

INFECTIOUS DISEASE

AEROBIC AND ANAEROBIC BACTERIOLOGY OF ADENOIDS IN CHILDREN: A COMPARISON BETWEEN PATIENTS WITH RECURRENT ADENOTONSILLITIS AND ADENOID HYPERPLASIA. I. Brook*, George Washington University, Washington, D.C.

Adenoids were obtained from 18 children with recurrent adenotonsillitis (RA) and from 12 others with adenoid hyperplasia (AH). The adenoids were sectioned in half after heat searing of the surface, and the core material was cultured for aerobic and anaerobic bacteria. Mixed aerobic and anaerobic flora were obtained from all patients, yielding an average of 7.8 isolates (4.6 anaerobes and 3.2 aerobes) per specimen. There were 97 anaerobes isolated. The predominant isolates in both groups were: *Bacteroides* sp. (including *B. melaninogenicus* and *B. oralis*), *Fusobacterium* sp., Gram-positive anaerobic cocci, and *Veillonella* sp. There were 138 aerobic isolates. The predominant isolates in both groups were: alpha and gamma hemolytic streptococci, beta hemolytic streptococci (Group A, B, C, and F), *S. aureus*, *S. pneumoniae*, and *Haemophilus* sp. *Haemophilus influenzae* type B, and *S. aureus* were more frequently isolated in RA Group. *B. fragilis* was only recovered in RA group. Beta lactamase production was noted in 27 isolates obtained from 18 patients. Fifteen of these patients belonged to RA group (83% of RA), while 3 were members of AH group (25% of AH). These bacteria were all isolates of *S. aureus* (11) and *B. fragilis* (2), 8 of 22 *B. melaninogenicus* group, 4 of 11 *B. oralis*, and two of 8 *H. influenzae* type B. Our findings indicate the polymicrobial nature of deep adenoid flora and demonstrate the presence of many beta lactamase-producing organisms in children with recurrent adenotonsillitis.

BACTERIAL STUDIES OF PERITONEAL CAVITY AND POSTOPERATIVE WOUNDS FOLLOWING PERFORATED APPENDIX IN CHILDREN. I. Brook*, George Washington University, Washington, D.C.

This study reports on bacterial specimens obtained from 112 children who presented with a ruptured appendix. Additional samples were studied from 11 of these patients who developed drainage from a postoperative surgical wound. Bacteria grew in 100 peritoneal fluid specimens. Anaerobic bacteria alone were present in 14 specimens, aerobes alone in 12, and mixed aerobic and anaerobic flora in 74. There were 144 aerobic isolates (1.4 per specimen). The predominant isolates were: *E. coli* (57); alpha-hemolytic streptococcus (16); gamma-hemolytic streptococcus (15), Group D streptococcus (12); and *P. aeruginosa* (9). There were 301 anaerobic isolates (3 per specimen). The predominant isolates were: 157 *Bacteroides* sp. (including 92 *B. fragilis* group and 26 *B. melaninogenicus* group); 62 Gram-positive anaerobic cocci; 27 (*Fusobacterium* sp.); and 16 *Clostridium* sp. Beta lactamase production was detectable in 98 isolates recovered from 74 patients. These included all isolates of *B. fragilis* group and 6 of the 23 *Bacteroides* sp. Forty nine organisms (16 aerobic and 33 anaerobic) were recovered from the wounds. The predominant ones were: *B. fragilis* (8); *E. coli* (6); *Peptostreptococcus* sp. (5); and 3 of each of *P. aeruginosa* and *Peptococcus* sp. Most of these isolates were also recovered from the peritoneal cavity of the patients. Our findings demonstrate the polymicrobial aerobic and anaerobic nature of peritoneal cavity and postoperative wound flora in children with perforated appendix, and demonstrate the presence of beta-lactamase producing organisms in three fourths of the patients.

EFFECT OF ASCORBIC ACID ON HOST RESISTANCE IN VIRULENT RODENT MALARIA. G.C. Bourke*, R.M. Coleman*, and N.J. Rencricco, Department of Biological Sciences, University of Lowell, Lowell, Massachusetts.

Virulent rodent malaria is characterized by a fulminating parasitemia, severe anemia and is fatal within several weeks. In view of the oxidant stress that *P. berghei* parasites exert on host erythrocytes and the metabolic effects of ascorbic acid (AA), in the present study we treated malarial mice with AA in an attempt to diminish the severity of parasitemia and enhance host resistance, as reflected by a prolonged survival time. Adult female CD-1 mice were given an intraperitoneal inoculum containing 5.0×10^5 *P. berghei*-infected erythrocytes. The levels of circulating erythrocytes were determined electronically (Coulter Counter) and the % parasitemias scored from methanol-fixed blood smears stained with Giemsa solution. The product of these values was employed to calculate the numbers of parasitized erythrocytes/mm³ of blood. The mean survival time (MST) was expressed as the number of days that 50% of the original population had survived, which equalled 9 days for the infected control group. Infected mice which received daily intraperitoneal injections of AA in a dose of 500 mg/kg, beginning 5 days prior to infection, exhibited a 38% depression in absolute parasitemia and a MST of 15 days. Other groups given AA in a dose of either 500 mg/kg or 1g/kg, beginning on the day of infection, showed an 18% and 23% decrease in parasitemia and a MST of 18 and 21 days, respectively. We conclude that *in vivo* ascorbic acid administration is effective in depressing parasitemia levels and extends the mean survival time of *P. berghei*-infected mice.

IMMUNOLOGIC, FUNCTIONAL AND GENETIC STUDIES OF A PATIENT WITH C8 DEFICIENCY AND REPEATED MENINGOCOCCAL INFECTIONS. P. Densen, R. Clark, G. O'Neill, E. Brown, M. Frank and D. Webb, Boston University, Boston, MA, Sloan Kettering Institute, New York, NY, and NIH, Bethesda, MD.

The association of repeated infections caused by pathogenic *Neisseria* with genetic deficiencies of the terminal complement components has recently been recognized and, in the case of C8 deficiency, a possible association with HLA type has been reported. The predisposition of these patients to repeated *Neisseria* infections has been ascribed to the absence of serum bacteriolytic activity. We have studied a male with 4 separate episodes of systemic disease caused by *Neisseria meningitidis*, all occurring after 15 years of age. His serum had no detectable hemolytic complement. The functional activity of C3, C5, C6, C7 and factor B were normal, but C8 was absent by functional assay and immunochemically using two different antisera in both Ouchterlony and immunoelectrophoretic techniques. Normal hemolytic activity could be restored by the addition of 1500 units of guinea pig C8 per ml of C8-deficient serum. Normal total hemolytic complement and functional C8 levels were present in immediate family members, all of whom had the common antigenic type of C8, HLA and mixed lymphocyte (MLC) response testing of the family revealed a male sibling who was HLA and MLC identical to the patient. Thus, the common antigenic type of C8 does not show HLA linkage. Studies of the bactericidal activity of the patient's serum demonstrated an inability to kill 4 strains of *Neisseria gonorrhoeae* that were readily killed (>90%) by normal pooled human sera. Paradoxically, the patient's fresh (but not heated) serum killed his own infecting strain of group Y *Neisseria meningitidis* as readily (>99%) as normal pooled human sera.

COMPLEMENT ACTIVATION BY CMV INFECTION OF FIBROBLASTS. L.H. Dinh*, D.B. Cines*, and H.M. Friedman, Department of Medicine, University of Pennsylvania, Philadelphia, Pennsylvania.

We previously reported that CMV infection of human embryonic fibroblasts activates human complement resulting in the deposition of membrane C3, as determined by the binding of ¹²⁵I-labeled anti-C3. Complement activation is predominantly by the alternate pathway since C3 binding occurs using C4 deficient serum, and Mg++-EGTA serum as complement sources, but not when sera are heated to 50°C for 25 minutes, or are depleted of properdin factors B or D. C3 binding does not require the presence of CMV antibody and occurs in sera immunochemically depleted of IgG, indicating that complement activation proceeds independent of IgG binding to CMV-induced Fc receptors.

We evaluated the effects of complement on growth of the virus. When mixed with virus, complement does not prevent CMV from initiating infection of fibroblasts. In addition, after onset of infection, adding complement to cultures does not alter progression of infection, as measured by cytopathology, membrane immunofluorescence, and viral titrations. However, the presence of complement markedly enhances the adherence of human erythrocytes to infected monolayers. Erythrocyte adherence is mediated by C3 receptors which are present on these cells since removal of receptors with trypsin treatment or using C3 receptor-negative sheep erythrocytes eliminates red blood cell adherence. These observations suggest that activation of complement following CMV infection may aid in host defence by binding infected cells to blood cells which possess C3 receptors such as monocytes, B lymphocytes, or neutrophils.

INVASIVE EXTERNAL OTITIS: A REPORT OF 21 CASES. R.M. Doroghazi, J.B. Nadol, Jr., N.E. Hyslop, Jr., A.S. Baker, and L. Axelrod, Massachusetts Eye and Ear Infirmary and Massachusetts General Hospital, Boston, MA

We analyzed retrospectively the presenting features, the pathologic, bacteriologic and radiologic findings and results of therapy in 21 cases of invasive external otitis seen at this institution from 1969-1979.

Nineteen patients were diabetic. Fifteen of these 19 had pre-existent, long-standing diabetes (avg. 15.8 yrs) and 11 had microvascular disease. Studies of temporal bones of 2 patients provided evidence of diabetic microangiopathy. *Pseudomonas aeruginosa* was isolated in all 21 patients. Only 3 of 12 patients without bony erosion on x-ray had cranial nerve deficits, restricted to the facial nerve in all 3. Four of 5 patients with bony erosion on x-ray had central nervous system (CNS) deficits. All 12 patients without CNS deficits survived, vs 6 of 9 with CNS deficits. All 13 patients cured with initial therapy were treated with an aminoglycoside (AG) and a semi-synthetic penicillin (SP). All 6 episodes of recurrent disease occurred when only 1 antibiotic was used. The overall mortality was 15 percent (3 of 20 in whom long-term outcome is known).

We propose that diabetic microangiopathy of the temporal bone results in poor local perfusion and creates an environment well-suited for invasion by *Pseudomonas aeruginosa*. There is a correlation between the extent of disease clinically and both radiographic findings and prognosis. Effective treatment requires early diagnosis and combination therapy with an AG and an SP.