

Novel Oral Anticoagulants as Treatment for Vertebral Artery Dissection: Case Report

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Abstract

Background and Purpose—Cervical artery dissections, which may be traumatic or spontaneous, account for a significant proportion of strokes in the young. Antithrombotic therapy is the mainstay of treatment, but new oral anticoagulants could be an alternative treatment to the optimal strategy of anticoagulation followed by antiplatelet drugs.

Summary of Case—We report the case of a 40-year-old patient with a spontaneous vertebral artery dissection who developed a cerebellar ischemic stroke, who had a favorable outcome and complete vessel recanalization after three months of treatment with the oral factor Xa inhibitor rivaroxaban.

Conclusion—New oral anticoagulant could constitute an alternative and new therapeutic option in cervical artery dissections.

Keywords

Anticoagulation; vertebrobasilar disease; stroke

Introduction

Carotid and vertebral artery dissections (VADs) account for a small proportion of all ischemic strokes, but they are a significant cause of stroke in those aged less than 45 years old [1]. Dissections may be the result of trauma or may occur spontaneously. Spontaneous dissection may be the result of inherent arterial wall abnormalities or in association with other predisposing factors. Although the clinical diagnosis can be difficult, computerized tomography or magnetic resonance imaging may be definitive [2]. VAD is associated with nonspecific symptoms such as dizziness, vertigo, headache, or neck pain, and although prognosis is variable and dissections may be asymptomatic, ischemic stroke is the most commonly reported cerebrovascular complication [3]. Optimal management of VAD is not well defined, but options include antiplatelet therapy, anticoagulation,

thrombolysis, and surgical or endovascular procedures [2,4,5]. Although medical therapy is most often used, there is controversy on whether antiplatelet therapy or intravenous heparin followed by oral anticoagulation with warfarin is to be preferred [4]. Recent guidelines recommend “antithrombotic treatment with either antiplatelet or anticoagulant therapy for at least three to six months” [5]. Here, we report a patient with spontaneous VAD treated with factor Xa inhibitor rivaroxaban, who had an excellent outcome and complete vessel recanalization.

Case Report

A 40-year-old right-handed man with no relevant history was admitted to the emergency room complaining of a

Vol. 10, No. 2, pp. 56–58. Published November, 2018.

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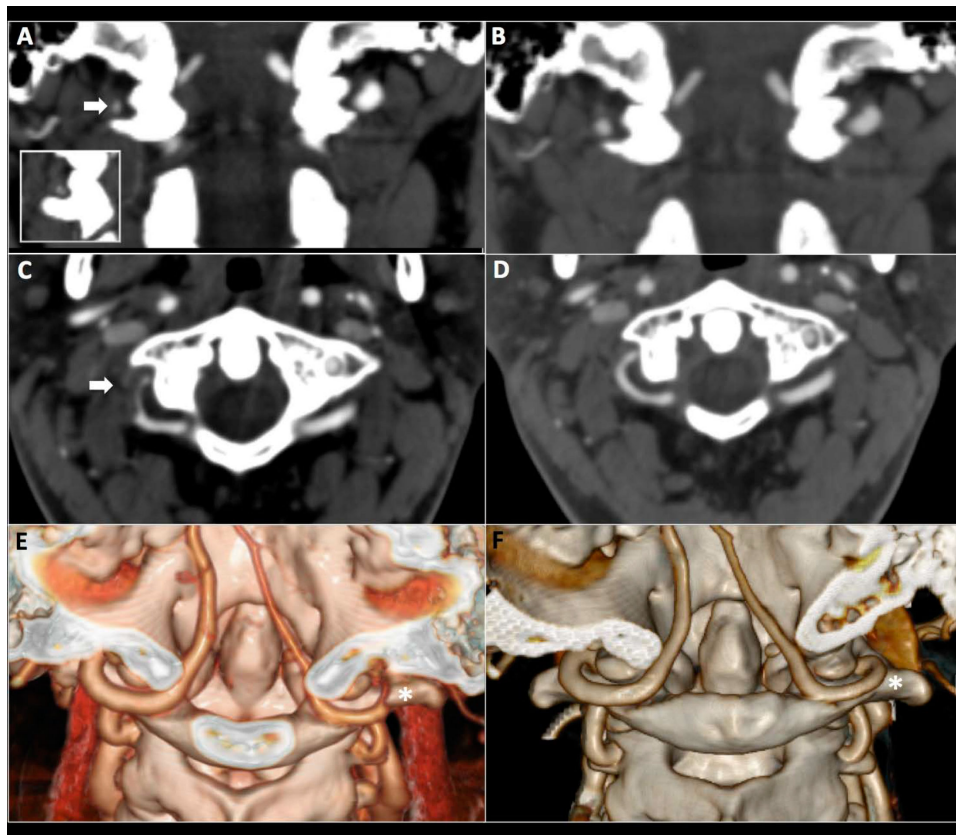


Figure 1. Computed tomography angiography showing a right VAD in V3 segment (a, C arrows; E, near the asterisk). Three months later, there was complete recanalization of the right vertebral artery (B, D, and E).

sudden onset of severe occipital headache, accompanied by vertigo and vomiting. There was no history of trauma or physical activity. On clinical examination, he had a skew deviation of the eyes, vertical and rotatory asymmetric nystagmus, and a right cerebellar syndrome. The rest of his vital signs were normal. Immunological and prothrombotic panels were negative. Brain magnetic resonance imaging showed a right cerebellar infarction. Computed tomography angiography showed a right VAD at the V3 segment, with an intramural hematoma and a narrowed lumen (Figure 1). He was diagnosed with a spontaneous VAD. He was started on rivaroxaban 20 mg/day. The patient improved and was discharged nine days later. After three months of follow-up, the patient was asymptomatic, and a new CT angiography revealed recanalization of the entire vessel (Figure 1).

Discussion

The risk of stroke is the greatest during the first days after dissection of a cervical artery. Although most ischemic strokes caused by dissection are a result of

early thromboembolism, some are attributed to hemodynamic compromise [1]. Traditional treatment consists of early anticoagulation with intravenous heparin, followed by oral anticoagulation with adjusted-dose warfarin (target international normalized ratio 2.0–3.0) for three to six months, with some authors recommending a follow-up imaging procedure to evaluate vessel recanalization. This regimen and the duration of therapy is based on expert opinion [5], and many studies have since confirmed the efficacy and relative safety of antiplatelet therapy [6]. Overall, existing observational data suggest that antiplatelet therapy and anticoagulation are associated with a similar risk of subsequent stroke but that the former is likely safer [4,5]. Recently, a randomized study between antiplatelet and anticoagulant drugs for preventing stroke and death in patients with symptomatic carotid and VAD did not find differences [7].

Anticoagulation is preferred by some authors when there is severe stenosis, recurrent symptoms or free-floating thrombus [4]. Heparin could potentially have protective effects by reducing endothelial dysfunction and reperfu-

sion injury [1]. However, anticoagulation may be associated with the hemorrhagic conversion of ischemic stroke, increased intramural hematoma, heparin-induced thrombocytopenia, a paradoxical hypercoagulable state or systemic bleeding. On the other hand, antiplatelet therapy is preferred in patients with large infarcts, intracranial dissection, or local compression without cerebral ischemia [4]. Intraarterial thrombosis is often platelet rich (“white clots”), making antiplatelet medications a reasonable alternative, and they are less expensive and easier to administer [1]. They are also associated with a lower risk of hemorrhagic complications compared to anticoagulation in other disease states, such as intracranial stenosis and cardioembolic stroke [1].

Rivaroxaban and other novel oral anticoagulants could combine the strengths of both of these alternatives. Rivaroxaban is a Xa factor inhibitor approved for the prevention of stroke in patients with nonvalvular atrial fibrillation [5]. It has a similar efficacy as warfarin, with fewer fatal and intracranial hemorrhages, but higher rates of gastrointestinal bleeding. Rivaroxaban is easy to administer and requires no monitoring and few dose adjustments (only in case of renal disease). There is also experimental evidence that rivaroxaban may have protective effects against arterial thrombotic occlusion. In a rat model of carotid artery injury, rivaroxaban prevented vessel occlusion and had endothelial function stabilizing properties, such as reduction of thrombin–antithrombin complex concentrations, leukocyte adherence, and microthrombus formation [8,9]. It is precisely these considerations have led to increased use of rivaroxaban in the context of arterial thrombosis in acute coronary syndromes [10].

Our patient had a favorable outcome, with complete resolution of his symptoms and complete recanalization after three months of treatment with rivaroxaban.

Although the results of a large clinical trial comparing antiplatelet therapy to traditional anticoagulants are underway, and its results might resolve the controversy over the optimal treatment of VAD, we suggest rivaroxaban could be considered as a rational alternative.

Acknowledgments

None.

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