Introduction

Neuromuscular disorders are a diverse group of neurological conditions. They range from childhood onset to adult onset, with specific diseases having the ability to present in either childhood or adulthood. These disorders, including muscular dystrophies and myopathies, are most commonly diagnosed by examining a muscle biopsy, although with some forms of muscular dystrophy, genetic testing has become standard as a result of increased accuracy coupled with its less invasive nature. The most common form of muscular dystrophy is Duchenne and Becker muscular dystrophy (DMD and BMD, respectively). DMD and BMD are caused by mutations within the dystrophin gene, located on the X chromosome and are inherited as an X-linked recessive conditions **(Lisa, 2007)**.

Cardiomyopathies and arrhythmias are common manifestations of neuromuscular disorders in children. As the gene mutations underlying the neuromuscular disorders are increasingly well-characterized, a significant overlap exists between the mutations found in the primary skeletal myopathies and those described in primary cardiomyopathies **(Hus, 2009)**.

As a result, the cardiac and neuromuscular manifestations of these mutations share common pathophysiologic processes. For example, specific mutations in the dystrophin gene may be predictive of the development of dilated cardiomyopathy in patients with Duchenne (DMD) or Becker (BMD) muscular dystrophy **(Jefferies et al., 2005)**.

Screening for cardiac involvement should be performed in all children with neuromuscular disorders that have the potential for cardiac involvement. Recommendations regarding the timing and frequency of initial and follow-up screening have been made by the American Academy of Pediatrics and experts in the field and are based on the onset and course of cardiac involvement in the individual disorders **(Beynon and Ray, 2008)**.

Cardiac screening should include a careful history and physical examination for symptoms of heart failure or arrhythmias putting in mind that the presence of clinical heart failure can be difficult to ascertain in patients with generalized neuromuscular weakness. At diagnosis, an electrocardiogram and a two-dimensional and Doppler echocardiogram should be performed as part of the cardiac screening process **(Muntoni, 2003)**.

Troponin is the regulatory complex of the myofibrillar thin filament that plays a critical role in regulating excitation–contraction coupling in the heart. Troponin is composed of three distinct gene products (T, I and C) **(Dellefave and McNaly, 2007)**.

It was concluded, that cardiac troponin I (cTnI) had been proven to be expressed in myocardium exclusively, and that cTnI can assess cardiac degeneration independently from skeletal muscle degeneration and is a practical index even in myopathic patients **(Parmacek and Solaro, 2004)**.

It was indicated that cTnI enable us to detect early stage of cardiac degeneration and initiate intervention at proper stage. In DMD, relatively high cTnI values were observed in patients with motor ability of rowing wheelchair in their second decade with strong correlation to Left Ventricular Ejection Fraction (LVEF) **(Castro-Gago et al., 2009)**.