Case:
65 year-old unresponsive female presents to the emergency department by EMS. Patient was shopping with her family when she suddenly felt weak and slumped to the floor. Per EMS, patient was initially acting confused but became unresponsive just prior to arrival to the ED. The family states that patient has a history of hypertension, diabetes, and an aortic and mitral valve repair secondary to rheumatic heart disease. The patient is taking warfarin, but was told multiple times that there were vegetations on her heart valves and that she was high risk for stroke. On exam the patient was significantly hypertensive and bradycardic. She was unresponsive with only minimal movement of her left side with painful stimuli and no movement of the right side. Her left pupil was nonreactive and dilated to 7 mm while her right pupil was minimally reactive and dilated to 5 mm.

The patient was intubated shortly after arrival and had a CT brain performed that was unremarkable. The stroke team saw the patient and a CTA head and neck was performed showing a large basilar artery occlusion. Neurointerventional radiology was consulted and the patient was transferred for endovascular clot retrieval. A week later the patient was not improving and received a tracheostomy and PEG tube. Her hospital course was complicated by PEG tube dislodgement and subsequent retroperitoneal bleed requiring an explorative laparotomy with placement of a G tube and a subsequent upper GI bleed at the gastrostomy site. One month later, the patient remains minimally responsive with fixed pupils bilaterally, minimal spontaneous movements on the left side and withdrawal to painful stimuli on the right side.

Background:
Basilar artery stroke is a rare but very devastating condition comprising around 3% of all ischemic strokes. The majority of cases are due to thrombosis due to atherosclerotic disease, but can also be secondary to cardio-embolic or infectious sources. The mortality has been estimated as high as 90% without acute intervention. Clinically, patients often present with eye findings such as diplopia or unequal pupils, vertigo, dysarthria, other cranial nerve abnormalities, limb weakness, and ataxia. However, the symptoms can often be vague making diagnosis difficult. The basilar artery occlusion can potentially infarct the cerebellum, midbrain, and/or thalamus as well as areas supplied by the posterior cerebral artery.

Management:
Due to the devastating morbidity and mortality of this disease, aggressive management is indicated. Intravenous thrombolysis can be used if within 4.5 hours of onset per guidelines, but this time limit is generally extended for several hours longer in basilar artery occlusion given the nature of the disease. In recent years, endovascular treatment has gained popularity including intra-arterial thrombolysis, stenting and angioplasty, and thrombectomy. Unfortunately, there is minimal data currently on the effectiveness of these techniques for posterior circulation strokes and more specifically for basilar artery occlusion.

Conclusion:
Basilar artery occlusion is an extremely devastating and often difficult to diagnose disease that should be aggressively managed as the morbidity and mortality are extremely high.

References: