study had leukocytopenia and neutropenia. In acute viral myositis according to our knowledge [1, 5–11] only 24 out of 63 and 4 out of 49 studied patients had leukocytopenia and neutropenia respectively. This difference between our results and those reported in the literature could be attributed to different causative viral strains or to a different methodology applied. The latter is supported by the fact that in 4 of our patients the initial blood count was normal and only a second blood count performed 2 days later, revealed the leukocytopenia and neutropenia. Thromboeytes were checked on the same occasions but mild thromboctopenia was found only in 7 of our patients. The mechanisms by which viruses induce neutropenia [3, 4] are incompletely understood. It is postulated that neutropenia in these patients may be the result of a number of processes including suppression of myelopoiesis by the infecting agent, excessive neutrophil margination along endothelial surfaces, increase in the peripheral destruction of leukocytes, activation of C5 to C5c complement or production of circulating antineutrophil auto-antibodies. We did not study the pathogenesis of neutropenia in our patients.

References


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Familial primary pulmonary hypoplasia

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Sir: With much interest we read the paper by Frey et al. on familial isolated pulmonary hypoplasia [1]. We know of another family with two male siblings with this condition. The first case was born at 35 weeks after an uneventful pregnancy and delivery. The amount of amniotic fluid was normal. Birth weight was 2770 g. He died a few hours after birth due to respiratory failure. Autopsy revealed very small, non-expanded lungs with a normal bronchi/ alveoli ratio. No other abnormalities were found.

His brother was born at 39 weeks after an uneventful pregnancy and delivery. There was no oligo- nor poly hydramnios. Birth weight was 3435 g and length 52 cm. He too died because of respiratory failure after 1 h. At autopsy a combined lung weight of 12 g was found with a body weight/lung weight ratio of 0.0035 (normal > 0.012 [2]). The bronchi/alveoli ratio was normal. No other abnormalities were found. Their parents are healthy and in seven generations not consanguineous; they have two healthy sons. Family history is not contributory.

Though in the first case pulmonary hypoplasia is strictly speaking not proven, there is in our opinion no doubt that both cases had primary pulmonary hypoplasia, suggesting a genetic aetiology. It is too early to differentiate between a multifactorial or a monogenic cause. In the latter case autosomal recessive inheritance seems the most likely, but as in our family, X-linked transmission cannot be ruled out.

References


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