Cachexia, a condition prevalent in many chronically ill patients, is characterized by weight loss and fatigue, resulting in part from decreases in muscle mass and function. Previous reports have suggested an association between chemotherapy and cachexia, although the mechanisms underlying this association are not clear. With no effective treatments for cachexia today, the purpose of this study was to investigate the effects of a common chemotherapy drug cocktail on myotube morphology and function. On day 4 of differentiation, L6 myotubes were exposed to either a chemotherapy drug cocktail or vehicle treatment. Myotubes treated with the drug cocktail exhibited abnormal cell morphology and ~2-fold decreases in the abundance of myofibrillar proteins Myosin Heavy Chain (MHC), Troponin and Tropomyosin compared to vehicle (n=4, p < 0.05 for each). In addition to a decrease in anabolic signalling observed through ~2-fold decrease in phosphorylated Protein Kinase B (p-AKT) (n=4, p < 0.05), phosphorylation of mTORC1’s downstream targets ribosomal protein s6 (S6) and its kinase (S6K1) were decreased in drug-exposed myotubes (n=4, p < 0.05 for each). Further, reductions in total protein synthesis and mitochondrial content markers Cytochrome C Oxidase (COXIV) and Succinate Dehydrogenase (n=3, p < 0.05) were observed in myotubes receiving drug treatment. Taken together, this data suggests that chemotherapy is associated with alterations in cell protein metabolism, as well as reductions in myofibrillar protein abundance, total protein synthesis and mitochondrial content. These findings suggest that the efficacy of chemotherapy as treatments for cancer comes at a cost to the health of skeletal muscle.