Many mitochondrial diseases are caused by mutations in mitochondrial DNA (mtDNA). Patients' cells contain a mixture of mutant and nonmutant mtDNA (a phenomenon called heteroplasmy). The proportion of mutant mtDNA varies across patients and among tissues within a patient. We simultaneously assayed single-cell heteroplasmy and cell state in thousands of blood cells obtained from three unrelated patients who had A3243G-associated mitochondrial encephalomyopathy, lactic acidosis, and strokelike episodes. We observed a broad range of heteroplasmy across all cell types but also found markedly reduced heteroplasmy in T cells, a finding consistent with purifying selection within this lineage. This finding has important implications for somatic mtDNA mutational heteroplasmy dynamics and poses new questions regarding the role of the immune system in primary mitochondrial diseases as well as the role of mitochondrial in the immune system, particularly in aging.