

Left Ventricular Assisted Device Placement in a Systemic Lupus Erythematosus Patient: Challenges in Maintaining Anticoagulation

Klomjit Saranapoom^{1,2}, Phumpattra Chariyawong², Arpana Bansal², Benjamin Hirsch³ and Nandini Nair^{1,2*}

¹Division of Cardiology, Texas Tech Health Sciences Center, Lubbock, TX, United States ²Department of Medicine, Texas Tech Health Sciences Center, Lubbock, TX, United States ³Department of Surgery, Texas Tech Health Sciences Center, Lubbock, TX, United States

*Corresponding Author: Nandini Nair, Division of Cardiology and Department of Medicine, Texas Tech Health Sciences Center, Lubbock, TX, United States.

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Abstract

We report here the case of a sixty three year old African American male with past medical history of well-controlled SLE on MMF, diabetes mellitus, diabetic gastroparesis, hypertension, chronic kidney disease stage III and non-ischemic cardiomyopathy. The patient has had recurrent gastrointestinal bleeding since the last 7 months on LVAD support. The first episode of bleeding occurred 8 months after implant and since then he has had frequent admissions for this problem. Despite arteriovenous malformations being corrected by gastroenterology the bleeding episodes have continued. He is now maintained off all anticoagulation (for 120 days) without any signs of pump thrombosis.

Keywords: Left Ventricular Assist Device; Systemic Lupus Erythematosus; Lactate Dehydrogenase; Pump Thrombosis; Gastrointestinal Bleed

Abbreviations

MMF: Mycophenolate Mofetil; ASA: Aspirin; LDH: Lactate Dehydrogenase; LVAD: Left Ventricular Assist Device; WBC: White Blood Cell; GI bleed: Gastrointestinal Bleed

Introduction

Systemic lupus erythematosus (SLE) is complex autoimmune disease affecting various systems in the body. Patients with SLE have a higher incidence of heart failure compared to general population [1]. Despite aggressive pharmacological intervention, a subset of patients eventually develops advanced heart failure. Though heart transplant remains the gold standard limited donor pool and a long waiting period precludes transplantation in many cases. Left ventricular assisted device (LVAD) therapy therefore has become the mainstay now to help improve survival and quality of life for advanced heart failure patients [2,3]. Common post LVAD implant complications are thromboembolism, hemorrhage, right ventricular failure and prior pre-existing right ventricular failure, GI bleeding and infection [3]. LVAD implantation is particularly challenging in people with autoimmune disease such as SLE. The need for immunosuppressive agents renders these patients highly susceptible to infection [4]. Here we report a case of SLE who successfully underwent left ventricle assist device implant despite recurrent GI bleeding and a serious challenge in maintaining appropriate anticoagulation.

Case Report

Sixty three year old African American male with past medical history of well-controlled SLE on mycophenolate mofetil (MMF), diabetes mellitus, diabetic gastroparesis, hypertension, chronic kidney disease stage III and non-ischemic cardiomyopathy was implanted with an LVAD (Heart mate II) as destination therapy. He has had a history of non-ischemic cardiomyopathy since 2004 and received an implantable cardioverter defibrillator (ICD) in 2013. In 2016, he was hospitalized due to congestive heart failure exacerbations and had to be discharged on continuous home milrinone infusion till LVAD implant in October 2016. His transthoracic echocardiography prior to LVAD implant in September, 2016 reported dilated left ventricle (8.5 cm), severe global hypokinesis with ejection fraction of 15%. Post Heartmate II LVAD hospital course was complicated by cardiac tamponade requiring operative a wash out with complete recovery. During hospitalization, laboratory tests for active SLE. ANA, anti-double strand DNA antibody and anti-Smith antibody were negative suggestive of controlled SLE. He stayed in the hospital for 35 days and was discharged home. Patient was sent home on anticoagulation and his usual dose of mycophenolate mofetil of 1500 mg po bid.

Following his initial post implant discharge patient was readmitted after 3 weeks with a GI bleed which required 24 prbc transfusions. INR was therefore reduced to a target of 1.8 to 2.0 with close monitoring. Von willebrand factor levels were within acceptable limits. An Esophagogastroduodenoscopy (EGD) and colonoscopy was done at this admission which showed acute gastritis and diverticulosis of sigmoid and descending colon. He was placed on intravenous proton pump inhibitor which was subsequently switched to oral route of administration.

Approximately seven months later patient was readmitted for another episode of GI bleed. Patient had a colonoscopy done on which showed non-thrombosed internal hemorrhoids, diverticulosis, blood in the entire examined colon. EGD showed acute gastritis. Biopsy was not significant for any pathology. Patient had a bleeding scan which showed a small GI bleed suspected at the splenic flexure. Pt was referred to interventional radiology for angiogram/embolization and had coil embolization of distal marginal artery branch supplying the superior left colon Coumadin and lovenox were resumed. INR and labs continued to be followed. Pt's activity increased as tolerated. H/H remained stable. Pt was discharged on warfarin, Aspirin (ASA), and full dose low molecular weight heparin (as a bridge to therapeutic INR) with follow up. Following this patient was admitted almost bimonthly for GI bleeding x 7 and for one episode of nasal bleeding which led to coil embolization of bilateral sphenopalatine arteries/internal maxillary arteries and left ascending pharyngeal artery. Finally patient is currently maintained on octreotide (100 mcg subcutaneous injections tid, Procrit 10,000u 3x week and B12 1000 mcg injections q monthly). This patient's course is well-illustrated in figures 1 to 3 showing the effect of discontinuation of MMF, ASA and warfarin on hemoglobin, hematocrit and lactate dehydrogenase. In figure 1 after discontinuation of MMF the white blood cell count increased. In figure 2 the effect of discontinuation of aspirin and warfarin over a period of time did not reduce the instability in hemoglobin/hematocrit levels. It is interesting that despite sub therapeutic INR the ldh has remained stable (Figure 3). The only ldh spike noted was on cauterization of nasal arteries.

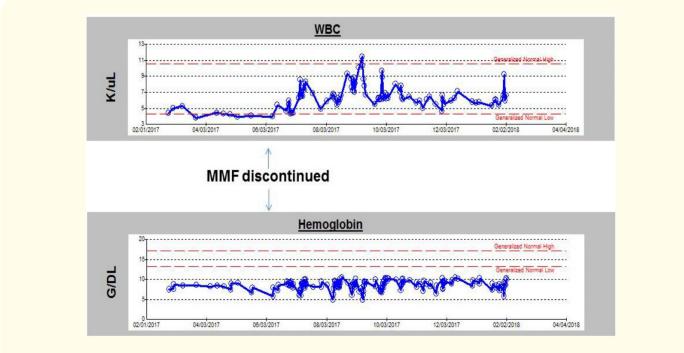
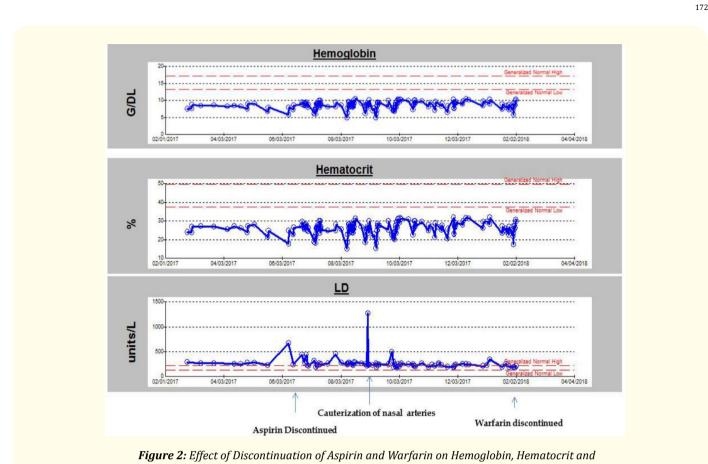


Figure 1: Effect of Discontinuation of MMF on WBC and Hemoglobin.

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Lactate Dehydrogenase.

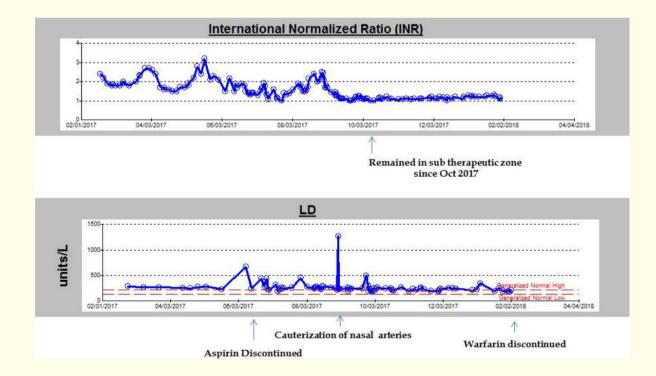


Figure 3: Effect of Discontinuation of Aspirin and Warfarin on INR and Lactate Dehydrogenase.

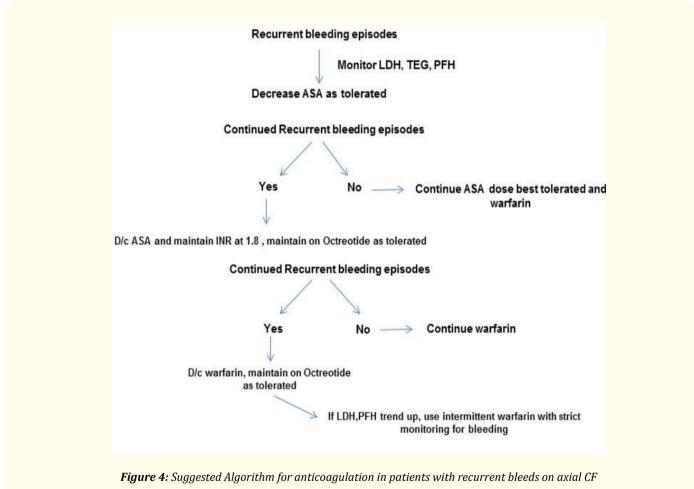
Discussion

We report here a case for the first time of a SLE patient who underwent LVAD placement and is maintained off all anticoagulation. There are several case reports of patients with antiphospholipid syndrome on LVAD support [5-7] but this is the first report in which the challenge of anticoagulation is well illustrated. SLE patients also have a higher a risk of thrombosis especially in those who are antiphos-

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pholipid antibody positive [8]. This patient was negative for antiphospholipid antibody. He did not have active disease at the time of LVAD placement and did not show signs of active disease post placement. Infection and thromboembolism in immunosuppressed patients such as this patient undergoing LVAD is another major concern. Monitoring signs of infection in the setting of immunosuppressive agents can be a challenge. SLE disease flares could be another challenge although this patient has well controlled SLE so far in the absence of any prophylaxis. He continues to have improved quality of life. A suggested algorithm to follow up such patients on continuous flow (CF) axial LVAD support is shown in figure 4. The first step with recurrent bleeds would be to try decreasing doses of aspirin using thromboelastogram (TEG) guidance and continue warfarin. If this fails aspirin should be discontinued. If bleeds continue to recur warfarin may have to be reduced and discontinued with strict surveillance of LDH and plasma - free hemoglobin (PFH). If LDH and plasma-free hemoglobin trend upwards, patient may be intermittently anticoagulated with close monitoring for bleeds. Such an approach may be far from ideal but could prolong fairly good quality of life.



LVAD support.

Conclusion

This case demonstrates that with careful monitoring and prompt intervention, LVAD implantation can be performed safely in patients with SLE. The challenge of sub-optimal anticoagulation still remains.

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