Fulminant Pertussis: A Multi-Center Study with New Insights into the Clinico-Pathological Mechanisms.

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Pertussis carries a high risk of mortality in very young infants. The mechanism of refractory cardio-respiratory failure is complex and not clearly delineated. We aimed to examine the clinico-pathological features and suggest how they may be related to outcome, by multi-center review of clinical records and post-mortem findings of 10 patients with fulminant pertussis (FP). All cases were less than 8 weeks of age, and required ventilation for worsening respiratory symptoms and inotropic support for severe hemodynamic compromise. All died or underwent extra corporeal membrane oxygenation (ECMO) within 1 week. All had increased leukocyte counts (from 54 to 132 x 10^9/L) with prominent neutrophilia in 9/10. The post-mortem demonstrated necrotizing bronchitis and bronchiolitis with extensive areas of necrosis of the alveolar epithelium. Hyaline membranes were present in those cases with viral co-infection. Pulmonary blood vessels were filled with leukocytes without well-organized thrombi. Immunodepletion of the thymus, spleen, and lymph nodes was a common feature. Other organisms were isolated as follows; 2/10 cases Para influenza type 3, 2/10 Moraxella catarrhalis, 1/10 each with respiratory syncytial virus (RSV), a coliform organism, methicillin-resistant Staphylococcus aureus (MRSA), Haemophilus influenzae, Stenotrophomonas maltophilia, methicillin-sensitive Staphylococcus aureus (MSSA), and candida tropicalis. We postulate that severe hypoxemia and intractable cardiac failure may be due to the effects of pertussis toxin, necrotizing bronchiolitis, extensive damage to the alveolar epithelium, tenacious airway secretions, and possibly leukostasis with activation of the immunological cascade, all contributing to increased pulmonary vascular resistance. Cellular apoptosis appeared to underlay much of these changes. The secondary immuno-compromise may facilitate co-infection.

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