RESULTS

Fifty newborns were enrolled this study . They were divided into 3 groups :-

Group 1 : The individual clinical data of group 1 infants shown in (table 1) . This group consists of 10 male and 10 female . The gestional age ranged from 37 wks to 40 wks with a mean of 38 . 60 wks . The mean age was 5. 05 days . Mean weight was 2.91Kg . Mean height was 49.60 cmm and Mean H.C was 35.00 cm .

- All infants had symptoms and Signs of sepsis including refusal to suck (17 cases) Hypothermia (5 cases) , Jundice (2 cases) cynosis (4 cases) . Sluggish moro reflex (17 cases)
- Postive blood culture for all infants . And the detected organism shown in (table 14) .

Group 2 : (Suspected group) include 20 infant (17 male and 3 female). The gestional age ranged from 38 wk to 41 wk .
(Mean = 39. 80 Wk) . The individual clinical data of group 2 infants shown in (table 2) .

Group 3 : (Control group) include 10 infant (6 male and 4 female) . The gestional age ranged from 38 to 41 wk (Mean 39.70 wks) . The individual clinical data of group 3 shown in (Table 3) .

- The three groups were compared as regard , age , gestional age and sex distribution shown in the (Tables 7 and 8) .
- As regard antental , natal, early post natal history, abnormal antenatal risk factors were found in 10 cases of Group1, 3 Cases of group 2 and no cases in group3 (Table 9) . Vaginal dilvery was the mode delivery in all group 3, and 2 C.S and 18 vaginal delivery in group2, and 7. C.S and 13 vaginal delivery in group1 (Table 9) .

- Comparing the three grops as regard to HC, weight and height shown in the (Table 10) .
- Comparing the three grops as regard to vital signs shown in the (Table 11) .

Results of laboratory investigations

Laboratory data of group (1) ..... Table 4

Laboratory data of group (2) ..... Table 5

Laboratory data of group (3) ..... Table 6

Mean ESR: - The Mean ESR in the septic group was 83.13± 16.36 and was significantly higher than suspected group 60.98 ±24.57 and the control group 5.55 ± 1.17. (Table 12) .

C. Reactive protein (CRP) :- In the septic group was + ve in all cases (100% + ve) and in the suspected group was - ve in all cases (100% - ve) and was - ve in all cases of control group (100% - ve). (Table 13) .

Haemoglobin (HB) : - In the septic group ; the Mean HB was 16.17 ± 1.77 and was significantly higher than suspected group 13.83 ± 2.74. But in the control group the mean HB was 17.30 ± 1.16 and was significantly higher than suspected group and septic group . ($P<0.05$) . And there was No significant difference in the septic VS. Control group . (Table 12) .

White blood cells (WBCS) : Mean WBCS in the septic group was 21.70 ± 2.45 and was not significantly higher than suspected group 21.30 ± 1.56 and significantly higher than the control group 13.35 ± 1.11 ($P<0.05$) . (Table 12) .

Interleukin - 1B . (IL-1B) : The means IL-1B plasma concentration was 27.66 pg/ml ± 24.21 and was significantly higher than suspected

group 6.65 ± 5.74 and control group 2.52 ± 2.23 . (P< 0.05) . (Table 15) .

Interleukin 1 receptor antagonist (IL-1ra): The mean IL-1ra plasma concentration the septic group was 4972.20 pg /ml ±1374.59 and was significantly higher than suspected group 2170.60 pg/ml ± 579.41 (P < 0.01) . (Table 16) .

- The Mean IL-1ra plasma concentration in the septic group was significantly higher than the control group 265.40 pg/ml ± 101.68 (P < 0.001)
- Mean IL-1ra plasma concentration in the suspected group was significantly higher than the control group (P < 0.001) .

Table (1) : Clinical data of group (1) (septic group)

Case Number	Sex	Age		Clinical picture (Symptoms & Signs)	Birth history			Feeding		Measurments						Systemic examination			
		Post natal (days)	Gestional (wks)		Ante natal	Natal	Post natal	Breast	Artifical	Vital			Physical			Chest	Heart	Abdomin	CNS
										T O C	HR/ Min.	RR/ Min.	Wt/ Gm.	Lt/ Cm.	HC/ Cm.				
1	F	4	39	Lethargy – Cynosis - Respiratory Distress (R.D) - Poor suckling .	Normal	-Premature Rupture of membrane C.S	-Delayed crying .	+	-	36.1	140	70	2500	50	34	Retaction Tachypnea Apnea	Free	Free	Sluggish Moro reflex (M.R)
2	M	6	38	R.D - Cynosis - Lethargy Poor suckling .	Antenatal fever	C.S	-Poor suckling . -Cynosis .	-	+	36	130	80	2800	50	34	Tachypnea	Free	Free	Normal
3	F	8	37	Apnea - Lethargy - Vomiting Cynosis - R.D - Poor suckling - Tachypnea .	Normal	Vaginal	Cynosis poor suckling .	-	+	36.7	150	80	2800	49	35	Retraction R.D. Apnea	Free	Distension	Sluggish M.R
4	M	7	39	R.D - Cynosis - Apnea - Jundice - Poor suckling .	Normal	Vaginal	Cynosis weak crying	-	+	36.8	175	90	2400	50	34	R.D. Apnea Retraction	Tachycardia	Free	Sluggish M.R
5	M	6	40	Poor suckling - Lethargy Resp. distress - Cynosis Apnea .	Normal	C.S	R.D. cynosis weak crying	+	-	37	180	70	3000	49	35	Apnea Retraction	Tachycardia	Free	Normal
6	M	5	39	R.D - Cynosis . Lethargy Poor suckling . Hypothermia Vomiting . Apnea	Normal	C.S	Poor suckling Apnea	-	+	35.3	140	70	2900	50	34	Retraction Tachy pnea	Free	Distension	Sluggish M.R
7	F	2	40	Poor suckling . Hypothermia Vomiting . Lethargy . Apnea Cynosis	Pyelo - nephritis	Vaginal	Cynosis Apnea Poor suckling	-	+	35.2	150	70	2800	49	34.5	Apnea Retraction Cripitation	Free	Free	Sluggish M.R
8	M	5	39	Poor suckling . Lethargy Bleeding from Mouth - Cynosis – Jundice .	Vaginal bleeding in 3rd Trimester fever	Vaginal	Post partum Hge Bleeding per mouth	-	+	36.2	160	65	2700	48	35	Tachypnea Retraction Apnea	Free	Free	Normal
9	M	3	37	Poor suckling - Lethargy Apnea - Cynosis - R.D.	Normal	Vaginal	Poor suckling Apnea cynosis	-	+	36.8	180	70	3150	50	35	Retractation Cripitation	Tachycardia	Free	Sluggish M.R
10	F	4	38	Poor suckling - Lethargy Hypothermia - Apnea Weak Crying - Bleeding	Normal	Vaginal	Poor suckling Cynosis	-	+	35.7	170	60	3300	50	34	Retraction Cripitation	Free	Free	Sluggish M.R

Table (1 cont.) : Clinical data of group (1) (septic group)

Case Number	Sex	Age		Symptoms & Signs	Birth history			Feedin		Measurments						Systemic examination			
		Post natal (days)	Gestional (wks)		Ante natal	Natal	Post natal	Breast	Artifical	Vital			Physical			Chest	Heart	Abdomin	CNS
										T O C	HR/ Min.	RR/ Min.	Wt/ Gm.	Lt/ Cm.	HC/ Cm.				
11	F	4	38	Bleeding per mouth. Poor suckling- R.D- Lethargy – Bleeding.	Fever with pregnany	Vaginal	Bleeding per mouth . Poor suckling .	+	-	36.9	180	70	3400	49	35	Retraction. cynosis.	Tachycardia	Free	Sluggish M.R
12	M	3	39	RD-Poor sckling Weak crying .	Pre-eclampsia	Vaginal	Muconium aspiration. Poor suckling .	-	+	37	180	75	3300	50	34	Retraction. Cripitation.	Tachycardia	Distension	Sluggish M.R
13	M	5	37	R.D-Weak crying- Poor suckling .	Pre-eclampsia	C.S	Cynosis . poor suckling.R.D	-	+	36.8	170	65	2700	49	35	Inter-costal retraction. Cripitation.	Free	Free	Sluggish M.R
14	F	4	38	Apnea - R.D - Crying Poor sucking .	Normal	Vaginal	R.D .Poor suckling.	-	+	36.5	165	70	2800	50	34	Cripitation. Retaction. Rhonchi.	Free	Free	Sluggish M.R
15	F	4	40	Poor suckling - Hypothermia R.D - Lethargy - Crying	Pre-eclampsia	Premature rupture. vaginal.	Poor suckling .	-	+	35.4	160	60	2800	50	35	Rhonchi. Cripitation. Retraction.	Free	Distension	Sluggish M.R
16	M	5	41	Poor suckling - Apnea-Res . Distress - Lethargy.	Normal	C.S	RD .poor suckling.	-	+	36	170	65	3000	50	34	Rhonchi. Cripitation. Retraction .	Free	Free	Normal
17	F	6	39	RD- Apnea - Lethargy Poor suckling.	Pre-eclampsia	Muconium stained Liquor. Vaginal.	Poor suckling. Apnea.	+	-	37	180	70	3100	50	35	Cripitation. retraction .	Free	Free	Normal
18	F	7	37	R.D . Vomiting - Poor suckling - Lethargy.	Pyelo-nephriits	Premature rupture. C.S	RD. Poor suckling	-	+	35.4	170	65	2400	49	34	Rhonchi-cripitation Retraction.	Tachycardia	Distension	Sluggish M.R
19	M	6	38	Vomitting frothy secretion Poor suckling - Disterss	Normal	Premature rupture vaginal	RD. Poor suckling.	-	+	35.5	170	70	3100	50	35	Cripitation. Retraction.	Tachycardia	Free	Normal
20	F	7	39	Poor suckling -R.D Hypothermia.	Drug intake	Premature rupture vaginal	Poor suckling. RD	-	+	36.6	180	75	2700	49	34	Cripitation. Retraction. Apnea.	Tachycardia	Free	Sluggish M.R

Table (2) : Clinical data of group (2) (suspected group)

Case Number	Sex	Age		Clinical picture (Symptoms & Signs)	Birth history			Feeding		Measurments						Systemic examination			
		Post natal (days)	Gestional (wks)		Ant natal	Natal	Post natal	Breast	Artifical	Vital			Physical			Chest	Heart	Abdomin	CNS
										T O C	HR/ Min.	RR/ Min.	Wt/ Gm.	Lt/ Cm.	HC/ Cm.				
1	M	5	40	RD -poor suckling - Cynosis grunting -weak crying .	Normal	Vaginal . twin	Poor suckling. Delayed Resp.	-	+	37.5	165	75	2800	49	34	Retraction Tachypnea	Tachycardia	Distension	Poor MR
2	F	6	40	Poor suck RD . Grunting cynosis -Jundice - Activity cynosis- RD	Normal	Vaginal twin	Delayed Resp weak. poor suck.	+	-	37.7	180	80	2700	50	34	Apnea retraction	Tachycardia	Free	Poor MR
3	M	9	41	poor suckling- cough- R.D	Normal	Vaginal	Normal	+	-	36.7	190	90	3000	50	35	Tachypnea Apnea	Tachycardia	Free	Abscent MR
4	M	6	40	Apnea- Bleeding Per mouth . cynosis.	Normal	Vaginal	Post par-tum Hge. delayed Resp.	-	+	36.6	130	95	3050	50	34.5	Tachypnea Apnea	Free	Free	Poor MR
5	M	4	39	RD- yellow discolouration- Apnic attack . Jundice- Pripheral cynosis .	Normal	Vaginal	Apnea Delayed Respiration. Cynosis.	+	-	37.1	160	70	2900	48	34	TachyPnea Retraction	free	Free	Poor MR
6	M	8	41	RD . slight cynosis poor suckling.	Normal	Vaginal	Delayed crying. slight cynosis.	+	-	37.2	170	65	3100	50	35	Cripitation Apnea	Tachycardia	Free	Poor MR
7	M	9	40	Dyspnea- cough -vomiting - slight cynosis- Apneic attack	Normal	Vaginal	Normal	+	-	37.6	170	85	3300	49	35	Tachy – pnea Rhonchi. Apnea.	Tachy-cardia	Distension	Poor MR
8	F	5	40	Poor suckling- Dyspnea cynosis - R.D.	Fever in 2nd Trimester	Vaginal	Delayed crying. cynosis .	+	-	36.8	160	65	2800	51	34	Tachypnea. Apnea.	Tachy-cardia	Free	Poor MR
9	M	8	40	Vomiting- poor suckling Tachypnea - Cynosis- RD	Normal	C.S	weak Crying. cynosis.	+	-	37.7	170	70	3200	50	34	Apnea. Tachypnea.	Tachy-cardia	Free	Poor MR
10	M	10	39	poor suckling - yellow colouration - R.D -pripheral cynosis.	Pyletitis in 2nd Trimester	Vaginal	Delayed crying. Cynosis.	+	-	37.5	170	80	300	50	35	Tachypnea. Apnea. Cripitation.	Tachy-cardia	Distension	Poor MR

Table (2 cont.) : Clinical data of group (2) (suspected group)

Case Number	Sex	Age		Clinical picture (Symptoms & signs)	Birth history			Feedin		Measurments						Systemic examination			
		Post natal (days)	Gestional (wks)		Ant natal	Natal	Post natal	Breast	Artifical	Vital			Physical			Chest	Heart	Abdomin	CNS
										T O C	HR/ Min.	RR/ Min.	Wt/ Gm.	Lt/ Cm.	HC/ Cm.				
11	M	5	40	Vomiting-Resp. Distress- poor suckling - Cynosis .	Normal	Vaginal	Cynosis. Delayed Crying .	+	-	37.5	170	60	3000	50	34	Tachypnea. Retraction .	Tachycardia	Distended	Poor M.R
12	M	10	41	Vomiting- fever poor suckling - cynosis -R.D.	Normal	Vaginal. Prolonged dilvery .	Delayed crying. cynosis.	+	-	38	170	90	2900	51	35	Tachypnea. Retractoin.	Tachycardia	Free	Good M.R
13	M	6	38	Vomiting- Apnea- Dyspnea refusal of feeding - R.D- Jundice cynosis .	Normal	Vaginal. prolonged labour.	Delayed weak cry. cynosis.	+	-	37.8	170	75	2700	49	34	Tachypnea	Tachycardia	Free	Sluggish M.R
14	M	5	39	Vomiting- R.D- poor suckling . cynosis.	Normal	Pro longcd labour. Vaginal.	Delayed crying. cynosis .	+	-	36.9	180	80	2900	50	33	Tachypnea. cripitation .	Tachycardia	Distended	Poor M.R
15	M	6	40	Dyspnea - weak cry- poor feeding- yellowish colouration .	Normal	C.S	Cynosis. Weak Delayed cry.	-	+	37.2	190	80	2700	51	35	Tachypnea Intercostal retiaction.	Tachycardia	Free	Sluggish M.R
16	F	9	40	Vomiting- fever- cough- Dyspnea . R.D- weak cry.	Fever in 2nd Trimester	Porlonged vaginal dilvery	Delayed cry. Cynosis. weak cry.	+	-	36.5	180	80	3100	50	34	Tachypnea	Tachycardia	Free	Poor M.R
17	M	8	40	Poor Feeding- fever- Dyspnea cough - R.D- cynosis.	Normal	Prolonged vaginal.	Delayed cry. cynosis	+	-	38	170	80	300	49	35	Tachypnea	Tachycardia	Distend	Sluggish M.R
18	M	1	40	R.D-Bleeding per mouth . weak cry. Cynosis .	Normal	Prolonged vaginal.	Delayed cry .R.D	+	-	36.2	160	70	3100	49	34	Tachypnea Aonea	Free	Free	Sluggish M.R
19	M	4	39	Vomiting- cough- Dyspnea - R.D weak cry -cynosis	Normal	Vaginal. prematture rupture	Delayed cry- cynosis- R.D	+	-	37.3	180	80	2800	48	34	Tachypnea grunting	Tachycardia	Free	Sluggish M.R
20	M	6	39	Poor suck. cough. vomiting . Pripheral cynosis-weak crying .	Normal	Vaginal. premature rupture.	Weal crying. pripheral cynosis.	+	-	37.8	185	70	2900	50	35	Tachypnea retraction Apnea	Tachycardia	distension	Sluggish M.R

Table (3) : Clinical data of group (3) (control group)

Case Number	Sex	Age		General examination	Birth history			Feeding		Measurments						Systemic examination			
		Post natal (days)	Gestional (wks)		Ante natal	Natal	Post natal	Breast	Artifical	Vital			Physical			Chest	Heart	Abdomin	CNS
										T O C	HR Min.	RR Min.	Wt Gm.	Lt Cm.	HC Cm.				
1	M	1	40	Pink . good crying . active . good suckling.	Normal	Vaginal	Normal	+	-	36.7	130	40	3100	50	35	Normal	Free	Free	Good M.R
2	M	2	39	Pink . good crying . active . good suckling.	Normal	Vaginal	Normal	+	-	37.1	120	39	2800	51	34.5	Free	Free	Free	Good M.R
3	F	3	40	Pink . good crying . active . good suckling.	Normal	Vaginal	Normal	+	-	37	130	40	300	50	36	Free	Free	Free	Good M.R
4	F	4	38	Pink . good crying . active good suckling.	Normal	Vaginal	Normal	+	-	36.9	125	44	3200	50	35	Free	Free	Free	Good M.R
5	M	1	39	Pink . good crying . active . good suckling.	Normal	Vaginal	Normal	+	-	37.2	130	42	2900	49	35	Free	Free	Free	Good M.R
6	F	2	41	Pink . good crying . active . good suckling.	Normal	Vaginal	Normal	+	-	37	130	40	2800	51	34	Free	Free	Free	Good M.R
7	M	3	40	Pink . good crying . active . good suckling.	Normal	Vaginal	Normal	+	-	37.2	135	39	3000	50	36	Free	Free	Free	Good M.D
8	F	2	40	Pink . good crying . active . good suckling.	Normal	Vaginal	Normal	+	-	37	140	38	3200	51	35	Free	Free	Free	Good M.R
9	M	2	41	Pink . good crying . active . good suckling.	Normal	Vaginal	Normal	+	-	36.6	125	40	2900	50	34	Free	Free	Free	Good M.R
10	M	1	39	Pink . good crying . active . good suckling .	Normal	Vaginal	Normal	+	-	37.1	130	41	3200	51	36	Free	Free	Free	Good M.R

Table (4) : Laboratory data of group (1) (septic group)

Number	Laboratory findings												IL-1ra (Pg/ml)	IL-1B (Pg/ml)	Organism
	Bl. culture	ESR		C R P	C.B.C										
		1st H	2nd H		HB% (g)	RB cs M/cmm^3	W.B.C								
							Count	B%	E%	Seg%	L%	M%			
1	+Ve	70	150	+Ve	17	5.5M	24000	0.4	3%	73	23	6%	4230	1.2	E.coli
2	+Ve	60	110	+Ve	16.1	5.3M	22000	0.5	4%	71	20	7	5054	77.2	Klebsiella
3	+Ve	60	120	+Ve	15	5.1M	13000	0.3	3	72	23	5	3020	30.8	Coagulase -ve staph.
4	+Ve	70	130	+Ve	16	5.6M	20000	0.5	5%	74	22	8	2875	165.9	Enterococci
5	+Ve	80	140	+Ve	18	6.4M	25000	0.4	4%	76	23	4	5628	90.8	L.Monocytogens
6	+Ve	50	120	+Ve	19.1	6.3M	23000	0.3	3	75	24	3	7530	2.2	streptopyrogens
7	+Ve	40	110	+Ve	16.1	5.4M	23000	0.3	4	76	22	5	6653	42.6	E.coli
8	+Ve	60	130	+Ve	15.4	5.4M	22000	0.6	2	73	21	6	6500	61.1	Staph.aureus
9	+Ve	70	140	+Ve	12.8	4.2M	23000	0.5	3	75	22	7	4641	7.1	Pseudomonas aeurginosa
10	+Ve	50	110	+Ve	15.3	5.1M	22000	0.5	4	72	23	5	4232	3.3	staph.aureus
11	+Ve	40	90	+ve	13.7	4.4M	20000	0.5	5%	76	23	5	6850	18.6	E-Coli
12	+Ve	50	120	+ve	14.5	4.9M	22000	0.5	4	71	22	6	5010	12.06	Staph-aureus
13	+Ve	40	80	+ve	17.2	5.7M	23000	0.5	3	75	24	2	3250	0.52	Klebsiella
14	+Ve	60	110	+ve	16.7	5.4M	22000	0.4	2	67	23	5	4841	7.8	E-Coli
15	+Ve	50	100	+ve	189	6.2M	23000	0.4	4	74	24	4	6845	2.6	Enterococci
16	+Ve	40	80	+ve	13.5	3.8M	22000	0.2	3	75	23	3	5243	7.5	Streptopyogens
17	+Ve	50	110	+ve	15.2	5.1M	20000	0.3	2	77	22	2	4712	2.98	E-coli
18	+Ve	60	130	+ve	18.8	6.2M	22000	0.5	3	75	20	3	5125	0.175	Streptopyogens
19	+Ve	40	90	+ve	17	5.5M	23000	0.4	5	72	22	4	3150	3.6	L-Monocytogens
20	+Ve	35	80	+ve	18	5.3M	20000	0.5	4	73	23	3	4055	15.1	Coagulase-ve staph .

Table (5) : Laboratory data of group (2) (suspected group)

Number	Blood culture	ESR		CRP	C.B.C								IL-1ra (Pg/ml)	IL-1B (Pg/ml)
		1st H	2nd H		HB% (g)	RB Cs M/cmm^3	W.B.C Count	B%	E%	Seg%	L%	M%		
1	-Ve	20	35	+ve	11	4.5M	21000	0.5	3	70	25	5	1520	2.2
2	-Ve	20	38	+ve	12	4.6M	20000	0.5	3	70	25	7	2108	6.4
3	-Ve	20	40	+ve	10	5M	19000	0	3	75	20	3	2050	-
4	-Ve	25	55	+ve	9	3.5M	22000	0.5	2	70	25	3	1814	-
5	-Ve	30	60	+ve	10	5.5M	23000	0.2	2	70	25	3	3250	18.2
6	-Ve	33	60	+ve	15	5M	20000	0.3	3	69	27	4	2112	2.6
7	-Ve	35	65	+ve	15.5	5.5M	23000	0.5	2	70	25	3	2620	0.75
8	-Ve	50	90	+ve	12	5.5M	24000	0.5	2	75	22	3	3204	2.2
9	-Ve	60	130	+ve	13	5M	21000	1	1	75	23	4	2352	3.6
10	-Ve	70	120	+ve	11	4.5M	20000	1.5	1	72	23	5	1651	2.6
11	-Ve	28	60	+ve	13	5M	1900	0.5	2	70	23	4	2417	15.1
12	-Ve	48	100	+ve	16	5.6M	22000	0	1	75	25	2	2529	6.4
13	-Ve	24	45	+ve	16	4.5M	21000	0.5	2	70	23	4	2300	16.9
14	-Ve	50	95	+ve	17	5.3M	20000	0.3	2	72	24	5	2080	9.9
15	-Ve	45	85	+ve	15	4M	22000	0.2	1	75	26	4	1156	4.0
16	-Ve	40	80	+ve	17	5.5M	20000	1	1	75	22	4	2355	10.6
17	-Ve	33	65	+ve	17	5M	22000	0.5	1	70	26	4	2756	2.9
18	-Ve	40	80	+ve	131	4M	23000	0.5	6	70	22	4	1250	5.4
19	-Ve	60	115	+ve	17	5.5M	20000	0.2	4	72	22	4	1445	14.1
20	-Ve	70	170	+ve	17	5.3	24000	0.5	2	72	23	6	2443	9.2

Table (6) : Laboratory data of group (3) (control group)

Number	Laboratory findings													
	Bl. Culture	ESR		C R P	C.B.C								IL-1ra (Pg/ml)	IL-1B (Pg/ml)
		1st H	2nd H		HB% (g)	RB cs	W.B.C							
							Count	B%	E%	Seg%	L%	M%		
1	-Ve	2	6	-ve	17.9	5.5M	12000	0.5	2	60	30	6	376	3.6
2	-Ve	3	7	-ve	18.9	5.3M	14000	0.5	2.5	63	29	7	463	0.175
3	-Ve	5	10	-ve	16.9	5.6M	13000	0.2	2	55	30	5	334	1.57
4	-Ve	2	8	-ve	16.9	6M	15000	0.5	2	62	25	6	212	—
5	-Ve	3	7	-ve	19.9	5.4M	14000	0.2	3	59	30	5	184	5.08
6	-Ve	5	9	-ve	18.9	5.6M	12000	5	2	61	30	70	275	3.6
7	-Ve	5	6	-ve	16.9	6.1M	14500	0.5	2.5	64	25	71	168	1.22
8	-Ve	3	9	-ve	17.9	5M	13000	0.3	3	61	30	6	285	2.9
9	-Ve	5	10	-ve	19.9	5.4M	12000	0	2	60	26	5	214	6.8
10	-Ve	2	7	-ve	16.9	5.1M	14000	0.5	2.5	55	23	5	143	0.8

Table7 : Comparison between studied groups as regards age & gestational age .

Variable	Septic n = 20		Suspected n =20		Control n=10		t1	t2	t3
	Mean	±SD	Mean	±SD	Mean	±SD			
Age in days	5.05	1.57	6.30	2.36	5.10	0.99	1.97	0.89	1.88
Gestational age in wk.	38.60	1.14	39.80	0.77	39.70	0.95	3.90 *	2.62 *	0.31

t1 Septic vs . Suspected

t2 Septic vs . Control

t3 Suspected vs . Control

* $P < 0.05$

Table8 : Sex distribution in the studied groups .

	Septic		*Suspected*		*Control*	
	No	%	No	%	No	%
M	10	50.0	17	85.0	6	60.0
F	10	50.0	3	15.0	4	40.0
Total	20	100.0	20	100.0	10	100.0

$X^2_2 = 5.66$ $p > 0.05$

Table9 :Comparison of the studied cases according to medical history.

	Septic *n = 20*		*Suspected* *n =20*		*Control* *n =10*		χ^2	*P*
	No.	%	No.	%	No.	%		
Antenatal								
Normal	10	50.0	17	85.0	10.0	100	10.76	<0.01*
Abnormal	10	50.0	3	15.0	0.0	0.0		
Natal								
Vaginal	13	65.0	18	90.0	10	100	6.98	<0.05*
CS	7	35.0	2	10.0	0.0	0.0		
Postnatal								
Normal	0	0.0	2	10.0	10	100	40.13	<0.001*
Abnormal	20	100	18	90.0	0.0	0.0		
Feeding								
Breast	4	20.0	17	85.0	10	100	25.59	<0.001*
Artificial	16	80.0	3	15.0	0.0	0.0		

Table10 : Distribution between studied groups as regards length , weight and HC

Variable	*Septic* *n=20*		*Suspected* *n =20*		*Control* *n=10*		*F*	P
	Mean	+SD	Mean	±SD	Mean	±SD		
Length (Cm)	49.60	0.68	49.70	0.87	50.30	0.68	2.99	>0.05
Weight(Kg)	2.91	0.26	2.95	0.17	3.01	0.16	0.77	>0.05
HC (Cm)	35.00	2.41	34.38	0.58	35.05	0.76	0.94	> 0.05

Table 11 : Distribution between studied groups as regards vital signs

Variable	*Septic n=20*		*Suspected n =20*		*Control n=10*		*t1*	*t2*	*t3*
	Mean	±SD	Mean	±SD	Mean	±SD			
HR	165.00	15.48	171.00	13.24	134.50	16.91	1.32	4.94 *	6.49
RR	70.50	7.05	77.00	40.30	40.30	1.70	2.53 *	18.12*	17.45*
Temperature	36.25	0.64	37.33	36.98	36.98	0.20	5.86 *	4.68 *	2.64*

t1 Septic vs . Suspected

t2 Septic vs . Control

t3 Suspected vs . Control

* $P < 0.05$

Table12 : Comparison between studied groups as regards ESR , CBC results.

Variable	*Septic n=20*		*Suspected n =20*		*Control n=10*		*t1*	*t2*	*t3*
	Mean	±SD	Mean	±SD	Mean	±SD			
HB	16.17	1.77	13.83	2.74	17.30	1.16	3.22*	1.83	4.87*
RBCs	5.34	0.68	4.92	0.60	5.50	0.35	2.10*	0.70	2.84*
ESR(mean)	83.13	16.36	60.98	24.57	5.55	1.17	3.36***	21.10**	10.07*
WBCs	21.70	2.45	21.30	1.56	13.35	1.11	0.62	10.19*	14.36*
Segmented	74.05	1.82	71.85	2.28	60.00	3.02	3.37*	15.94*	12.05*

t1 Septic vs . Suspected

t2 Septic vs . Control

t3 Suspected vs . Control.

* P < 0.05

** P < 0.01

*** P <0.001

Table13 :Comparison between studied groups as regards CRP.

	Septic		*Suspected*		*Control*	
	NO.	%	No.	%	No	%
+ve	20	100.0	20	100.0	0	0.0
-ve	0	0.0	0	0.0	10	100.0
Total	20	100.0	20	100.0	10	100.0

X^2_2 = 50.00 * P < 0.001

Tabel 14 : Distribution of the blood + ve culture neonates according to type of organisms .

Septicemic neonates / Blood culture	Septicemic + Ve Blood cluture	
	No	%
Escherechia coli	5	25.0
Staphylococcus aureus.	3	15.0
Coagulase - ve staphylococcus.	2	10.0
Streptococcus pyogens.	3	15.0
Klebsiella	2	10.0
Enterococci	2	10.0
Pseudomonas aeruginosa	1	5.0
Listeria monocytogens	2	10.0

Table15 : - Comparison between studied groups as regards IL-1B .

grougs	*mean ± SD*	*t*	*P*
Septic vs . Suspected	27.66 ±42.21 6.65 ±5.74	2.20	<0.05
Septic Vs. Control	27.66 ± 42.21 2.52 ±2.23	2.66	<0.05
Suspected Vs. Control	6.65 ± 5.74 2.52 ±2.23	2.82	< 0.01

Septic	vs. Suspected	CI 1.13 - 40.88
Septic	vs. Control	CI 5.32- 44.95
Suspected	vs. Control	CI 1.12-7.13

Table16 : Comparison between studied groups as regards IL-1 Ra .

Groups	*mean ± SD*	*t*	*P*
Septic Vs. Suspected.	4972.20 ± 1374.59 2170.60 ± 579.41	8.40	< 0.01
Septic vs control	4972.20 ± 1374.59 265.40 ± 101.68	15.23	> 0.001
Suspected vs. control	2170.60 ± 579.41 265.40 ± 101.68	14.27	< 0.001

Septic	Vs. Suspected	* CI	2116-3487
Septic	Vs. Control	* CI	4060-5354
Suspected	Vs. Control	* CI	1628-2183

Table17 : Correlation between IL-1receptor antagonist and other variables in septic group .

Variable	*r*	*P*
Age	-0.475	<0.05*
ESR (mean)	-0.008	>0.05
Gestional .age	0.443	<0.05 *
HB	0.104	>0.05
RBCs	0.137	>0.05
WBCs	0.361	>0.05
IL receptor	-0.143	>0.05
Seg .	0.360	>0.05

Table18: Evaluation of IL-1RA in prediction of sepsis .

IL- 1RA	Septic group	%	Suspected group	%	Total
> 3000	19	95.0	2	10.0	21
<3000	1	5.0	18	90.0	19
Total	20	100	20	100	40

*** Blood culture is considered the reference test for sepsis .**

Sensitivity = 95.0%

Specificity = 90.0%

Positive predective value = 90. 5%

Negative predective value = 94.7%

Diagnositic efficiency = 92.5%

Figure 1 : Distribution of the blood + ve culture neonates according to type of organisms .

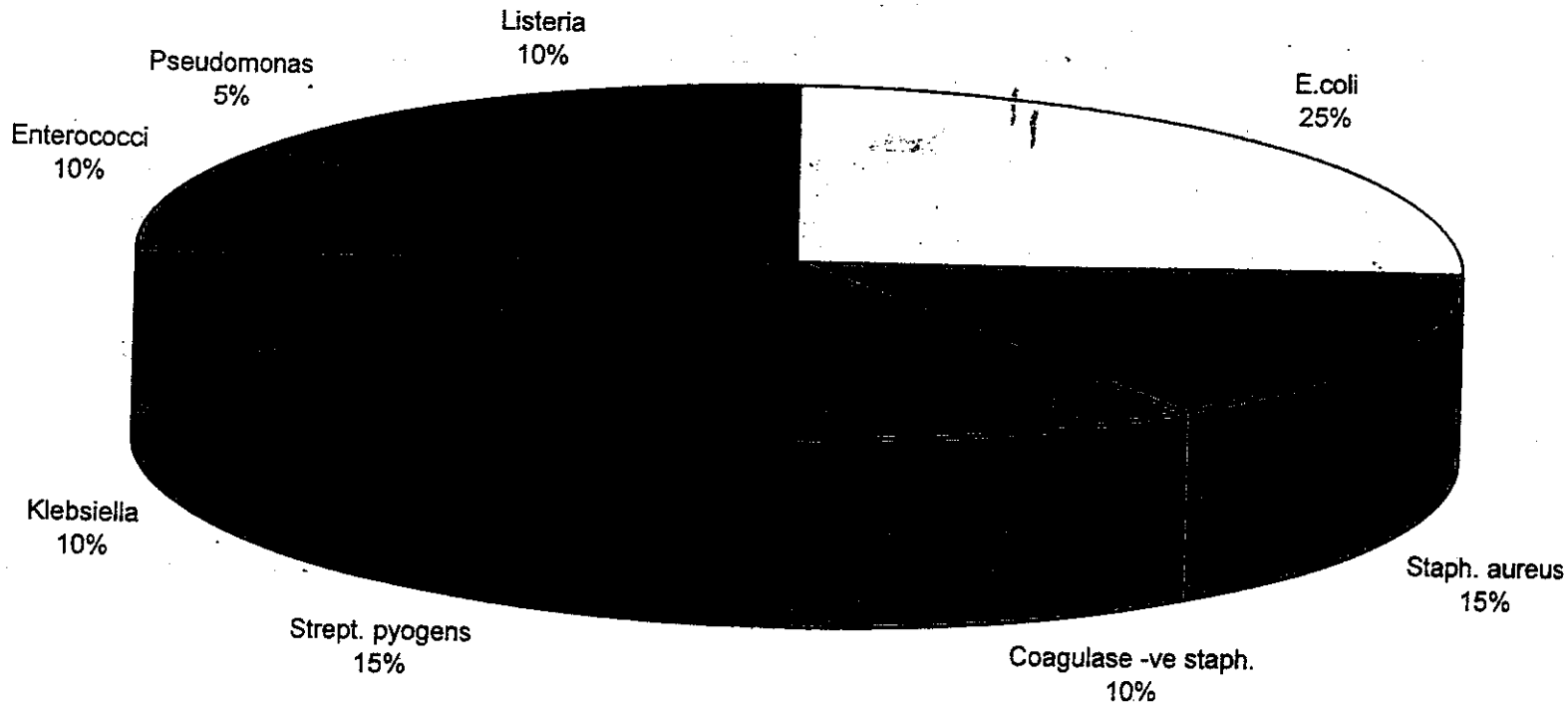


Figure 2 : Comparison between Studied groups as regards IL-1B .

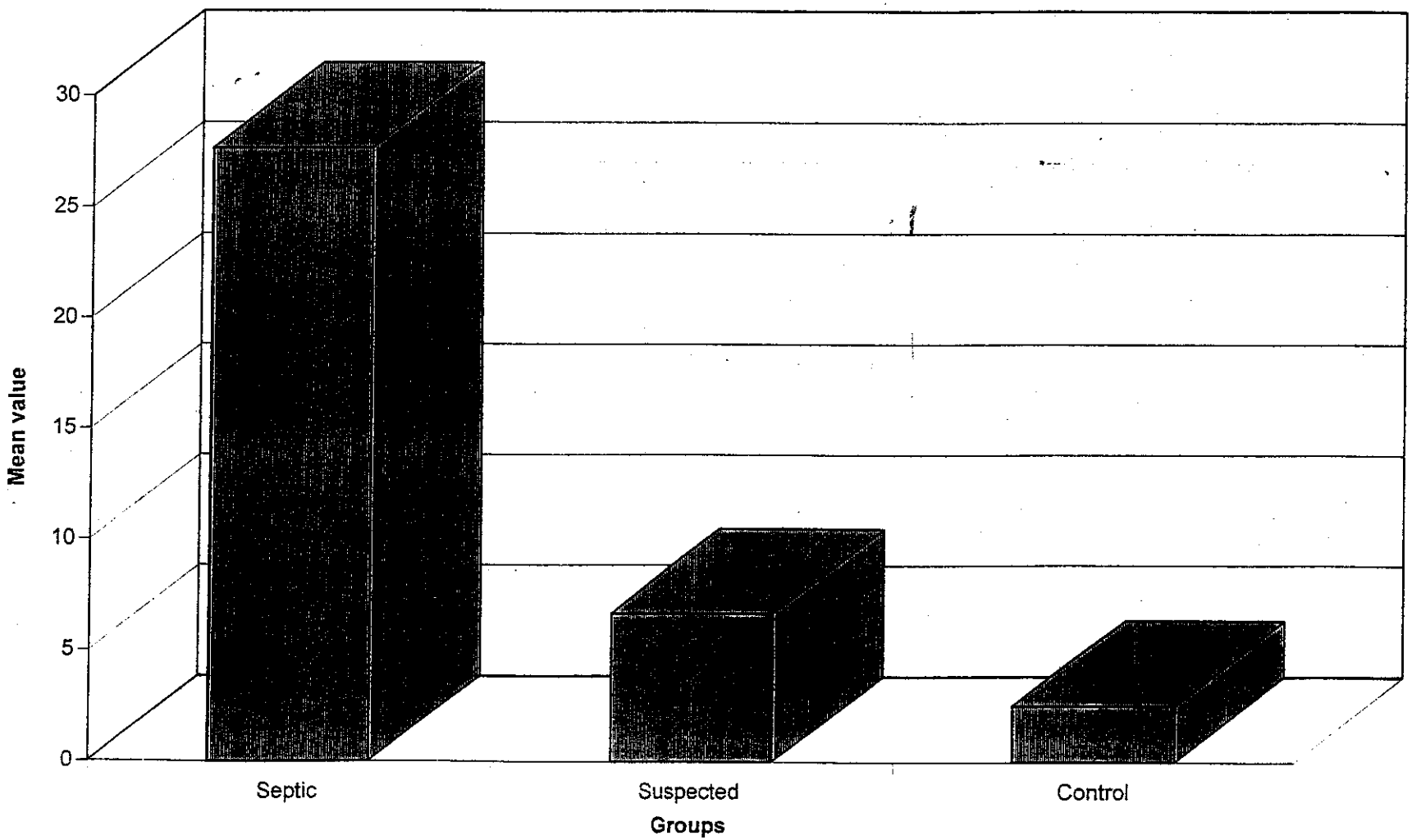
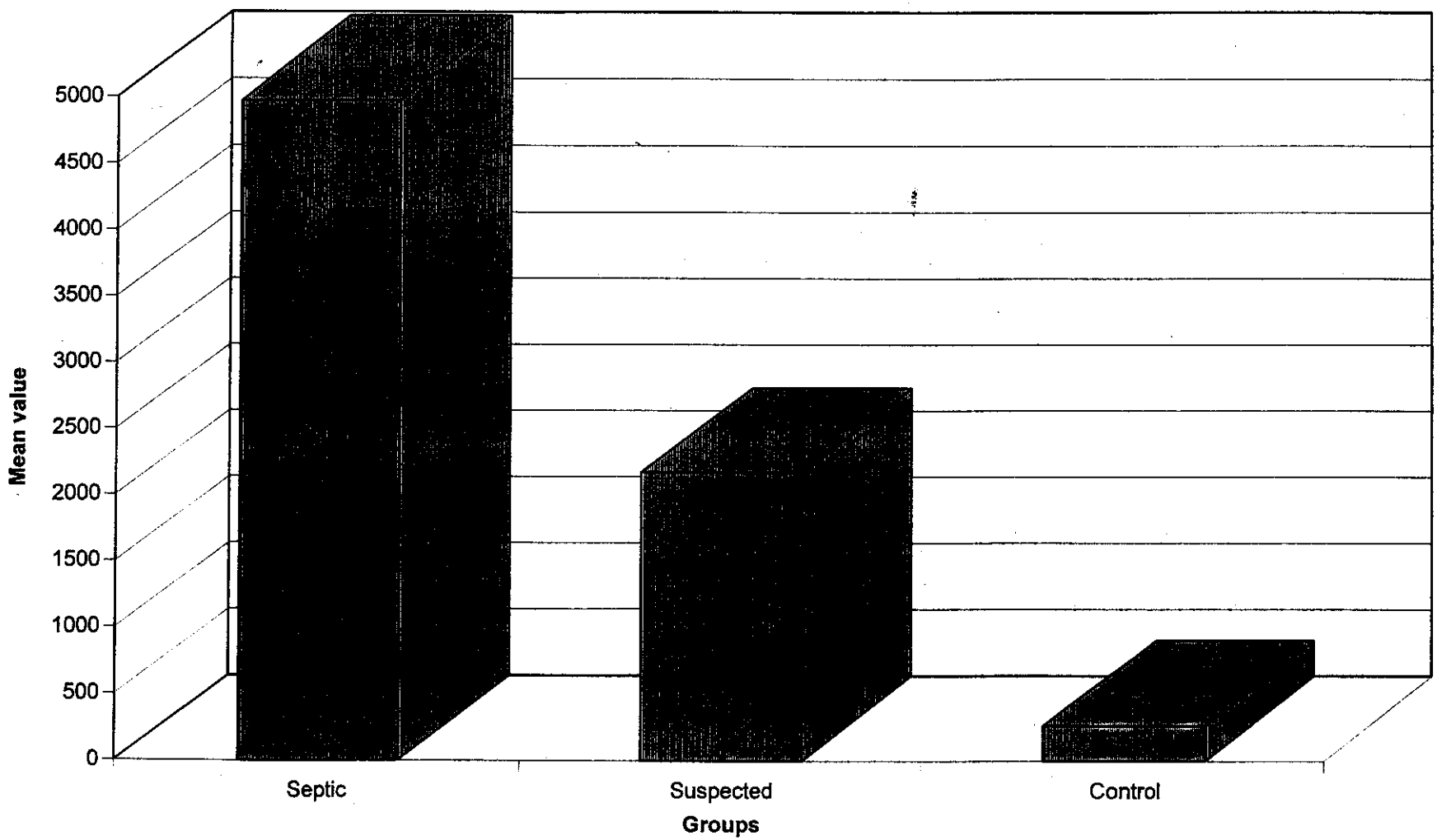
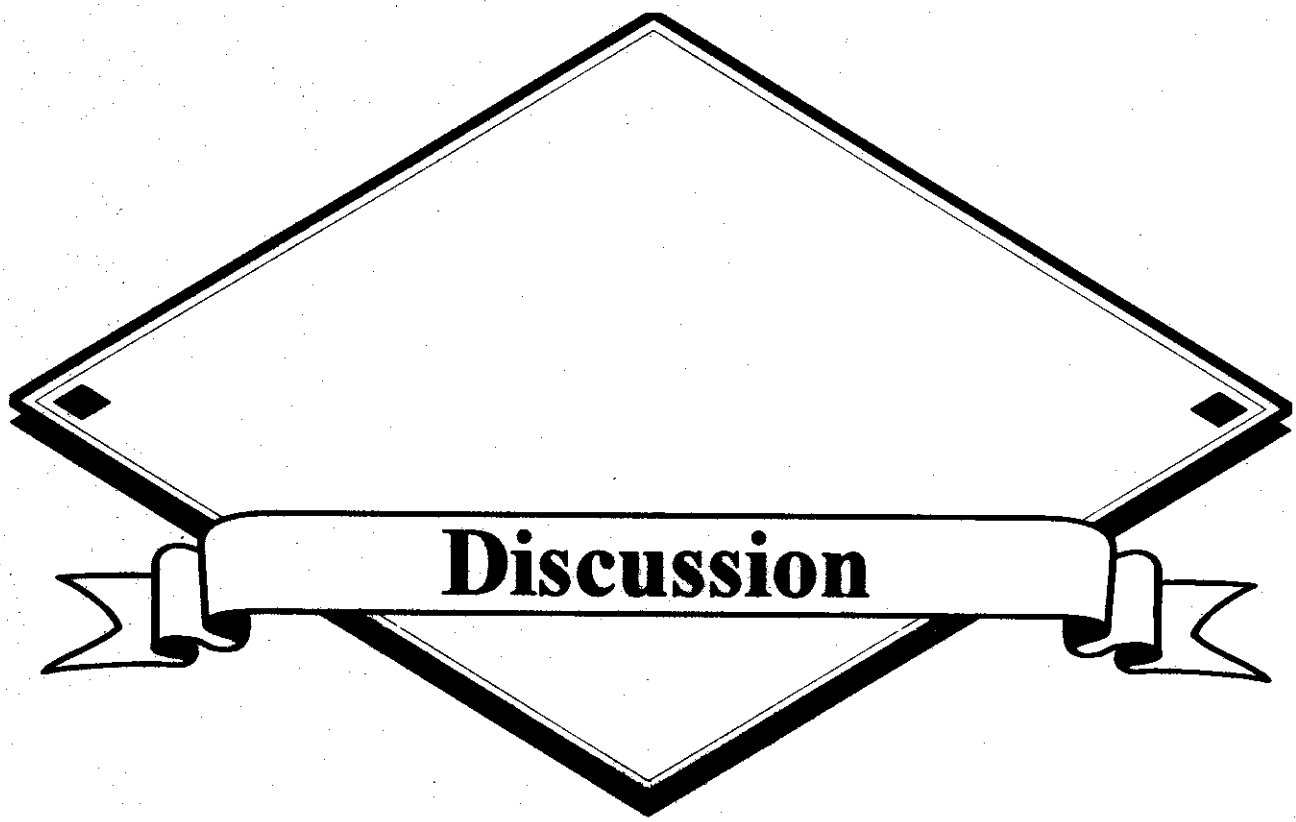


Figure 3 : Comparison between Studied groups as regards IL-1ra .




Discussion

DISCUSSION

The newborns particularly the preterm infants are immunocompromised in several ways, which predispose them to infection (**Hague, 1992**) .The incidence of neonatal sepsis ranged from 1-10 per 1000 live birth (**Gotoff, 1996**) . The high incidence may be due to low socioeconomic standard and most of the patients have one or more risk factors to develop septicemia . Early clinical diagnosis of neonatal infection is difficult because of non specific presentation of infection, and adelay in diagnosis may be associated with increase morbidity and mortaliy (**Russel et al., 1992**) .

Although the high incidence of bacterial infection in the newborns appears to be multifactorial, one of the most important deficits in the neonatal immune system is the quantitative deficiency in the myeloid and phagocytic system . Additionally, despite normal circulating numbers of mature effector phagocytes, the presence of serious in vitro neutrophil abnormalities may still exist and predispose the neonate to an impaired immune response during over whelming bacterial infection **(Cairo, 1991)** .Cytokines including colony stimulating factors, tumor necrosis factor and many interleukines mediate the complex series of responses to infection **(Dinarello, 1991)** . The present study carried out on (50) neonates including (20) septicemic with postive blood culture, (20) infants with same clinical manifestations of sepsis but with negative blood culture (suspected group) and (10) healthy control neonates The mean body weight of septicemic group of infants was **(29100 $\pm$ 0.26)** gm, in suspected group it was **(2950 $\pm$ 0.17)** gm and in

control group was (**3010 ± 0.16**) gm with significant difference between the 3 groups (**p< 0.05**) .

Bhakoo and singh; (1988); found that the incidence of neonatal sepsis was higher among lower birth weight infants .

We found that (13) septic neonates (65%) delivered vaginally and (7) septic neonates (35%) by C.S but (18) suspected neonates **(90%)** delivered vaginally and (2) suspected neonates **(10%)** by C.S without significant difference from the control neonates . A similar finding was recorded by **EL. Hefney et al, (1986).** Who found that out of 25 septicemic cases 22 **(80%)** were delivered vaginally and 3 cases **(12%)** by C.S .

Most of our septicemic babies were presented by poor feeding **(85%)** followed by respiratory distress **(75%),** poor reflexes **(70%),** Lethargy (**70%),** cyanosis and apnea**(45%)** and hypothermia **(25%)** and Jundice **(10%)** .

In other study carried out by **soliman et al., (1993**). In pediatric department of AL. Hussain university hospital, they found that septicemic presentations were poor feeding **(58.5%),** Lelhargy (48%), apnea **(40%),** Hypothermia and fever **(32.5%)** each, hepatomegally **(25%),** Jundice **(18%),** sclerema and petichea **(45%)** each .

Baker (1994) showed that septicemic presentations were feeding difficulties **(57.5%)** , hepatomegally **(40%)** respiratory distress **(25%),** splenomegally and Jaundice **(25%)** each, diarrhea and shock **(22.5%)** each, poor reflexes, lethargy and vomiting **(20%)** each, oliguria, hypothermia and fever **(15%)** each , convulsions **(10%),** abdominal distension, dehydratron and cynosis were **(7.5%)** each .

EL.Naggar; (1995) found that the common clinical manifestatrons of septicemic full terms were, poor feeding **(84%),** lethargy **(64%),** respiratory distress **(46%)** and abdominal distension **(40%);** the less common presentations were scleroderma, irritability, seizures and vomiting .

Studies by **El- Sallab et al; (1987) and Al-Saleh et al., (1984)** found more or less similar clinical findings .

Regarding risk factors ; The present study was carried out on (40) septicemic neonates . In septicemic + ve culture neonates **(25%)** suffered from prolonged labour, premature rupture of membrane (PROM) in **(25%)** , Meconium aspiration in (**15%),** urinary tract infection (UTI) in **(10%),** and intrapartum fever in (**5%),** and no risk factor in **(40%)** of cases . These results are in agreement with the study done by **kawamura et al; 1995** which included the same risk factor .

In the present study; All cases of septic group (100%) showed postive C.R.P, all cases of suspected group showed postive C.R.P and the control group (100%) showed negative CRP with a significant difference between each of septic and suspected groups and control group **(P < 0.001)** .

Infection activates the acute phase response which may be detected by alternations in the peripheral blood neutrophil and an increase in serum proteins such as CRP. Although such changes are non specific, they are thought to give indirect evidence of infection **(Russel et al; 1992) . Alt et al., (1982)** found with CRP **(48.2%)** false negative results in the first 12 hours of life . **kushner et al; (1973)** claimed that an increased level of CRP without infection can be seen in cases with

PROM, maternal fever during labour and /or perinatal asphyxia . **Aiebender et al., (1982)** added fetal distress, shock and /or Meconium aspiration as other causes of rise in CRP. On the other hand, some authors pointed out false negative results of CRP in cases of early streptococcus B infections (**Sonn et al., 1984**) . This is a major drawback since group B streptococcus is frequently present in maternofetal infections and the mortality in these streptococcal infections was negative (**philip and Hewitt, 1980**) .

In the present study the mean of RBCs count and HB were significantly lower among suspected group than both septic and control groups (**P < 0.05**) . This findings did not agree with that of **Feigin et al; (1987)** who showed that lower level of HB and RBCs in neonatal sepsis are secondary to hemolytic process in sepsis .

In the present study the mean ESR was significantly higher in septic group than the suspected group (**P < 0.05**) and control group (**P < 0.001**) . The mean ESR was significantly higher in the suspected group than in the control group (**P < 0.01**) .

In the present study; Septic group as suspected group showed significant higher WBCs count than control group (**P < 0.05**) .
Some authors considered the change in leucocyic count is useful in direct indication of bacterial infection (**Avery ; 1987, Benuck and David, 1983).** On the other, **Rozychietal, (1987)** and **christensen (1987)** warned that a nomal blood leucocytic count should not exclude diagnosis of systemic bacterial infection and that antimicrobial treatment should not be withheld from ill neonates on the basis of normal neutrophil count or morphology.

Rozychi et al., (1987) Found that the tolal WBCs count was of poor predictive value in neonatal sepsis as a wide variation exist in the tolal WBCs count in neonatal peroid . **Manroe el al., (1979)** found that maternal and prenatal factors, other than Sepsis could influence the total WBCs count . Change in morphology of neutrophils such as vacuolization and toxic granulation also suggest the presence of infection **(Lio et al., 1984) .**

The Caustive organisms of neonatal sepsis vary form nursery to nursery between different geographical area and in the same area with time Hospital to hospital variability in incidence may be related to sate of prematurity, prenatal care, conduct of labour and enviromental condition in nurseries **(Ohlasson and vemcombe, 1987) .**

In our study, in the Septicemic + ve culture, the most prevalent Caustive oraganisms were E.coli **(25%),** staph aureus **(15%),** streptococus pyogens **(15%),** coagulase-ve staphylococcus **(10%),** Klebsiella **(10%),** Enterococci **(10%),** Listeria monocytogens **(10%)** and pseudomonas aeroginosa **(5%)** .

Vesikari et al; (1989) in his study performed from 1981- 1985 to difine the caustive organisms in neonatal sepsis found that GBS, staph aureus and E. Coli were the predominant organisms causing **(29%), (25%) , (24%)** of cases respectively .

Rodwell et al, (1993) found that GBS was the most predominant organism Causing **(50%)** of Cases, while E.Coli Causes **(25%) ,** stephylococci Causes **(12%)** and hemophilus influenza Causes **(6.2%)** of all cll cases .

In Tanta faculty of medicne NICU, Shohaib et al ; (1989) found that the staphylococci and E.coli were the most prevelant organism causing neonatal septicemia .

In our study we found that the mean plasma concentration of IL-1ra in the septic group was significantly higher than the suspected group **(p< 0.001)** and control group **(P<0.001)**. Suspected group was significantly higher than control group **(p<0.001)**.

These results are in agreement with that of **De Bont, et al;(1995);** who reported that mean plasma concentration of IL-1ra in the septic group was significantly higher than both the suspected **(P<0.01)** and control **(p<0.01)** groups . Suspected group was significantly higher than control group **(p<0.01)** .

Geiger R et al; (1996); found that neonates with severe illness (Septicemia, asphyxia, neonatal respiratory distress syndrome), who received invasive intensive care, circulating IL-1ra levels were significantly higher than in the reference group of healthy neoborns .

In our study, in the septic group the senstivity of IL-1ra was **(95%)** and specificity was **(90.0%)** with a positive predictive value of **(40.5%)** and a negative predictive value of **(44.7%)** and Diagnostic efficiency **(42.7%)** .

Also; In our study; in the septic group there was insignificant postive correlation between IL-1ra and Mean ESR, HB, RBCS, WBCS and segmented and there was significant postive correlation between IL-1ra and age and gestional age **(P < 0.05)**.

In the septic group the mean plasma concentration of IL-1B was significantly higher than both suspected group **(P<0.05)** and control group **(P < 0.01)** .

These results are in agreement with that of **De Bont et al; (1995)**; Mean plasma concentration of IL-1B was significantly higher in the septic neonates than suspected group (**P < 0.05**) and control group .

Also; they also found that IL-1 plasma concentrations of IL-1ra was 50 - 100 fold Higher **(De Bont et al; 1995)** .

In our study we found that IL-1ra plasma concentrations was 180 fold Higher than IL-1B in the septic group .

Granowitz (1991); shows that newborns with infections, bacteremia and /or sepsis have elevated interleukin - 1receptor antagonist (IL-1ra) plasma concentrafion as high as adult during sepsis .

Interleukin-1 receptor antagonist (IL- 1ra) is a natural occuring inhibitor that blocks the action of interleukin 1 (IL-1) by competitive binding to its receptor . IL-1ra can block E.coli induced shock in rabbits and baboons when given 15 minutes before bacterial infusion **(Wakabuyashi; et al 1991)** .

In addition the IL-1ra infused in baboons attenuated the sustained IL- 1B response **(Fischer E; et al 1992)** .

During in vivo studies with human mononuclear cells, IL-1ra given 30 minutes . before stimulation inhibits IL-1 induced IL-1, TNF and IL.6 prduction in a dose dependant manner as well as endotoxin induced cytokine synthesis (**Granowitz; 1992)** .

Anderson et al; (1992) demonstrated that the peak of IL-1ra production by peripheral blood mononuclear cells was at 4th hour after stimulation . Monocytes can produce IL-1B and IL-1ra at the same time .