Effect of the use of hydrocortisone on the WBC count in follow up of neonatal sepsis

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Abstract

Neonatal sepsis is a clinical syndrome characterized by many signs and symptoms which are non specific for diagnosis. Blood culture is standard measure but needs time to give it's results. Monocyte count is now used for early detection and follow up of patients of neonatal sepsis. Hydrocortisone therapy in neonatal sepsis is still controversial for many years as clarified by many studies. The aim of the study is to evaluate the role of hydrocortisone therapy on the WBC count in the follow up of patient with neonatal sepsis. The number of studied cases were 46 neonates diagnosed as a cases of neonatal sepsis after positive blood culture. Each one was assessed clinically by prepared questionnaire including history and clinical assessment. WBC count was done before hydrocortisone therapy for all included cases. The included cases were divided into two groups ,one group were given hydrocortisone and the other group were treated without hydrocortisone. Eight babies were died during the first week of the therapy and 38 cases were followed up after 1wk.by the same parameter that mentioned above. Very early neonatal sepsis was the commonest clinical type of sepsis 30(65,2%) with poor feeding is the common presentation 40(87%).Group B. *streptococcus* was the commonest bacteria isolated in 17 cases (37%).Before the hydrocortisone therapy WBC count was high in 28(60,9%). After 1 week of hydrocortisone therapy WBC count was abnormal in 4 cases (22,2%) in the group 1(with hydrocortisone therapy) while in patients with group 2(without hydrocortisone therapy) abnormal WBC count was(0) respectively.

The WBC count is not a good predictor test for diagnosis and follow up in non hydrocortisone using cases.

Introduction:

Sepsis is a clinical syndrome that complicates severe infection and is characterized by systemic inflammation and widespread tissue injury. In this syndrome, tissue is removed from the original insult that displayed the signs of inflammation, such as vasodilatation, increased microvascular permeability, and leukocyte accumulation. Multiple organ dysfunction is a continuum, with incremental degrees of physiological derangements in individual organs; it is a process rather than an event. [(1)].

The infectious agents associated with neonatal sepsis have changed over the past 50 years. *Staphylococcus aureus* and *Escherichia coli* were the most common bacterial infectious hazards for neonates during the 1950s in the United States. Additional organisms, such as *L monocytogenes, Chlamydia pneumoniae, H influenzae, Enterobacter aerogenes*, and species of *Bacteroides* and *Clostridium* have also been identified in neonatal sepsis. The most common risk factors associated with early onset neonatal sepsis include maternal GBS colonization (especially if untreated during labor), premature rupture of membranes (PROM),and prematurity. Risk factors also associated with early-onset neonatal sepsis include low Apgar score (<6 at 1 or 5 min), maternal fever, poor prenatal care, poor maternal nutrition, low socioeconomic status, low birth weight, difficult delivery, meconium staining, and congenital anomalies. The clinical signs of neonatal sepsis are nonspecific and are associated with characteristics of the causative organism and the body's response to the invasion. These nonspecific clinical signs of early sepsis syndrome are also associated with other neonatal diseases, such as respiratory distress syndrome (RDS), metabolic disorders, intracranial hemorrhage, and a traumatic delivery. Given the nonspecific nature of these signs, providing treatment for suspected neonatal sepsis while excluding other disease processes is prudent[(2,3,)].

Total neutrophil count (PMNs and immature forms) is slightly more sensitive in determining sepsis than total leukocyte count (percent lymphocyte + monocyte/PMNs + bands). Abnormal neutrophil counts at the time of symptom onset are only observed in two thirds of infants; therefore, neutrophil count does not provide adequate confirmation of sepsis[(4)].

The effect of sepsis and steroid on W.B.C count

White blood cells, or leukocytes, are classified into two main groups: granulocytes [(5)].

The life span of white blood cells ranges from 13 to 20 days, after which time they are destroyed in the lymphatic system. When immature WBCs are first released from the bone marrow into the peripheral blood, they are called "bands" or "stabs." Leukocytes fight infection through a process known as phagocytosis. During phagocytosis, the leukocytes surround and destroy foreign organisms. White blood cells also produce, transport, and distribute antibodies as part of the body's immune response[(6)].

Therapy with steroids modifies the leukocytosis response. When corticosteroids are given to healthy persons, the WBC count rises. However, when corticosteroids are given to a person with a severe infection, the infection can spread significantly without producing an expected WBC rise.

An important concept to remember is that, leukocytosis as a sign of infection can be masked in a patient taking corticosteroids[(6)].

Patient and method:

A prospective study was done on 46 cases of patients with neonatal sepsis admitted at the Tikrit Teaching Hospital during the period from 10th of march to 15th of july,2008 to identify the role of steroid in treatment of neonatal sepsis. The diagnosis of neonatal sepsis is done by clinical features of sepsis with positive blood culture which was done for all included cases. Each patient was evaluated clinically and by laboratory investigations by prepared questionnaire that include ;name, age, sex, onset of disease, maturity, weight, risk factors of neonatal sepsis and clinical presentation. WBC count at time of diagnosis and repeated 1 week after therapy were done to all the study cases. The study cases were divided into two groups .One group was given hydrocortisone 10 mg/kg in 4 divided doses for 1wk in addition to the line of treatment and another group treated without steroid. Eight cases were died during the first week of therapy and 38 case were followed up after one week by the same parameter that mentioned above.

Patients inclusion criteria :

1.Patients less than 28 days of age .
2.Signs and symptoms of sepsis .
3. presence of 2 or more of the following:[2]
A.Temperature greater than 38°C or less than 35°C.
B. Heart rate greater than 160 beats per minute.
C .Respiratory rate greater than 60 breaths per minute .
D .WBC count greater than 20,000 cells/µL, less than 5000 cells/µL.
4.positive blood culture .
5.No previous treatment with antibiotics .

A sample of at least 2ml of blood per set was taken from peripheral vein from 2 separate sites after adequate skin disinfection using iodine solution that left to dry and then whipped off with (70%) alcohol, both samples were taken before antibiotic administration, samples were cultured aerobically.

One to three milliliter of blood from 46 neonate were aspirated .The blood sample was inoculated in bottle containing 25 ml of brian heart infusion broth (Oxoid),this media contain Sodium Polyanthol Sulfonate (SPS) in a final concentration of 0.05%.This bottle then incubated for 18-24hr at 37C°[7.]

Macroscopic changes were observed in the next day. Gram stain was performed irrespective of macroscopic evidence of growth. And blind subculture on blood and chocolate agar plate were carried out, the first was incubated aerobically for 48 hr at 37C°.

If gram negative bacilli were detected by Gram stain or colony characteristics other samples from the bottle were subcultured on the third and seventh day before discarding the specimen as negative[7].

Blood Picture:

Blood samples were taken from neonates using Ethylene diamine tetra acetic acid (EDTAK3) anticoagulants. Impedance method(Sysmex NE 8000 cell counter,Toa Medical Electronic 'USA'Inc)which is fully automated hematology analyzer for the diagnostic testing of total WBCs count were used. The normal ranges for WBC count given in the reference range. [5]

Results:

The total number of cases was 46 neonates diagnosed as sepsis. Eight of them died during the first week of admission and 38 case were followed up after 1wk.Most of the cases were males 28 (60.9 %)and 18 (39.1%) of cases were females with male :female ratio was 1: 0.56 at presentation.

Table(1) Shows the distribution of cases according to the gender in regard to the age of onset of disease. Most of male and female cases presented in the very early onset (less than 12hr) of age 30(65.2%), then late onset 13(28.3%), and 3 (6.5%) in the early onset. And in all these onsets of disease the male cases were more than female cases.

Table(1) Distribution of cases according to the gender in regard to the age of onset of disease.

Onset	Male	Female	Total
<12 hr.very early onset	18(39.1%)	12 (26.1%)	30(65.2%)
12-72hr.early onset	2(4.3%)	1(2.2%)	3(6.5%)
>72hr.late onset	8(17.4%)	5(10.9%)	13(28.3%)
Total	28(60.9%)	18(39.1%)	46(100%)

Figure 1. Shows the distribution of study cases according to the maturity. Most of cases of neonatal sepsis occur in preterm patients 32(69.6%) and 14(30.4%) occur in full term neonates.

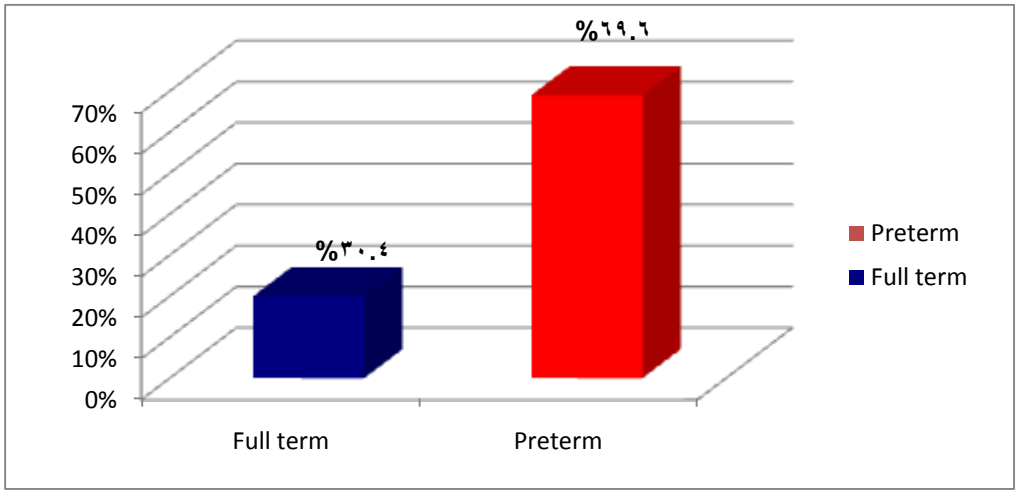
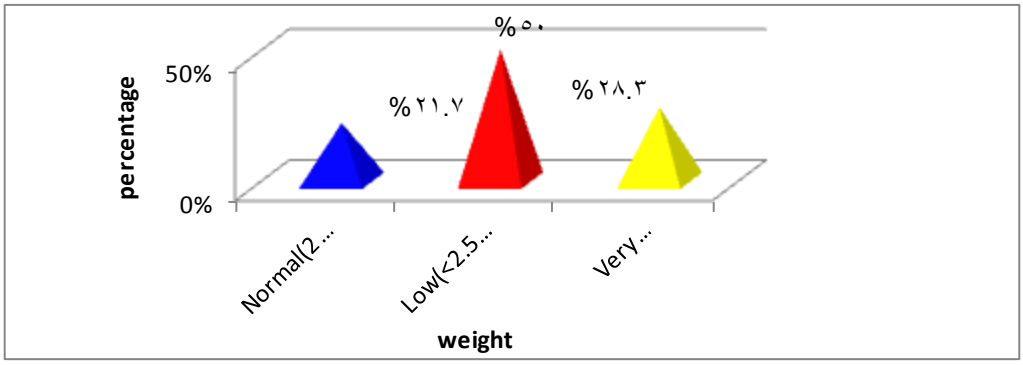


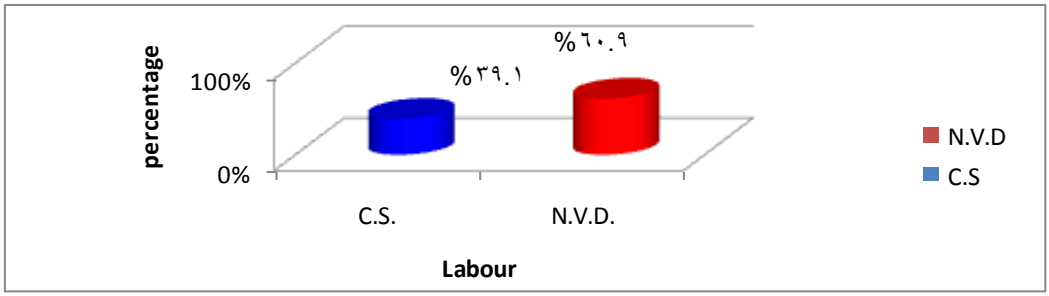
Figure (1) Distribution of cases according to the maturity .

Figure 2. Shows the distribution of study cases according to the weight. Most of neonatal sepsis cases occur in low birth weight patients 23(50%), in very low birth weight 13 (28.3%) and 10(21.7%) occur in normal birth weight patients.



Figure(2) Distribution of cases according to the weight .

Figure (3). Shows the distribution of cases according to the type of labour. Most neonatal sepsis cases occur in normal vaginal delivery 28(60.9%) and 18(39.1%) occur in C.S.



Figure(3) Distribution of cases according to the type of labour .

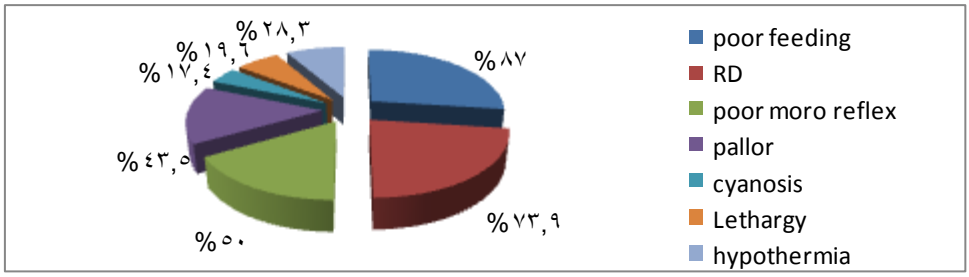
Table (2) Shows the most common risk factors of neonatal sepsis. Neonates with preterm delivery represents the most common risk factor of neonatal sepsis 32(69.6%), followed by male gender 28(60.9%).

Table(2) The most common risk factors of neonatal sepsis.

Risk factors	N0.	%
Preterm	32	69.6
Male gender	28	60.9
Maternal fever	28	60.9
Poor hand washing practice	27	58.7
Bottle feeding	20	43.5
Interference*	30	
Meconium aspiration	19	41.3
Prolonged rupture of membrane	15	32.6
Previous admission to incubator	12	26.1
Superficial skin infection	7	15.2

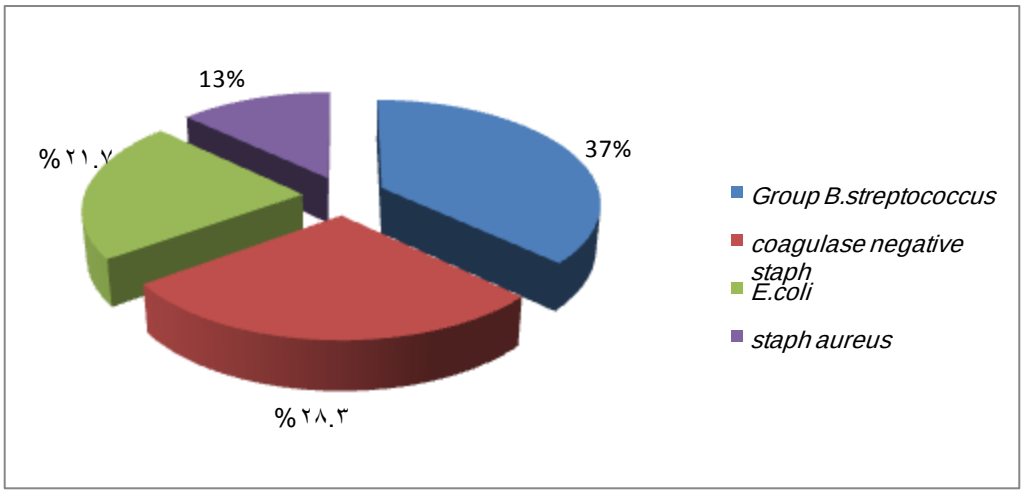
Interference*:vaccum, forceps, episiotomy delivery, umbilical catheterization. Endotrachial intubation, I.V. fluid users , suction.

Figure 4.Shows the most common signs and symptoms of neonatal sepsis. These were poor feeding 40 (87%), followed by RD 34 (73.9%), poor moro reflex 23 (50%), pallor 20 (43.5%), hypothermia 13 (28.3%), Lethargy 9 (19.6%), and 8 (17.4%) with cyanosis.



Figure(4) Distribution of cases according to presentation of the patients

Figure 5.Shows the distribution of cases according to blood culture results. As observed the most common bacteria that cause neonatal sepsis was group B. *streptococcus* 17(37%) followed by coagulase negative *staph* 13 (28.3%),then *E.coli* 10(21.7%), and 6(13%) *staph aureus*.



Figure(5) Distribution of cases according to blood culture results

Table (4.3)Shows the distribution of study cases according to the W.B.C count in regard to the age of patients. In regard to those who were less than 24 hr.of age, most of cases had High W.B.C. count 15 (53.6%) while normal W.B.C. count found in 13(46.4%), While those between 24hr. to 1month, WBC count was normal in 5(27.8%), and high in 13(72.2%)respectively.

Table (4.3)Distribution of study cases according to the W.B.C count in regard to the age of patients

Age	W.B.C		Total
	Normal	High	
<24hr N.R(9-34,000cells)	13 (46.4%)	15(53.6%)	28 (100 %)
24hr-1mo. N.R(5-19,000cells	5(27.8%)	13(72.2%)	18(100%)
Total	18(39.1%)	28(60.9%)	46(100%)

Table(3) Shows the distribution of study cases according to the W.B.C count in regard to the gender. Most of male cases have high W.B.C. count 20(71.4%), while in female cases, normal W.B.C. count was 10(55.6%)and high in 8 (44,4%) respectively.

Table(4)Distribution of study cases according to the W.B.C count in regard to the gender.

Gender	W.B.C		Total
	Normal	High	
Male	8(28.6%)	20(71.4%)	28(100%)
Female	10(55.6%)	8(44.4%)	18(100%)
Total	18(39.1%)	28 (60.9%)	46(100%)

Chi-Square = 3.346 DF = 1 P Value at 0.05 = 3.84 not significant

Table (5) Shows the distribution of study cases according to the W.B.C count in regard to the type of bacteria .Most of the bacteria show nearly similar presentation of normal and high W.B.C. count except for *staph aureus* which shows all W.B.C. count is high 6(100%).

Table (5) Distribution of study cases according to the W.B.C count in regard to the type of bacteria .

Bacteria	W.B.C		Total
	Normal	High	
Group B.streptococcus	8 (47.1%)	9 (52.9%)	17(100%)
Coagulase –ve staph.	5(38.5%)	8(61.5%)	13(100%)
E.coli	5(50%)	5(50%)	10(100%)
Staph aureus	0 (0%)	6(100%)	6(100%)
Total	18(39.1%)	28 (60.9%)	46(100%)

Chi-Square =4.78 DF =3 P Value at 0.05 = 7.84 not significant

Table (6) Shows the distribution of study cases according to the W.B.C count before the use of hydrocortisone therapy. Most of the W.B.C. count were normal 14(60.9%)in those who received hydrocortisone therapy, while in those who had not received hydrocortisone most of cases have high W.B.C. count 19(82.6%).

Table(6) Distribution of study cases according to the W.B.C count before the use of hydrocortisone therapy.

Patients	W.B.C. count at day 0		Total
	Normal	High	
Group 1	14(60.9%)	9(39.1%)	23(100%)
Group 2	4(17.4%)	19(82.6%)	23(100%)
Total	18(39.1%)	28 (60.9%)	46(100%)

Table (7) Shows the distribution of study cases according to the W.B.C count in regard to the use of hydrocortisone therapy after 1wk.of treatment. All of high W.B.C return to normal count in those patients who not received hydrocortisone, while 4(22.2%) is still high in those who received hydrocortisone therapy.

Table (7) Distribution of study cases according to the W.B.C count after 1wk of hydrocortisone therapy.

Patients	W.B.C count at day 7		Total
	Normal	High	
Group 1	14(77.8%)	4(22.2%)	18 (42%)
Group 2	20(100%)	0(0%)	20(58%)
Total	34(89.5%)	4(10.5%)	38(100%)

Chi-Square =4.83 DF =1 P Value at 0.05 =3.84 significant

Discussion:

Regarding the age of onset, the study shows that the higher incidence of neonatal sepsis is in the very early onset followed by the late onset and early onset ,this goes with Wilson[9] and Greenough[1o] studies which shows that the high incidence of very early onset sepsis is 1 to 10 cases per 1000 live birth with amortality rate of 15 to 50%). This is may be due to presence of many risk factors for very early onset sepsis like preterm and LBW among the study cases.

The initial event of neonatal early-onset sepsis is supposed to occur prior to birth, since the majority of infected newborns present clinically as sepsis syndrome within the first 12h. of life. As a rule, early-onset sepsis results from an ascending infection of bacteria from the maternal recto vaginal flora invading the amniotic fluid and coming into contact primarily with mucosal cells of the fetal gastrointestinal and respiratory tract[11].

Males were predominantly affected by neonatal sepsis than females. This highly significant distribution is approved by Remington and Klien[13] who mentioned that male have approximately 2 fold higher incidence of sepsis than females, suggesting the possibility of sex- linked factor in host susceptibility to infection[11].

In this study neonatal sepsis cases were reported more frequently in premature than mature patients. Similar results was observed in Eisenfeld and Usmaniet studies who found that in preterm infants, chemotactic maturation begins after 2 to 3 weeks of life, proceeding slowly. In term infants, normal chemotactic function is established by the age of 2 weeks, whereas in preterm infants, chemotactic motility remains impaired for at least 3 weeks[12].Total neutrophil mass and the capacity to increase progenitor proliferation in preterm infants are even lower [12].

This is may be due to the fact that phagocytosis and microbicidal activity of phagocytes of healthy term newborn infants appear to be mature ,although in preterm infants and in septic or stressed infants, the neutrophil respiratory burst activity, phagocytosing capacity, or killing capacity are significantly depressed[13].

Low birth weight and very low birth weight are more prone to neonatal sepsis than normal birth weight this, goes with Stoll study which shows the incidence is increased ten fold in very low birth weight babies[14].

The incidence of sepsis is significantly higher in infants with very low birth weight (<1000 g), at 26 per 1000 live births, than in infants with a birth weight of 1000-2000 g, at 8-9 per 1000 live births. The risk for death or meningitis from sepsis is higher in infants with low birth weight than in full-term neonates. This is due to that LBW babies have depressed immunity system due to that most of LBW babies are either pre term or SGA or both[14].

The higher number of cases were born by normal vaginal delivery. This explain why most of study cases were occur in the very early onset sepsis because during the fetal life the fetal environment is normally sterile until the onset of labor and delivery. After rupture of the membranes, the infant becomes colonized with micro-organisms from the maternal genital tract[15]. This leads to ascending infection from the genital tract and neonatal colonization with bacteria.

This study shows the most common signs and symptoms of neonatal sepsis is poor feeding followed by RDS ,poor Moro reflex, pallor, hypothermia, lethargy, and cyanosis. This results nearly similar to Rodriguez (2003) study which show among the clinical signs and symptoms: poor feeding, lethargy, coffee ground vomiting, respiratory distress, signs of dehydration, hypothermia, pallor, cyanosis, apnea, mottled skin, sclerma & prolonged capillary refilling time, reported significant association with outcome of death in neonatal sepsis. This wide range of presentation may be due to that the sign and symptoms of neonatal sepsis are non specific and differ from patient to another. [16]

This study shows that group B. *streptococci* is the most common bacteria isolated from neonatal sepsis patients followed by coaglulase negative *staph*, *E.coli* and *staph aureus*. This goes with Kaftan (1998)study which shows that the micro-organisms recognized to have significant association with neonatal infections are group B. *streptococci*, coagulase negative *staphylococci*, group A *streptococci*, *Hemophilus influenzae*, and *E. coli*.[17].This may be due to presence of many risk factors for GBS neonatal sepsis among the study cases such as maternal intrapartam fever, preterm delivery, and preterm rupture of the membranes which enhance colonization of baby by GBS.

In Cordero study Gram negative microorganisms were the most common microorganisms isolated from those neonates with sepsis; especially Klebsiella pp. while low incidence of gp.B.hemolytic Streptococci was reported[18].Other study by VanAmerfoorts shows that (52,1%) gram-positive, (37.5%) gram-negative, (4.7%) polymicrobial, (4.6%) fungal, and (1.0%) anaerobic bacteria. Remarkably, gram-positive infections increased during the study period. This increase is attributed to increased nosocomial infections from such sources as catheterization and is particularly alarming considering that reported rates of methicillin-resistant Staphylococcus aureus isolates range from 29% to 45% and demonstrate an increasing trend [19].

This study shows that from total of 46 cases, the high W.B.C. count is found in 28 (6o.9%)and this is goes with Crain[20] and Dagan studies which shows that (50%) of infants had high WBC count ,and none had low count[21].This high percentage of high WBC count due to fact that in any newborn infant, PMN accumulate poorly at the sites of infection as a result of chemotactic deficiencies [22],and this lead to increase its count in the peripheral blood.

During sepsis, however, newborn infants frequently become neutropenic, Because of their limited neutrophil storage pools in the bone marrow and their inability to increase stem cell proliferation. In addition newborn infants born to mothers with hypertension have abnormally low blood neutrophil concentrations due to decreased neutrophil production [22].

Also this study goes with Munroe and Rod well study which showed impaired sensitivity of a single WBC count assay in neonatal sepsis as shows from 61cases 48(10%) with abnormal WBC count and 13(23%) with normal WBC count.White blood cell count, band form count and related ratios have served as diagnostic tools for neonatal infections. The specificity and sensitivity of these tests, however, are insufficient to serve as the only markers for sepsis [23,24].

After using hydrocortisone for 7 days, the study shows that, the high WBC count is more than the normal. The same results were found by Athens et al.,[25]and Nakagawa et al., [26] who showed that pharmacologic effects of GCs in humans are leukocytosis [27].This fact due to inhibition of leukocyte recruitment to inflammed areas retention of lymphocytes in the lymphatic circulation with shrinkage of peripheral lymph nodes, and promotion of microbial infection.

Glucocorticoids inhibit the release of inflammatory mediators and cytokines from alveolar macrophages in vitro. Systemic GC increases peripheral neutrophil counts and survival time by inhibiting their apoptosis [28].

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تاثير عقار الهايدروكوتزون على اعداد خلايا الدم البيض في متابعة الاطفال المصابين بانتان الدم الوليدي

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الملخص

ان انتان الدم الوليدي متلازمةُ سريريةُ تتميّز بالعديد مِنْ العلامات والأعراضِ التي تعتبر غير محدّدة للتشخيصِ. ان زرع الدم يعتبر اجرائا قياسيًا للتشخيص لكنه يحتاج وقتاً لإعْطاء نَتائِجه. تعداد الخلايا وحيدة النواة يستخدم الان للكشفِ المبكّرِ للمرض وايضا لمتابعة مرضى ًانتان الدم الوليدي. العلاج بالهايدروكورتيزونِ ما زالَ محل جدل ولسنوات عدّة كما هو مبين بالعديد مِنْ الدِراساتِ.تهدفَ هذه الدراسةِ لتقييمَ دورَ عقار الهايدروكورتيزون على اعداد خلايا الدم البيض في متابعة الاطفال المصابين بانتان الدم الوليدي. ان عدد الحالاتِ المرضية المدروسة كَان ٤٦ مريضا حديث الولادة شخصت اصابتهم بانتان الدم الوليدي استنادا لزرع الدم الموجب. كُلّ واحد منهم قُيّمَ سريرياً بواسطة استبيان تضمن تاريخ الحالة والتقييم السريري، وكذالك مختبريا باجراء فحوصات تَتضمّنُ تعدادَ خلايا الدم البيض عُمِلَ قبل العلاجِ بالهايدروكورتيزون لكُلّ الحالات. قسّمتْ الحالات إلى مجموعتين متساويتين احداهما أعطيتْ هايدروكورتيزون والمجموعةَ ألاخرى لم تعط هايدروكورتيزون. ثمانية أطفال رُضّع توفوا أثناء الإسبوعِ الأولِ و٣٨ حالةِ توبعتْ بعد اسبوع بنفس الفحص التي ذَكر اعلاه.كان انتان الدم الوليدي المتقدم جدا الحالة الاكثر شيوعا بين الحالات ٣٠ (٦٥,٢%) وكانت قلة الرضاعة العارض الاكثر شيوعا بين المرضى المصابين بالمرض ٤٠(٨٧%)،كذالك لوحظ ان المكورات السبحية المجموعة B عُزِلتْ في البدايةِ المبكّرةِ جداً في أكثر الحالاتِ المرضية ١٧(٣٧ %). و قبل العلاجِ بالهايدروكورتيزون لوحظ ان النسب المرتفعة في تعداد خلايا الدم البيض حيث كانت ٢٨(٦٠,٩ %). وبعد اسبوع من العلاج بالهادروكورتيزون لوحظَت نتائج مرتفعة في تعدادِ كريات الدم البيض في حالات ٤ (٢٢,٢%) من المجموعة الاولى ، بينما في المرضى الذين لم يعطو علاجِ الهايدروكورتيزون،كانت النتائج المرتفعة بعد إسبوعِ من المتابعة في إحصاءِ كريات الدم البيض (صفر). إنّ احصاء كريات الدم البيض يعتبرإختبارُ ومؤشرغير جيد في متابعة مرضى انتان الدم الوليدي.