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Antithrombotic Therapy Post Endovascular Stenting for Superior Vena Cava Syndrome

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Short Communication

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ABSTRACT

Superior vena cava syndrome results in obstruction of the superior vena cava and commonly requires intervention. Stents are often used to increase the patency of the vena cava and to prevent reocclusion. The role of antithrombotic therapy and agents to use has been highly debated due to stent location, hematologic complications, and limited clinical evidence available on the topic. This commentary seeks to determine the appropriate choice, if any, for antithrombotic therapy after superior vena cava stent placement.

INTRODUCTION

Superior vena cava syndrome (SVCS) is a diverse clinical condition characterized by the compression or obstruction of the superior vena cava (SVC). The symptomology of SVCS stems from impaired venous return commonly including upper body edema, dyspnea or orthopnea with progression to respiratory distress, convulsions or cerebral edema if left untreated [1]. While the most common etiology is malignancy, endovascular angioplasty with stent placement is considered to be the first line therapy for all origins of SVCS [2]. The use of antithrombotic therapy in conjunction with endovascular stents, however, has been highly controversial.

DISCUSSION

Despite a limited robustness, the pertinent literature reveals a dichotomy of potential antithrombotic therapies. The thrombogenic nature of stent introduction suggests using antiplatelet therapy can reduce platelet activation, aggregation, and stent thrombosis. However, the venous location of stent deployment suggests anticoagulant therapy with a vitamin K antagonist (VKA) may be more favorable [2,3]. Although limited by their retrospective and descriptive nature, the largest studies to date provide insight into the role of antithrombotic therapies [4,5].

In an evaluation of 208 SVCS stenting procedures, *Lanceigo et al*. found no association with dipyridamole ($p = 0.321$) or VKA therapy to mortality ($p = 0.463$) [4]. However, an association with stent thrombosis and an increase in mortality was found regardless of antithrombotic choice ($p = 0.018$) [4]. Antiplatelet medications may therefore be beneficial by reducing stent thrombosis, though this was not explicitly described by Lanceigo et al. [4]. In a similar fashion, Fagedet et al. found no association with the use of aspirin 75-325 mg/day ($p = 0.69$) or VKA ($p = 0.76$) on reoccurrence of SVCS in a study of 164 consecutive SVCS stent recipients [5]. Similar results were also reported in smaller studies and case reports which reported no impact on mortality [5-9]. These reports do however, suggest that antiplatelet therapies may reduce stent thrombosis in the acute setting.

Given the associated burden with VKA interactions, particularly with chemotherapeutics, and its associated monitoring or safety and efficacy, the utility of antiplatelet therapy is compelling. The lack of difference in survival benefit with anticoagulation therapy despite presumed superiority in a venous system model, leads us to conclude that antiplatelet therapy may be appropriate in the absence of other compelling anticoagulation indications. Antiplatelet therapy likely reduces thrombotic risk while having lower potential for hemorrhage compared to VKAs [4,5-9].

CONCLUSION

We recommend using short term antiplatelet therapy with aspirin 75-325 mg/day over anticoagulation in conjunction with endovascular stenting for SVCS.

CONFLICT OF INTEREST STATEMENT

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