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Abstract
Atrial fibrillation (AF) is the most common sustained arrhythmia, affecting approximately 34 million worldwide. The pathophysiology of AF remains incompletely understood but is clearly complex with multiple underlying genetic, physiologic and environmental factors. Very early-onset AF (vEAF) (defined here as onset <45 years and without significant comorbidities), while rare (only ~0.5-3% of AF cases), is highly heritable, with a greater prevalence of rare variants in genes previously associated with AF. Patients with vEAF, therefore, represent an ideal population for discovering novel genes involved in the underlying genetic basis of AF. Notably, the Framingham study showed that patients with AF without comorbidities have a three-fold higher risk for heart failure. Conversely, several forms of inherited cardiomyopathy have been strongly associated with AF suggestive of a shared etiology.

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