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Duchenne Muscular Dystrophy in Monozygotic Twins

Duchenne muscular dystrophy (DMD), an X-linked disorder, is a rare occurrence in monozygotic twins, previously reported on five occasions(1-5). We report a rare case of DMD in a pair of monozygotic twins confirmed by DNA analysis for both monozygosity and mutation in the dystrophin gene.

Five-year-old twin boys were admitted to our hospital with history of progressive difficulty in walking, climbing stairs and frequent falls. There was delay in the attainment of motor milestones. Both boys had bilateral calf muscle hypertrophy with weakness of proximal muscles of lower and upper limbs. Serum creatinine kinase levels were grossly elevated (>11,000 IU/L) in both boys, and muscle biopsy showed dystrophic changes. DNA analysis for mutation detection in the dystrophin gene revealed intragenic deletion of exons 45-48 in both children. The monozygosity was confirmed by DNA analysis. Mother's CPK level was within normal limits. Four generation pedigree revealed no other involved member in the family suggesting that the deletion in the dystrophin gene represented a new mutation.

The rare occurrence of such a genetic disorder in monozygotic twins gives us an opportunity to study to what extent genetic and environmental factors control the different manifestations of the disease.

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A Case of Carbon Monoxide Poisoning

A 12-year-old boy was brought unconscious to our casualty nearly 12 hours after he fell asleep in a covered tractor, close to a working generator. He was normotensive, had decerebrate rigidity, equal and reacting pupils, normal optic fundi and no focal deficits. Pulse oximetry showed 95% saturation. ECG revealed sinus tachycardia. Liver and kidney functions were normal. Blood spectrophotometry showed a band of carboxyhemoglobin (COHb). CT scan revealed bilateral symmetrical white matter hypodensity and bilateral round hypodensities in the globus pallidus extending to the internal capsule (*Fig. 1*). He was treated with cerebral decongestive measures and oxygen (10 L/min) delivered by facemask. He expired on the third day of admission. Autopsy disclosed softening of the globus pallidus and other evidence of generalized hypoxic damage.

Carbon monoxide poisoning is the leading cause of death by poisoning in industrialized countries(1). There is a dearth of Indian literature on this subject; a PUBMED search yielded only two references(2,3). Underdiagnosis is probably the reason for this. Nonlethal exposure often goes undetected; the estimate is that 30% of cases are undiagnosed even in the best of centers(4). Exposure to vehicle exhaust fumes, generator fumes, fires in closed spaces, "bukhari" burning and vapours of paint removers containing methylene chloride can all lead to CO poisoning(2).

CO alters the dissociation properties of Hb and reduces oxygen delivery to tissues, leading to central hyperventilation and respiratory alkalosis, which further shifts the oxygen- hemoglobin dissociation curve to the left. The half-life of COHb is 320 minutes, which is drastically reduced to 80 minutes by 100% oxygen at 1 atmosphere and to 23 minutes by 100% oxygen at 3 atmospheres(5). This is the basis for the use of hyperbaric oxygen in the treatment of poisoning.

Diagnosis can be made by spectrophotometry and estimation of COHb If facilities are not available, Kunkel's test may be done. A few drops of 3% tannic acid are added to patient's blood diluted 1:10 with distilled water; the appearance of a crimson red coagulum indicates the presence of COHb(2). Early initiation of oxygen therapy is essential. 100% oxygen should be provided with a non-rebreathing mask to the conscious patient and via endotracheal tube if comatose.

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