Some *LMNA* mutations are responsible for partial lipodystrophies with peripheral subcutaneous lipoatrophy, facio-cervical fat accumulation and insulin resistance. Pathophysiological mechanisms are still unknown. We aimed to characterize the enlarged cervical subcutaneous adipose tissue (scAT) from patients with *LMNA* mutations. We studied the histological, immuno-histological, ultrastructural and protein expression features of cervical scAT obtained during plastic surgery in patients with lipodystrophic syndrome of the Dunnigan type (FPLD2) (*LMNA* p.R482W) or metabolic laminopathies (*LMNA* p.R439C, p.H506D and p.R28W). These samples were compared with control cervical scAT, buffalo humps samples from patients under HIV-antiretroviral treatment and dorso-cervical lipomas from patients with mitochondrial (mt)DNA mutations. *LMNA*-mutated enlarged cervical scAT and HIV-related buffalo humps were dystrophic as compared to controls, with increased proportion of small adipocytes, increased fibrosis without inflammatory features, altered expression of adipogenic proteins, and accumulation of prelamin A. Moreover, these fat samples but not controls showed brown fat-like features, with an increased number of mitochondria and overexpression of uncoupling protein 1 (UCP1). Conversely, mtDNA-mutated cervical lipomas showed inflammatory fibrosis with distinct mitochondrial abnormalities but neither UCP1 expression nor prelamin A accumulation. We show here that enlarged cervical fat from *LMNA*-linked and HIV-related lipodystrophies accumulated prelamin A and presented similar fat remodeling towards a brown-like phenotype with UCP1 overexpression, mitochondrial alterations and fibrosis. Prelamin A could be involved in adipocyte transdifferentiation and fibrosis.