

## DEGLUTITION TRIGGERED ATRIAL FIBRILLATION IN A PATIENT WITH LAING DISTAL MYOPATHY

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Case Vignettes

Deglutition induced atrial fibrillation is a rare clinical entity with a reported prevalence of 0.6%<sup>1-3</sup>. Laing distal myopathy is an extremely rare autosomal dominant muscular dystrophy with an unknown prevalence that is the result of mutations within the slow skeletal muscle fiber myosin heavy chain gene (*MYH7*). Atrial Fibrillation has not been previously reported specifically in patients with Laing distal myopathy. We describe the first reported case of deglutition triggered atrial fibrillation in a middle-aged female who has a history of Laing distal myopathy.

A 44-year-old-female with a history of hyperlipidemia and Laing distal myopathy diagnosed at age 32, began experiencing intermittent episodes of pre-syncope and palpitations which occurred intermittently after swallowing, predominantly during deglutition with food. Episodes of palpitations and pre-syncope post deglutition occurred mostly with warm liquids or food intake. She did not consume alcohol, caffeine and was a non-smoker. An ambulatory 30-day patient triggered event monitor recorded episodes of atrial fibrillation with rapid ventricular response. Each patient triggered event occurred after eating, associated with lightheadedness and/or palpitations. Family history was significant for Laing distal myopathy in her father who is still alive at 72 years old, atrial fibrillation in her mother, as well as sudden cardiac death in two maternal uncles and a maternal grandfather who died in their 50's. Patient blood pressure on presentation was 123/64mmHg, pulse 76 beats /minute and regular, respiration rate 18 breaths/minute, weight 109 pounds and BMI of 22.07 kg/m<sup>2</sup>. Oxygen saturation was 100 % on ambient air. Laboratory data including a basic metabolic panel and thyroid function tests were all within normal range. Resting baseline electrocardiogram demonstrated sinus rhythm, right axis deviation. Transthoracic echocardiogram and cardiac MRI were normal. An exercise treadmill SPECT Imaging stress test revealed normal left ventricular systolic function and no evidence of ischemia or infarction. A fluoroscopic esophagram revealed a mild esophageal dysmotility with no abnormalities. She was started on flecainide 50 mg po every 8 hours and verapamil 40 mg po every 8 hours with no further episodes of atrial fibrillation. There has been no documented of recurrent atrial fibrillation and her symptoms of palpitations have resolved.

Given the strong genetic component of this myopathy, one could postulate as to a possible genetic component in the development of vagally mediated atrial fibrillation in our patient. Consideration should be given to the genetic determination of atrial fibrillation in this patient who has a structurally normal heart, given the associated family history. Although we cannot make definite correlation between deglutition induced atrial fibrillation and Laing myopathy, it is important to report this unusual association which has not been described before. Flecainide in combination with non-beta blocker atrioventricular blocking agents such as verapamil have been effectively used for the treatment of vagally mediated atrial fibrillation.