In 1896 Bernard-Jean Antonin (1858-1942), a French pediatrician, described a syndrome which subsequently held up his name. This syndrome is characterized by a hereditary disarrangement of connective tissue affecting one or more of three systems namely the skeletal, visual and cardio-vascular. The term "abiotrophy" of connective tissue has been employed by some author for the basic defect of the syndrome. Two or more of four criteria have to be found in diagnosis of Marfan’s syndrome.  

1. Hereditary Features. Marfan’s syndrome is a familial syndrome inherited as a Medelian dominant without sexual preponderance.

2. Ocular Manifestations. The patient usually has bilateral congenital dislocation of lens. Myopia is common. Strabismus, hyperopia or other abnormalities involving lens, cornea and sclera may also be present.

3. Cardio-vascular Manifestations. The aorta, especially the ascending portion, frequently shows medionecrosis because of degeneration and fragmentation of the elastic fibers, resulting in occasional dissecting aneurysm. Degeneration of the connective tissue may involve the endocardium such as the aortic and mitral valves, producing impairment of the cardiac function. Varicosity is another common finding in these persons. They may die with ruptured aortic dissecting aneurysm and massive hemorrhage, or congestive cardiac failure.

4. Skeletal Manifestations. These individuals usually have disproportional elongation of all extremities as well as dolichocephaly with long facial features and malpositioned ears. These physical abnormalities are related to excessive growth of long bone. Classically, elongation of bones is best seen in the miniature long bones of extremities as spider fingers and toes (arachnodactyly). Excessive elongation of the ribs leads to thin chest wall with various pectus deformities. Weakness of ligaments, tendons and fasciae may result in kyphosis, scoliosis, pes planus, looseness of the joint and occurrence of hernia. Severe deformity of the thoracic wall often disturbs the cardio-vascular and pulmonary functions. The subcutaneous adipose tissue is usually scant and exaggerates the linear feature of the patient.

It has been well documented that Abraham Lincoln (1809-1865), the

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sixteenth President of the United States, was unusually tall and thin. This was also noted by the writer on visiting Lincoln’s memorial statue at Washington, D.C. While living, he was 193 cm in height, and the body weight varied between 72.6 to 81.6 Kgm. His feet were notably large and flat. His fingers and toes were bony and remarkably elongated. The scantiness of subcutaneous fat accentuated his already linear proportions. His elongated head was small for the bony, face was long. He was hyperopic, and the eyes were small. The heavy eyelids had a tendency to droop. Squint was present in his left eye. The narrow shoulders were sloping. The thin thoracic wall was sunken. Unsteady gait was noted when he move around, suggesting laxity of the joint. Familiarly, his father was blind in one eye, and the fellow eye was weak. One of Lincoln’s sons had considerable ocular problem with a vertical strabismus. The other son suffered a speech impairment related to congenital anomaly of the palate, and died at 18 years of age with orthopnea. The latter symptom suggests cardio-vascular failure. Similar ocular and cardio-vascular manifestations were also noted in some grandsons of Lincoln. From this cumulative evidence, it was believed that President Abraham Lincoln had Marfan’s syndrome.

REFERENCES

