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UNIVERSITY OF KANSAS MEDICAL CENTER
COLLEGE OF HEALTH SCIENCES AND HOSPITAL
Department of Pathology and Oncology
SURGICAL PATHOLOGY REPORT

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S-80-9944
Pathology Number

Patient's Name: Hallagin, Janet

December 9, 1980

Physician and Service: Medicine II

32 F

Clinical symptoms: proximal muscle weakness since May, 1980.

GROSS DESCRIPTION:

Submitted in the fresh state is a portion of red brown soft pliable skeletal muscle about 1 x 0.8 x 0.2 cm. Several small pieces are fixed in gluteraldehyde for possible E. M. studies. A portion is quick frozen in isopentane/liquid nitrogen and sections prepared for histochemical studies. The remainder is formalin fixed for H&E sections.

MICROSCOPIC EXAMINATION:

Sections show skeletal muscle with no loss of the general architecture. There is no inflammation, vasculitis, scarring or undue fatty replacement. There are scattered moderately atrophic and slightly angular fibers seen. Hypertrophy is not conspicuous. Many fibers show fiber splitting and fragmentation of the sarcoplasm.

~~enzyme stains there is a very marked preponderance of type I fibers with depletion of type II fibers, particularly II/B fibers. In addition, almost all atrophic fibers are type II. Most of the fibers that show the above mentioned splitting, are type I fibers. No excess lipid droplets in the sarcoplasm are seen on fat stains.~~

Diagnosis:

Skeletal muscle biopsy with type II atrophy and overall preponderance of type I fibers.

COMMENT:

The cause of this myopathy is not evident from the biopsy. There are no denervation changes or evidence of polymyositis. Type II atrophy may be seen in collagen-vascular diseases, forced inactivity, upper motor neuron lesions and steroid treatment, it is a fairly non-specific finding, but this muscle is certainly abnormal in a myopathic sense.

J. J. Kepes
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