those who survived but this difference was not significant. These were found significantly lower in those who died in other studies<sup>12,19, 23,33,34</sup> since the toxins produced by the causative bacteria suppress the bone marrow hematopoietic process.

The mortality was higher in septic neonates with C-reactive protein level > 10 mg/dl. This agrees with other studies<sup>10,13,19,34</sup>. CRP has a high sensitivity and specificity with high negative predictive values and high positive predictive values as well<sup>12</sup>.

The main limitation of this study was that serum procalcitonin was not measured for the neonates with sepsis since it is more sensitive and specific than CRP.

In conclusion, Neonatal sepsis is still a common cause of mortality in neonates. There is a change in the pattern of organisms causing sepsis in the newborn. This requires more monitoring and periodic surveillance, and there is a real need to find out the local antibiotic sensitivities of pathogens to establish an optimal empirical treatment before the results of culture and sensitivity are available.

#### REFERENCES

- MW, Carlin JB, Gatchalian D, Muhe L, Mulholland S, Lehmann EKetal. Predictors of neonatal sepsis in developing countries. Pediatric Infect Dis J 2003; 22(8):711-717.
- report. Explore simplified antimicrobial regimens for the treatment of neonatal sepsis. WHO, Geneva 30th September- 1st October 2002;1.
- results<sup>15,20-23,26,32</sup>.The 3. Mathai E, Christopher U, Mathai M, Jana AK, Rose D, Bergstrom S. Is Creactive

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protein useful in differentiating infected from uninfected neonates among those at risk of infection?. Indian J Pediatrics 2004; 41(9):895-900.

- Stoll BJ, kleigman RM. The fetus and the neonatal infant. In: Behrman RE, Kliegman RM, Jenson HB (ed). Nelson's Textbook of Pediatrics. 17ed. Philadelphia. WB Saunders CO 2004; 552, 623- 639.
- Khalid N. Neonatal infection. In: McIntosh N, Helms P, Smyth R (eds). Forfar and Arneil textbook of pediatrics. 6th ed. Philadelphia. Churchill Livingstone CO 2003; 336-343.
- Gross M. Infection of Neonates. In: Rudolph A, Hoffman J, Rudolph C (eds). Rudolph's Pediatrics. 20th ed. United States of America. Prentice Hall International CO 1996;530-536.
- Finer N. Neonatal sepsis. San Diego Journal of Pediatrics for Neonatology 2003; 15(5): 855-867.
- Chiesa C, Panero A, Osborn JF, Simonetti AF, Pacifico L. Diagnosis of neonatal sepsis: a clinical and laboratory challenge. Clinical Biochemistry 2004; 50: 279-287.
- 9. Loo S. Neonatal Sepsis. Hawaii Journal of Pediatrics 2002; 10 (3): 49-55.
- Isaac man D, Burke B. Utility of serum C-reactive protein for detection of occult bacterial infection in children. Archives of Pediatrics and Adolescent Medicine 2002; 156: 903-909.
- Nuntnarumit P, Pinkaew O, Kitiwanwaichs. Predictive values of serial C-reactive protein in neonatal sepsis. J. Med Assoc Thai 2002; 85(4): 1151-1158.
- 12. Ahmed Z, Ghafoor T, Waqar T, Ali S, Aziz S, Mahmud S. Diagnostics value

of C-reactive protein and hematological parameters in neonatal sepsis. J Coll Physician Surgpak 2005; 15(3): 152-156.

- 13. Chiesa C, Pellegrini G, Panero A. Creactive protein, interleukin-6, and procalcitonin in the immediate postnatal period: influence of illness severity, risk status, antenatal and perinatal complications and infection. Clin Chemist 2003; 49(1):60-68.
- 14. Bassuni W, Abbag F, Asindi A. Neonatal Death in Asir region of Saudia: Experience in a referral Neonatal intensive care unit. Saudi Med J 1995; 21 (2): 16-24.
- 15. Koutouby A, Habibullah J. Neonatal sepsis inDubai, United Arab Emirates. J tropical pediatrics 1995; 41: 177-180.
- Stoll B, Holman R, Schuchat A. Decline in sepsis associated Neonatal and infant death in United States, 1979 through 1994. J of Ped. 1995; 102 (2): 18-26.
- Dawadu A, AL-Umran K, TwumDanso K. A case control study of Neonatal sepsis: Experience from Saudi Arabia. J tropical pediatrics 1997; (43): 84-88.
- Ezechukwa CC, Ugochukwu A, Egbuonu I, Chukwuka JO. Risk Factors for neonatal mortality in a regional tertiary hospital in Nigeria. Nigerian Journal of clinical practice 2004; 7(2):50-52.
- JumahDS ,HassanMK. Predictors of mortality outcome in neonatal sepsis. Medical Journal of Basrah University2007;25(1):11-18.
- 20. Radhy H. Neonatal sepsis causative agents and outcome. Thesis submitted to the Iraqi commission for medical specialization 2001; 1-36.
- Shrestha S, Dongol Singh S, Shrestha NC, Shrestha RPB, Madhup SK. Comparision of Clinical and Laboratory

Parameters in Culture Proven and Unproven Early Onset Sepsis in NICU. *Kathmandu Univ Med J*2013;44(4):310-314.

- Asindi A, Bilal N, AL-shehri M, Fatinni YA, Manna N, Habeeb SM. Neonatal sepsis. Saudi Med J 1999; 20 (12): 942-946.
- Rodriguez M, Canadiani C, Garcia J, Gutiérrez P, Sánchez F.. Morbidity and Mortality from neonatal sepsis in a tertiary care level hospital. Saludpublica de Mexico 2003; 45 (2): 90-95.
- 24. Schuchat A, Zywieki S, Dinsmoor M, Mercer B, Romaguera J, O'Sullivan MJ*et al.* Risk factors and opportunities for prevention of early onset neonatal sepsis: A multicenter case-control study. Georgia J of Pediatrics 2000; 105 (1): 21-26.
- 25. Stoll B, Hansen N, Fanaroff A. late onset sepsis in very low Birth weight neonates: the Experience of the NICHD Neonatal Research Net work. J of pediatrics 2002; 110(2): 285-291.
- 26. Obi J, kafrawi M, Igancio L. Neonatal septicemia. Saudi Med J 1999; 20(6): 433-437.
- 27. Gebrehiwot A, Lakew W, Moges F, Moges B, Anagaw B, Unakal C *et al.* Predictors of positive blood culture and death among neonates with suspected neonatal sepsis in Gondar University Hospital, Northwest Ethiopia. *Euro. J. Exp. Bio., 2012;2 (6):2212-2218*
- 28. Morgan M, Ruel T, Kumar G S, Sabnis A, Kaiser S. Predictors of Neonatal

Sepsis in Rural Karnataka, India. Asian Journal of Clinical Pediatrics and Neonatology 2013;1(4):73-6.

- 29. Sharma D, Kumar C, Pandita A, Pratap OT, Dasi T, Murki S. Bacteriological profile and clinical predictors of ESBL neonatal sepsis. J matern Fetal Neonatal Med. 2016;29(4):567-70.
- 30. Kayange N, Kamugisha Ε, Mwizamholya1 D, Jeremiah S, Mshana S. Predictors of positive blood culture among neonates and deaths with suspected neonatal sepsis in a tertiary Mwanzahospital. Tanzania. BMC Pediatrics 2010; 10:39.
- Puopolo K, Draper D, Wi S, Newman T, Zupancic J, Lieberman E *et al* .Estimating the Probability of Neonatal Early-Onset Infection on the Basis of Maternal Risk Factors. *Pediatrics* 2011;128:e1155–e1163.
- 32. Jaber E, AL. Zwaini k. Neonatal septicemia in the neonatal care unit in AL- Anbar governorate in Iraq. East Med. Health J 2002; 8 (4): 30-36.
- 33. Guida JD, Kunig AM, Leet KH, McKenzie SE, Paul DA. Platelet count and sepsis in very low birth weight neonates: is there an organism-specific response? Pediatrics 2003;111:1411-1415.
- 34. Dhananjay, Kumar S .Comparison Of Biochemical and Pathological Markers in Neonates with Sepsis and Neonates without Sepsis. Int J Biol Med Res. 2011; 2(4): 1131 – 1134.

## ثوختة

# سيمايى ثيسبونا خوينى لدةف زاروكيت سافال دهوك و ثيَشبينيكرنا ئةنجامى وى ذجورى مايلويدا دذوار هةى. ظةكولينةكا ثاشظةيى لسةر 105 نةخوشان

## ثيَشةكى:

ثيسبوناخوينىًادةفز اروكيَنسافائةطةر ةكستر ةكديةيمر نىَاسةر انسةر ىجيهانيَ . نيشانيَنكاينيكنديار دكةنو ةكدةظةر دارياننةجوري . هوكاريَنتوشبونىَبثيسبوناخوينيَ هةظركي ية ناظا ئاكنجيبونا ميكروبي لدةف زاروكي ودايكيَ لطةل برطريا لدةف زاروكيَ سافا.

دةستنيشانكرنا سيماييَن ثيسبونا خوينيَ لدةف زاروكيَن ساظا ذ ئالييَ ذينطةهي و كلينيكي و لابوري و ثيَكظة طريدانا وان لطةل ئةنجاميَ ظيَ ثيسبونيَ و ثيشبيني كرنا ئةنجامي .

#### ريكيَن ظةكولينيَ:

ئةظ ظةكولينة هاتة كرن لسةر زاروكين ساظابين توشبوينة ثيسبونا خوينى ئةوين هاتينة نظاندن ليةكةيا ظرينكى لنةخوشخانا زاروكبونى ل دهوكى ذ ( 1 ى ئادارا 2015 بو 1 ى ئادارا 2016 ) دمارا ظان نةخوشان ( 126 ) بوون كو هةميا نيشانين ثيسبونا خوينى لى دياربون كو ذيين وان ذ ( 1 تا 30 ) روذابوون ذ هةر زاروكةكى ئةظ ثيرَ انينيَن خوارى هاتنة وةرطرتن :- ناظ ، ذى ، رةطةز ، جورى بونى ، ميدويا نظاندنى ، ذيى كورثةلةى دةمى بوونى .... هند . ثيرَ انين ذ دايكا هاتينة وقرطرتن :- ماط ، ذى ، رةطةز ، جورى بونى ، ميدويا نظاندنى ، ذيى كورثقلةى دةمى بوونى ..... هند . ثيرَ انين ذ دايكا هاتينة وقرطرتن :- ماوى نابةينا ثةقينا سةرئافكى و بوونا زاروكى ، تا ، بكارئينانا بوونى ..... هند . ثيرَ انين ذ دايكا هاتينة وقرطرتن :- ماوى نابةينا ثقينا سةرئافكى و بوونا زاروكى ، تا ، بكارئينان بودنى بايوتيكا ...... هند . نيشانين توشبونى لدةف زاروكى هاتنة توماركرن . زاروك هاتنة دابةشكرن بو ئةوين ماينة دذيانى داو ئةويَن مرين . خوين هاته كيشان ذ هةر زاروكى ذ بو جاندنا خوينى و همرومسا ثيطانا هيمو طوبينى و دذيانى داو ئةويَن مرين . خوين هاتة كيشان ذ هار زاروكةكى ذ بو جاندنا خوينى و هرومسا ثيطانا هيمو طوبينى و يتبكين خوينى مرين . خوين هاته كيشان ذ هار زاروكه كى ذ بو جاندنا خوينى و هرومسا ثيطانا هيمو طوبينى و دنيانى داو ئةويَن مرين . في مرين . حوين هاته كيشان ذ هو زاروكى هاتنة توماركرن . زاروك هاتنة دابةشكرن بو ئويَن ماينة دنيانى داو ئةويَن مرين . خوين هاته كيشان ذ هار زاروكةكى ذ بو جاندنا خوينى و هرومسا ثيطانا هيمو طوبينى و يطرنى خوينى خوينى ماين ... ياطرنطة ...

#### ئةنجام:

ذ 126 بتشدار بويان ، 32 ( 25.39 % ) مرن ذيني كيَمتر ذ 7 روذا لدقف ( 61.6 % ) ذ وان بو . تقنطةبيَن بيني لدقف ( 69.48 % ) هتبوو توشبونا ميَشكي لدقف ( 7.93 % ) هتبوو . بوونا ثيَشوةخت ( ظرنيكي ) لدقف ( 60.31 % ) هتبوو . ( 61.6 % ) ذ بتشدار بويان سروشتي ببوون . ريَذا نيَر بو من ( 1.73.1 ) ثيَكظة طريدانا طرنط هتبوو نابقينا مرني و هترئيك ذ تنطة بيني ، توشبونا ميَشكي ، بونا ظرينكي ، كيَشا كيَم دةمي بونني و رقطةزي نيَر . ماوي دريَذ نابقينا ثقينا سترئيلك ذ تنطة بيني ، توشبونا ميَشكي ، بونا ظرينكي ، كيَشا كيم دةمي بوني و رقطةزي نيَر . ماوي دريَذ نابقينا ثقينا سترئاطكي وبوني لدقف ( 3.17 % ) هتبو . ( 3.93 % ) ذ دايكان ئتنيتي بايوتيك يي ثير . ماوي دريَذ نابقينا ثقينا سترئاطكي وبوني لدقف ( 3.17 % ) هتبو . ( 3.93 % ) ذ دايكان ئتنيتي بايوتيك يي ثير . ماوي دريَذ نابقينا شترين نيشان لدقف توشبويان :- خاطي ، كيم خارن ، ثينا بلةز ، شين بون ، رقق بون و يي ثير طنا بيتي . ثيكظة طريدانا طرنك هتبو نابقينا مرني و هترئيك ذ دل رابوني ، راوقستانا ينني ، رقق بون و راوستانا بيتي . ثيرين ميكروب (E. coli ) بو ثاش ( 1.68 % ) بو ، ثينا بلةز ، شين بون ، رقق بون و بوني و بينا بلةز . ثترين ميكروب (E. coli ) بو ثاش ( .798 هجوانا ميزين ، رو مي ، ماين يوني و بينا بلةز . ثترين ميكروب (E. coli ) بو ثاش ( .700 هت) المراد . هترينيك ذ همو طويين و اقراص و توني و بينا بلةز . ثترين ميكروب (Staph aureus) ) ثير ذ ( 10 ) ملغم/ دل ثتريو لدةف مريان . تةتكين ستي كيمتر بون لدة طنوي مرين بيةس نقياطرنط بو ( 201 ) ثير ذ ( 10 ) ملغم/ دل ثتربو لدف مريان .

#### دةرئةنجام:

ثيسبونا خوينىَ هيَشتا ئةطةرةكىَ طرنطة بو مرنىَ لدةف زاروكيَن ساظا لطةل طهورينا جورى ميكروبان ئةظةدى ثيَويستة كو ديظجونا لدويف بيَتة كرن . طقلةكا طرنطة جورىَ ئةنيتبا يوتيكيَن كاريطةر بهيَتة دياركرن داكو بشيَوةيةكي نموونةيي بهيَتة دان بةرى ئةنجاميَن خاندنا خوينيَ .

#### الخلاصة

## صورة انتان الدم عند حديثي الولادة في دهوك ومتنبئات الوفاة

**الخلفية والأهداف**: انتان الدم عند حديثي الولادة سبب رئيسي للموت في جميع انحاء العالم, تظهر علاماته السريرية بشكل اعراض موقعية او غير محددة للانتان, عوامل الخطورة الممهدة لانتان الدم تمثل تداخلا بين الاستيطان الجرثومي للام و الطفل وبين المناعة المنتقلة خلال المشيمة و اليات الدفاع عند حديث الولادة. اهداف البحث هو تحديد مواصفات حديثي الولادة المصابين بأنتان الدم من الناحية الوبائية و السريرية و المختبرية وارتباطها بنتيجة هذا الانتان و تحديد المتنبأت بالنتيجة.

**طرق البحث**: شملت الدراسة الاطفال حديثي الولادة المصابين بإنتان الدم الذين ادلخلوا الى وحدة العناية لحديثي الولادة في مستشفى الولادة في دهوك للفترة من (1 أذار 2015 الى 1 أذار 2016) شملت الدراسة 126 طفل حديث الولادة تظهر عليهم علامات الاصابة بإنتان الدم مع استثناء الاطفال الذين اعطوا مضادات حيوية و المصابين بتشوهات خلقية . تراوحت اعمار المشمولين بين ( 1-30 ) يوم ، اخذت من كل طفل مشترك المعلومات الاتية : الاسم . العمر . الجنس . تراوحت اعمار المشمولين بين ( 1-30 ) يوم ، اخذت من كل طفل مشترك المعلومات الاتية : الاسم . العمر . الجنس . تراوحت اعمار المشمولين بين ( 1-30 ) يوم ، اخذت من كل طفل مشترك المعلومات الاتية : الاسم . العمر . الجنس . طريقة الولادة . تاريخ الدخول . العمر الجنيني عندة الولادة . اصابات حادة مثل متلازمة عسا النقس او اعتلال الدماغ الناتج عن قلة الدم و الاكسجين للدماغ ، المعلومات المأخوذه من الام شملت : فترة طويلة فاصلة بين تمزق الاغشية و الولادة . الناتج عن قلة الدم و الاكسجين للدماغ ، المعلومات المأخوذه من الام شملت : فترة طويلية يا منوق الاغشية و الولادة ، استعمال المضادات الحيوية . الحمى التهاب المجاري البولية . تم تسجيل العلامات السريرية ايضاً و تم تقسيم و الولادة ، استعمال المضادات الحيوية . المعلومات المأخوذه من الام شملت : فترة طويلة فاصلة بين تمزق الاغشية و الولادة ، استعمال المضادات الحيوية . المعلومات المأخوذه من الام شملت : فترة طويلة و من تقسيم و الولادة ، استعمال المضادات الحيوية . الحمى التهاب المجاري البولية . تم تسجيل العلامات السريرية ايضاً و تم تقسيم و الولادة ، المعواني المناعان و تم تقليم من الدم لاجراء فحوص مكونات الدم ، البروتين التفاعلي ، وزرع المرض الى احياء ومتوفين . اخذت من كل طفل عينة من الدم لاجراء فحوص مكونات الدم ، البروتين التفاعلي ، وزرع و المرض الى الموائي و النوائي و يردي و و اللاهوائي و يردي و المرض الى و اللاهوائي، ثم تحليل النتاًج احصائياً بأستخدام نظام ( SPSS حيث 2005) حيات من كل طفل و الكم و الدم الهوائي و اللاهوائي النائع النائي المتخدام نظام ( SPSS حيث 2005) حيات الدم الولياً .

النتائج:من اصل 126 مشارك توفي 32 ( 25.9% ) . عمر المصابين اقل من 7 أيام من 61.9% من الحالات . عسر التض وجد عند 60.48% امتلال الدفاع عند 7.93% . والولادة الحذية وجدت عند 60.31% . والولادة المهبلية وجدت عند 61.9% من الحالات . الذكور الى الاناث 1:7.31وجد ارتباط هام احصائياً بين الوفيات وكل من عسر التنفس . اعتلال الدماغ . الولادة الخديجةز قلة الوزن عند الولادة وجنس الذكر .الفترة الطويلة بين تمزق الاغشية و الولادة وجدت عند 7.15% من الحالات . استعمال المصادات الحيوية من قبل الام قبل الولادة وجد عند 3.05% من الحالات . الحميفي عند 7.15% من الحالات . استعمال المصادات الحيوية من قبل الام قبل الولادة وجد عند 3.05% من الحالات . الحميفي عند 7.15% من الحالات . استعمال المصادات الحيوية من قبل الام قبل الولادة وجد عند 3.05% من الحالات . الحميفي عند 7.15% من الحالات . استعمال المصادات الحيوية من قبل الام قبل الولادة وجد عند 3.05% من الحالات . الحميفي عند 7.15% من الحالات . المعار عن البولية عند 17.46% من الحالات و لكن دون وجود ارتباط هام احصائياً . اكثر العلامات السريرية شيوعاً عند المصابين هي الخمول قلة الرضاعة تسارع اتننفس . الازرقاق . التصالب و توقف التنفس . اكثر الجراثيم شيوعاً عند المصابين كان Klebsiellaspp . على نسبة وفيات كانت من Acenatobacter ثم معد 3.05% من الحالات من معد الهيموكلوبين و الاقراص و الكريات البيضاء كل اقل عند المتوفين دون علاقة ماهمة . البروتين التفاعلي > 10 ملغم /دل كان اكثر شيوعيا عند المتوفين مع علاقة هام.

الاستنتاجات: اتنان الدم لايز ال سبباً مهماً للوفاة عند الولادة مع تغير نمط الجر اثيم اgمسببة مما يتطلب مزيداً من المراقبة والاستقصاء الدوري . هناك حاجة حقيقية لإيجاد حساسية الجر اثيم للمضادات الحيوية لغرض بدأ عيلاج تجريبي مثالي قبل نتائج الزرع والحساسية للجر اثيم .