Myasthenia gravis (MG) is the most common disorder affecting the neuromuscular junction (NMJ) of the skeletal musclesIt usually involves muscles of the eyes, throat, and extremities.The differences suggest different pathophysiologic mechanisms of the two subtypes of MG. Neurogenic atrophy seems to be prominent in n-AchR MG, whereas mitochondrial abnormalities in MuSK MG.[8] Thymus: In n-AChR MG, thymus shows epithelial hyperplasia and extra-parenchymal involvement with T-cell areas and germinal centers (GCs).In MuSK MG, the thymus shows age-related changes, and hyperplastic changes are very uncommon.These include myasthenic crisis, an acute respiratory paralysis that requires intensive care, as well as adverse events due to long term medication treatment like opportunistic infections and lymphoproliferative malignancies.Pathophysiology The pathophysiologic mechanisms in MG are dependent on the type of antibodies present.The reduced transmission of electrical impulses across the neuromuscular junction due to the formation of autoantibodies against the specific postsynaptic membrane proteins consequently causes muscle weakness.Muscle: In n-AChR MG, the muscles show significant atrophic changes, whereas, in MuSK MG, there are minimal muscle atrophic changes and significant mitochondrial abnormalities (giant, swollen, and degenerative features).The weakness is more pronounced with the repeated use of a muscle group since it causes depletion of the ACh store in the NMJ.Histopathology The histopathological findings in MG differ by the type of antibodies present.