DIAGNOSTIC AND PROGNOSTIC ROLE OF CRP AND m-ESR IN NEONATAL SEPTICEMIA

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ABSTRACT

Serial estimation of CRP and m-ESR was done in 65 clinically suspected cases of septicemia and 25 healthy controls. Of these 65, 12 (18%) had a negative CRP test at the time of diagnosis and rest all had significantly elevated CRP and m-ESR compared to matched controls at the time of diagnosis. A persistently negative CRP test indicated bad prognosis. With treatment a declining trend of CRP was seen in survivors, but in deteriorating/expired babies the levels kept on increasing. However, m-ESR had no prognostic significance.

Key words: *Neonatal septicemia, CRP, m-ESR.*

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Received for publication: June 1, 1992;
Accepted: September 10, 1992

A number of tests have been employed for the rapid diagnosis and follow-up of the course of neonatal septicemia(1). C-reactive protein (CRP)(2-8) and micro-ESR (m-ESR)(3,9) are two such tests which are simple, rapid and easily available tests, that can be done at most of the centres without the help of sophisticated procedures. The present study, reports observations on the role of CRP and m-ESR neonatal septicemia.

Material and Methods

The study group comprised of Group A of 50 clinically suspected neonates and Group B of 15 clinically suspected neonates who in addition had an obvious focus of infection, *e.g.,* pyoderma, pneumonitis, *etc.* The control Group C comprised of 25 healthy full term newborns.

Various investigations done in the study group were complete blood cell count, differential count, band cell neutrophil ratio and blood culture for pyogenic organisms. In the study group, CRP and m-ESR estimations were done on days 1, 7, 14 and in between if the baby showed clinical deterioration, while in the control group CRP and m-ESR were done only once.

CRP estimation was done by latex agglutination slide test Semiquantitative Rapitex CRP (Hoechst Pharmaceutical Ltd. Bombay), and the cut off value was <6 μg. m-ESR was measured by heparinized capillary tube and cut off value was >10 mm fist hour.

The response to treatment was assessed by clinical examination and fall of CRP and mico-ESR values.

Results

The mean age of neonates in Groups A and B was 11.5 ± 0.9 and 15 ± 1.5 days, respectively ($p<0.05$). The overall inci-

dence of positive blood culture was 14 (21.5%), being 10 (20%) in Group A and 4 (26.7%) in Group B. The mean values of CRP and m-ESR, on day 1 are shown in the *Table*. Since there was no statistically significant difference, for further analysis Groups A and B were combined.

By 14th day, CRP levels touched zero in improved babies, whereas in expired/deteriorated babies the value showed increasing tendency (*Fig.*). Although on 3rd day an increased level was observed in both the groups, but this was an apparent increase only as: (*i*) the test was done only those cases which had shown clinical deterioration; and (*ii*) the difference was not statistically significant. Same is the case of apparent fall in mean CRP values in expired/deteriorated babies on 7th day. The curve of m-ESR also showed a similar trend, but there was no significant difference between expired/deteriorated and improved group. Moreover, the values of m-ESR still remained abnormal even on day 14 for the improved babies.

TABLE –*CRP and m-ESR Levels on Day 1.*

Tests	Mean values on day 1			Statistical significance
	Group A	Group B	Group C	
S.CRP (mg/ml)	12.7 ± 2.6	9.2 ± 1.3	0.7 ± 0.4	A&C $p<0.01$ B&C $p<0.001$ A&B p NS
m-ESR	15.3 ± 1.7	21.1 ± 2.6	2.6 ± 0.2	A&C $p<0.001$ B&C $p<0.001$ A&B p NS

A negative CRP test was observed in 12/65 cases (18%) of which 9 (75%; all LBW) died and only 3 (25%) weighing more than 2.5 kg survived. Of 9 deaths, 6 died within 72 hours, so CRP test could not be repeated. In the other two, the CRP test was persistently negative upto 7 days when they died, and in yet another case which had expired a positive CRP test was observed on day 3 when he died. All had a lower value of m-ESR. The three survivors showed a positive CRP test on day 3, and had higher value of m-ESR compared to CRP negative expired babies. The statistical analysis was not done due to lesser number of cases.

In the study group, the difference in the mean CRP in culture positive and culture negative cases (18.9 ± 22.6; 14.6 ± 14.9) and mean ESR in culture positive and culture negative cases (18.2 ± 8.8 mm; 19.3 ± 13.3 mm), respectively were statistically not significant. Therefore, the culture positive and culture negative cases were pooled together.

Discussion

Like other studies, the present study also shows the diagnostic utility of CRP(2-8) and m-ESR in neonatal septicemia(3,9).

At the time of diagnosis, a higher value of CRP in survivors group, and 75% mortality in 12 babies with initial negative CRP

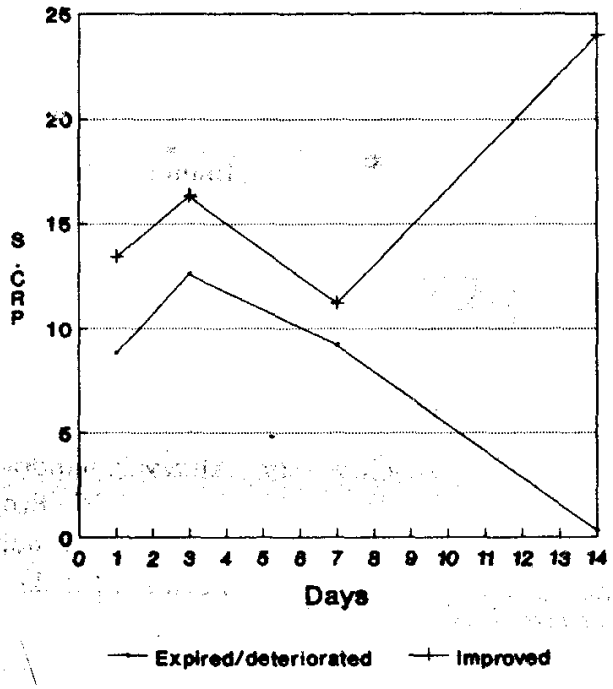

Fig. Mean CRP levels in improved and expired/deteriorated septicemic babies.

value, probably indicates a protective role of CRP in combating infection(1,10). The mode of action is either by acting as a phagocytosis promoting factor(11) or by modifying lymphocyte responsiveness(12). At the time of diagnosis, a negative CRP test indicates presence of excess of antigen in sera of these patients(13).

A good response to antibiotics was indicated by early return of CRP values to normal and persistence or insignificant decline of CRP reflected inadequacy of treatment or development of complications thus requiring change of antibiotics. Similar findings have been previously reported(5,14). As reported in literature(14), no prognostic value of m-ESR was observed in the present study.

REFERENCES

1. Philips AGS, Hewitt JE. Early diagnosis of neonatal sepsis. Pediatrics 1980, 65: 1036-1041.
2. Sann L, Bienvenu F, Bienvenu J, Bourgeois J, Bethenod M. Evolution of serum prealbumin, C-reactive protein and orosomucoid in neonates with bacterial infection. J Pediatr 1984, 105: 977-981.
3. Sann L. Acute phase protein for diagnosis and follow-up of neonatal infections. Indian J Pediatr 1986, 53: 8-10.
4. Pepys MB. Aspects of acute phase response; the C-reactive protein system. *In:* Clinical Aspects of Immunology, 4th edn. Vol 1, Eds Lachmann PJ, Peters D. Oxford, Blackwell Scientific, 1982, pp 50-71.
5. Sann L. Acute phase proteins for diagnosis and follow-up of neonatal infections. Indian J Pediatr 1986, 53: 8-10.
6. Vyas PK, Patel MP, Sheth KR, Shah RC. C-reactive protein in early diagnosis of neonatal sepsis. J Indian Med Assoc 1985, 83: 408-410.
7. Singh, M, Narang A, Bhakoo ON. Evaluation of a sepsis screen in the diagnosis of neonatal sepsis. Indian Pediatr 1987, 24: 39-43.
8. Chandna A, Rao MN, Srinivas M, Shyamala S. Rapid diagnostic tests in

neonatal septicemia. J Pediatr 1988, 55: 947-953.

9. Parida SN, Verma IC, Singh MB, Thomas S. Evaluation of micro erythrocyte sedimentation rate in the diagnosis of neonatal sepsis. Indian J Pediatr 1980, 47: 381-384.
10. Philip AGS. The protective effect of acute phase reactants in neonatal sepsis. Acta Pediatr Scand 1979, 68: 481-483.
11. Kindmark CO. Stimulating effect of C-reactive protein on phagocytosis of various species of pathogenic bacteria. Clin Exp Immunol 1971; 8: 941-948.
12. Mortensen RF, Gewurz H. Effects of C-reactive protein on the lymphoid system. Inhibition of mixed lymphocyte reactivity and generation of cytotoxic lymphocytes. J Immunol 1976, 116: 1244-1250.
13. Hindocha P, Campbell CA, Gould JDM, Wojciechowski A, Wood CRS. Sequential study of C-reactive protein in neonatal septicemia using a latex agglutination test. J Clin Pathol 1984, 37: 1014-1017.
14. Ali SM, Chandra J, Ahmed P, Ahmed KN, Agrawal M. Prognostic value of m-ESR and CRP in neonatal septicemia. Indian Pediatr 1988, 25: 86-866.

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Old Operation Theatre Building,
All India Institute of Medical Sciences,
New Delhi 110 029.

or

Miss Madhumita Roy Choudhury,
Department of Pediatrics,
Genetic Unit,
Old Operation Theatre Building,
All India Institute of Medical Sciences,
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