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## Acute retinal necrosis: A comparison of cases

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Acute retinal necrosis (ARN) progresses quickly to severe vision loss with delayed diagnosis. This comparison of ARN cases shows one diagnosed early and the other misdiagnosed and treated late. A 42-year-old Caucasian male had a respiratory infection treated with prednisone. Facial shingles developed two weeks later treated with Valtrex. Vision was 20/20 and 20/25, respectively. Right eye was normal. Left eye showed anterior chamber inflammation and retinal necrosis. ARN was diagnosed and treated with Valtrex and Durezol. The disease completely resolved and medications discontinued. Two months later, anterior segment inflammation and cystoid macular edema developed in the left eye. This was treated with Valtrex, Pred Forte and Prolensa. Eye drops were discontinued and Valtrex will continue indefinitely. Patient's vision remains 20/20 bilaterally. A 40-year-old Caucasian male presented with one week of rapidly decreasing vision in the left eye. He had a history of herpes treated with Valtrex. An outside physician diagnosed optic neuropathy and retinopathy and started prednisone; vision was 20/200. On presentation to our institution, vision was hand motions and ARN diagnosed. Prednisone was discontinued and started Valtrex and Durezol. Two intravitreal ganciclovir injections were given. Vision improved to counting fingers. He later presented with light perception vision due to retinal detachment. This was repaired and vitreous fluid sent for PCR, which was herpes simplex positive. Vision improved to 20/400 and prophylactic Valtrex continued. This highlights devastating visual consequences with delayed diagnosis of ARN. It is important to have a high index of suspicion for early treatment administration to prevent vision loss.

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## Freeman-Sheldon syndrome: what one illustrative, severe case tells us about pathophysiology and needed research?

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In the context of discussing a patient experiencing the most severe non-neurological case of Freeman-Sheldon syndrome (FSS) reported, of which we are aware, recent basic science research on pathophysiology is described and possible ways these findings complement each-other and resultant implications considered during recent clinical practice guideline development is addressed. While fulfilling Stevenson's 2006 diagnostic criteria, the patient described presented with several previously unreported and rare findings that may serve as 'missing links' in the effort to better appreciate pathophysiology in FSS. Previously unreported findings include: thyroid and cricoid cartilage hypoplasia, a vertical ridge of ossified elevation (right dorsal mid-clavicular line from inferior scapular border to inferior costal margin), left atrophy and right hypertrophy of the latissimus longus and right atrophy and left hypertrophy of the latissimus dorsi muscles, paroxysmal resting tachycardia of typically 110-130 bpm, with a range of 56-215 bpm, without evidence of pathology, severe hyperhidrosis, post-traumatic stress disorder, undersized external auditory canals, and secondary limitation of extra ocular movement. These findings are all consistent with a myopathic aetiology, which was suggested by previous authors and studies. Patient described also experienced subjectively high energy expenditures, estimated to be on the order of 10-25% above normal range. This clinical hypothesis may be at least partially substantiated by recent *in vitro* muscle fibre studies showing impaired cross-bridging in two patients with one of the more common mutations. Mutations for FSS were previously suggested to impair adenosine triphosphate binding at the embryonic myosin head. Physiological appreciation is critical for FSS outcomes.