urogenital carcinoma.

We conclude that the addition of dextran sulphate provides a simple way of avoiding direct binding of commercially available enzyme-labelled anti-immunoglobulin conjugates to solid-phase Clq in ELISAs for the demonstration of immune complexes.

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References


IgA deficiency in Israeli blood donors

Immunoglobulin A deficiency occurs rarely, yet potentially fatal anaphylactic reactions may occur if such individuals are transfused with plasma-containing blood components from donors who are not absolutely deficient of IgA (aIgA).2,3 Several national registers of IgA deficient blood donors have been established and large scale screenings to identify IgA deficient individuals have been reported.4-5

Our study was based on the application of a latex agglutination inhibition test (LAI) for the determination of the frequency of IgA deficiency in the Israeli population. The primary advantages of LAI, compared with the conventional radial immunodiffusion (RID), are rapidity and the ability to perform individual tests as required.

Sera were obtained from 4075 healthy Israeli blood donors, comprising 2192 Ashkenazi Jews originating from European communities and 1883 Sephardi Jews originating from Mediterranean or Middle Eastern communities. Known IgA deficient sera were obtained from 10 immunodeficient patients and from 32 newborns.6

Two test methods were used: (i) LAI—based on the inhibition of agglutination of (human) IgA-latex spheres by goat anti-IgA when an IgA-containing serum or other biological fluid is added. Portions (2-0 ml) of standardised goat antihuman IgA serum were mixed mechanically with 20 μl of the serum to be tested, then incubated at 37°C for 10 min. Subsequently, 100 μl of the IgA-latex conjugate were added and after brief mechanical mixing the test-tube was reincubated at 37°C for 90 min in dry heating blocks. Sera with IgA concentrations below 80 μg/ml do not inhibit the agglutination of the IgA-coated latex spheres and the end point of the reaction is a clear solution with white flocculates and sedimented latex spheres. (ii) RID—1803 (44-2%) of the 4075 blood donor sera were tested in duplicate by conventional radial immunodiffusion.7 8

Of the donors screened, only 0-09% (4/4075) were found to be IgA-deficient (<80 μg/ml) by means of the LAI, while 2-7% (49/1803) were deficient by the RID method. All cases which turned out to have low level IgA as detected by the regular RID tests were retested in low level RID plates: only one serum sample was found to contain <40 μg/ml. The 42 known IgA-deficient sera samples were used as controls. The frequency of IgA deficiency was found to be markedly lower among Sephardi Jews—0-053% (1/1883)—than among those of Ashkenazi origin—0-13% (3/2192). In both ethnic groups, the higher sensitivity of the LAI as compared to the RID test was evident.

An attempt was made to relate the frequency of low IgA concentrations to the age of blood donors tested. The highest frequency of IgA deficiency was observed in the 17-19 yr age group (7-7%). However, all the four cases of complete IgA deficiency as determined by the LAI tests occurred in the 30-39 yr age group.

The immediate onset of a severe transfusion reaction—which cannot be ascribed to red cell incompatibility—may be due to the response to drugs in the transfused blood.9-11 Another possible cause for immediate reaction is the transfusion of IgA-containing blood or blood components to an IgA-deficient patient; only a few millilitres of blood or plasma may be sufficient to evoke urticaria, throbbing headache, increased warmth and flush, severe prostration, chest pains and shortness of breath.

In 1968 Fudenberg et al12 first reported the presence of anti-IgA in human sera, applying the haemagglutination inhibition method.13 Anti-IgA occurs in subjects with a selective absence of IgA.14 In such subjects, anti-IgA may occur without a previous history of transfusion. In contrast to Fudenberg's method, a direct approach for the determination of IgA deficiency was used in this study. Both the radial immunodiffusion and latex agglutination tests provide a far more sensitive method for the detection of IgA deficiency, thus making the search for the cause of a transfusion reaction simple and reliable.

Total IgA deficiency (<80 μg/ml) was found in four of 4075 donors (1:1000). This frequency is far lower than that of 1:360 or 1:700 as estimated by CollinsWilliams15 and Bachman16 respectively. The incidence of IgA deficiency seems to be even lower among Sephardi Jews (1:1800). Nevertheless, once the presence of allergy-causing drugs in the transfused blood has been eliminated, the possibility that immediate transfusion reaction results from IgA deficiency in the patient must be investigated.

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References


Letters to the Editor


Separate or combined disk agar diffusion techniques in cotrimoxazole sensitivity testing and use of single versus combination therapy

The report of SGB Aymes and WA Telfer Brunton in your issue of February 1981 showed data remarkably similar to ours (Table). We tested 284 bacterial isolates for sensitivity to cotrimoxazole, sulfa- Diazine and trimethoprim by the disk diffusion agar-overlay method. Antibiotic disks were commercially prepared (Mast Laboratories, England), and contained 250 µg sulfadiazine, 23-75 µg sulfamethoxazole plus 1-25 µg trimethoprim (cotrimoxazole) or 1-25 µg trimethoprim.

Of the strains sensitive to cotrimoxazole, 72% were sensitive to sulfadiazine alone, 87% to trimethoprim alone and only 1% to the combination alone. Of the 173 isolates of *Escherichia coli* and *Klebsiella pneumoniae* which were susceptible to cotrimoxazole, 98% were sensitive to trimethoprim alone. Although we did not study synergistic effects in great detail, we did observe that with organisms sensitive to cotrimoxazole, resistant to sulfadiazine and sensitive to trimethoprim, the zone diameters of inhibition around the cotrimoxazole and trimethoprim disks were identical.

In view of our findings, and those of Aymes and Telfer Brunton, there is certainly a place for the laboratory testing of sulfu and trimethoprim sensitivities singly. Since most organisms are sensitive to either sulfadiazine or trimethoprim, a case could certainly be made for using single drug therapy in different clinical situations.

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References


In vitro susceptibility of bacterial strains to cotrimoxazole, sulfa and trimethoprim

<table>
<thead>
<tr>
<th>Organism</th>
<th>Strains tested</th>
<th>Strains (%) sensitive to cotrimoxazole, sulfa and trimethoprim</th>
<th>Strains (%) sensitive to cotrimoxazole and sulfa, resistant to trimethoprim</th>
<th>Strains (%) sensitive to cotrimoxazole and trimethoprim, resistant to sulfa</th>
<th>Strains (%) sensitive to cotrimoxazole, resistant to sulfa and trimethoprim</th>
<th>Strains (%) resistant to cotrimoxazole, sulfa and trimethoprim</th>
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<td>77 (53)</td>
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<td><em>Klebsiella pneumoniae</em></td>
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<td>10 (14)</td>
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<td><em>Enterobacter spp</em></td>
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<td><em>Proteus mirabilis</em></td>
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<td>21 (4)</td>
<td>16 (70)</td>
<td>1 (4)</td>
<td>0 (0)</td>
<td>5 (22)</td>
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<td><em>Proteus spp</em></td>
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<td>3 (30)</td>
<td>8 (67)</td>
<td>0 (0)</td>
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<tr>
<td><em>Shigella spp</em></td>
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<td>4 (33)</td>
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<td>8 (67)</td>
<td>0 (0)</td>
<td>0 (0)</td>
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<tr>
<td>Total</td>
<td>285</td>
<td>141 (50)</td>
<td>27 (10)</td>
<td>63 (22)</td>
<td>3 (1)</td>
<td>51 (18)</td>
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</table>
IgA deficiency in Israeli blood donors.

R Sharon and A Amar

*J Clin Pathol* 1982 35: 582-583
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