Increased Frequency of High Serum IgM among Mothers of Infants with Neonatal Group-B Streptococcal Septicemia

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Abstract
Total serum IgM levels were studied in 84 mothers of infants with group-B streptococcal (GBS) septicemia/meningitis and compared to IgM concentrations in 91 parturients who were urogenital carriers of GBS but nevertheless gave birth to healthy infants. In all, 22 (27%) in the study group showed IgM levels above the arbitrarily selected limit of 2.40 g/l, in contrast to 12 (13%) of 91 controls (p = 0.02). Among the study group members whose infants were infected with GBS type III, 8 of 34 (24%) were high in serum IgM, compared to only 2 of 34 (6%) of the corresponding controls (p = 0.04). The total serum IgG levels did not differ between the two groups.

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Group-B streptococci (GBS) are a major cause of serious infections in newborns [1, 7, 9]. Low levels of type-specific antibodies against GBS in maternal serum are associated with an increased risk of GBS infection in the neonate [3, 5]. Antibodies against the carbohydrate-type antigens are particularly important for protection [2, 4], although antibodies to protein antigens might also be crucial [13].

Mothers of GBS-infected infants are not only deficient in IgG antibodies against GBS, but also against other bacterial carbohydrate antigens, including pneumococcal and Gram-negative bacterial antigens [8]. A divergent IgG-allotype distribution among these women indicated a genetic base for the diminished anti-carbohydrate IgG production [11].

In contrast to the IgG, serum IgM levels against bacterial carbohydrate antigens seem to be elevated [7, 8]. We found it therefore interesting to study the total IgM serum levels in mothers of infants with GBS-septicemia.

Sera from 84 mothers of infants with GBS septicemia/meningitis, 80 early and 4 late onset, were investigated. The sera were obtained from various hospitals in Sweden. All infants had growth of GBS in the blood and/or liquor. Strains from 57 patients were available for serotyping, performed as previously described [7], and 10 were type-la, 6 type-lb, 6 type-II and 34 type-III GBS.

The sera from the study group were compared to specimens from a control group of 91 parturients who were urogenital carriers of GBS, but nevertheless gave birth to non-infected infants. Among the controls, 30 harbored type-la, 7 type-lb, 19 type-II and 35 type-III GBS.
The IgG and IgM levels in these sera were quantitated using single radial immunodiffusion [14]. For IgM quantitation, the sera were reduced with dithio-treitol and alkylated using iodoacetic acid in order to equalize the molecular weight distribution [10]. Care was taken to run half/half sera from controls and study group members on each immunodiffusion plate. All sera were tested in duplicates and compared to an international human serum pool. The results were read in a blind manner.

The mean serum IgM level in the study group was 1.84 ± 0.12 g/l, with a range of 0.28–4.50 g/l. The corresponding results for the controls were 1.50 ± 0.10 g/l, range 0.08–4.30 g/l. These values did not differ significantly when compared using Student’s t test (p > 0.05).

To test the hypothesis that the study group might include more individuals with high IgM levels, the number of women with IgM levels above the arbitrarily selected concentration 2.40 g/l were compared between the two groups. In all, 22 of 83 (27%) in the study group, in contrast to 12 of 91 (13%) controls showed high IgM (p = 0.02; Fischer’s exact test). Among the study group members whose infants were infected with type-III GBS, 8 of 34 (24%) where high in serum IgM, compared to only 2 of 34 (6%) controls (p = 0.04; Fischer’s exact test). No significant differences were detected for carriers of any of the other serotypes.

The total serum IgG levels did not differ between the two groups.

The data indicated that some of the mothers with infants contracting GBS septicemia might have a hampered IgM/IgG switch, taking the low levels of IgG antibodies against bacterial carbohydrate antigens in consideration. This deficiency might be a new type of immunological disorder which manifests itself mainly during pregnancy and childbirth [6], since the mothers themselves did not suffer from an increased rate of infections. A recent report has indicated that even a total deficiency of IgG antibodies can be compensated by an increased production of IgM and the IgG-deficient individual can thereby avoid recurrent infections [12], corroborating this theory.

References


