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Skeletal Muscle Phenotype in Patients Undergoing Long-Term Hemodialysis Awaiting Kidney Transplantation
Jean-Sébastien Souweine, Fares Gouzi, Éric Badia, Pascal Pomies, Valérie Garrigue, Marion Morena, Maurice Hayot, Jacques Mercier, Bronia Ayoub, Morgie Le Quintrec, Fabrice Raynaud, and Jean-Paul Cristol
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Airflow Limitation, Fatigue, and Health-Related Quality of Life in Kidney Transplant Recipients
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COVID-19–Associated Mortality among Kidney Transplant Recipients and Candidates in the United States
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Training Nephrology Fellows in Home Dialysis in the United States
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On the Cover

Case Description
A 62-year-old male with a history of kidney failure secondary to vesicoureteral reflux on long-term hemodialysis (HD) presented for kidney transplant. He had been on hemodialysis for 3 years. There was no history of diabetes mellitus or arterial hypertension. Physical examination revealed asthenia, and the patient was overweight (29 kg/m²) without muscle atrophy but with a significant decrease in muscle endurance. Interestingly, 1 year after kidney transplant, the patient did not report tiredness. Muscle biopsy was performed just before the kidney transplant.

Image Description:
Transmission electron microscopy analysis on muscle biopsy showed sarcomeric disorganization and proteolysis in the vastus lateralis muscles of long-term HD patients (center). We also observe an altered repartition of mitochondria coupled with an increase of mitochondrial size (left and center). Furthermore, patients present mitochondria with swelling or disrupted internal structures. Mit, mitochondria; Z, Z-line; C, control; P, patient. Scale bar 500 nm.

Teaching Points:
In HD patients, cellular muscle abnormalities are difficult to understand because of comorbidity, malnutrition, age, or sedentary lifestyle. In order to overcome these confounding factors, muscles were analyzed in highly selected HD patients undergoing kidney transplantation who were free of major comorbidities. Our study describes a specific muscle dysfunction characterized by a transition from type 1 (aerobic) to type 2 (anaerobic) muscle fibers without proteasome activation. Moreover, we observed alteration of mitochondria structure related to mitochondrial dysfunction through the activation of autophagy and mitophagy. These observations strongly suggest an energy deficiency in oxidative muscle and could explain the impaired endurance observed in long-term HD patients free of malnutrition and major comorbidities.

(Images and text provided by Fabrice Raynaud, Montpellier University, INSERM, Montpellier, France)
The cover image can be found in this issue of CJASN as Figure 3 A and C in the article titled “Skeletal Muscle Phenotype in Patients Undergoing Long-Term Hemodialysis Awaiting Kidney Transplantation,” by Dr. Jean-Sebastien Souweine and colleagues on pages 1676–1685 (doi: 10.2215/CJN.02390221).