JVIN | Journal of Vascular and Interventional Neurology

OFFICIAL JOURNAL OF ZEENAT QURESHI STROKE INSTITUTE

High Cortical Subarachnoid Hemorrhage Associated with Treatment of Internal Carotid Artery Aneurysm using Flow Diverter Stent

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Abstract

Background— Isolated high convexity subarachnoid hemorrhage is a rare complication of flow diversion treatment. We present two cases of ipsilateral focal sulcal subarachnoid hemorrhage not related to either aneurysm rupture or arterial wire perforation following flow diverter placement with its probable hypothesis.

Case Description—

Case 1—An adult male presented with painful left oculomotor nerve palsy. Patient underwent balloon assisted coiling for left anterior choroidal artery aneurysm. Angiography after six months showed recanalisation of aneurysm. Pipeline flow diverter was deployed covering aneurysm. After fourteen hours he developed right sided weakness. CT brain showed high convexity subarachnoid hemorrhage.

Case 2—An adult female presented with painful right abducent nerve palsy. Angiography showed dissecting cavernous internal carotid artery aneurysm with right direct carotid cavernous fistula. Surpass flow diverter (4.5 X 20cm) was placed from distal cavernous into ascending limb of internal carotid artery along with multiple detachable coils. Post procedure CT showed high convexity subarachnoid hemorrhage.

Conclusions— High convexity subarachnoid hemorrhage following flow diverter placement is a rare complication. Sudden hemodynamic change / stress at the level of collateral conduits due to watershed shift, augmented systolic flow and in certain instances impaired vasomotor reserve could have resulted in collateral rupture. Flow studies are necessary to confirm our hypothesis

Keywords— Flow diverter, Subarachnoid hemorrhage, Aneurysm, Windkessel, collateral rupture.

INTRODUCTION

Flow diverters reduce flow in aneurysm causing aneurysmal thrombosis while maintaining the patency of artery. Delayed intraparenchymal hemorrhage and subarachnoid hemorrhage due to aneurysm rupture or wire perforation are a known complication post flow diverter.[1–3] We report two cases of high convexity subarachnoid hemorrhage with its probable hypothesis.

CASE DESCRIPTION

Case 1

An adult male presented with painful left oculomotor nerve palsy. Digital subtraction angiography showed multiple aneurysms. Balloon assisted coiling was undertaken for the symptomatic left anterior choroidal artery aneurysm measuring 10.3 x 4.6 x 4 mm. A week later right posterior communicating artery (3.6 x 2.8 mm) and anterior choroidal artery (2.86 x 2.75 mm) aneurysms were treated with single flow diverter (FRED 4 x 23 mm, Microvention, California) from middle cerebral artery to cavernous internal cerebral artery (Fig 1A). He was maintained on aspirin 150mg and prasugrel 10mg daily. Angiography after 6-months showed recanalisation of left anterior choroidal artery aneurysm (Fig 1B). Thus flow diverter (Pipeline embolization device 2.75 x 18 mm, Medtronic, Ireland) was deployed from middle cerebral artery to cavernous internal cerebral artery (FIG 1C). Activated clotting time was maintained twice normal by intravenous heparin. Check angiogram showed

Vol.12, No. 2, pp. 31-34, Published November, 2021.

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stasis in aneurysm sac with good wall opposition. He was extubated with no neurological deficit but fourteen hours later he developed right hemiparesis. CT brain showed high convexity subarachnoid hemorrhage (Fig 1D). Repeat angiography showed patent stent with no thrombosis or embolism with watershed shift towards anterior cerebral artery and collaterals from middle cerebral artery. Focal caliber change of a distal artery in the watershed region with contrast extravasation was noted (Fig 1E,1F,1G). Blood pressure was reduced and repeat angiogram after five minutes showed cessation of extravasation. Single donor platelets were given. CT brain after six hours showed no worsening. His hemiparesis improved and was discharged on antiplatelet agents. Angiography after 6 months showed occlusion of left A1 anterior cerebral artery and distal branches were perfused through collaterals from middle cerebral artery (Fig 1H,1I).

Case 2

An adult female presented with painful right abducent nerve palsy. Digital subtraction angiography showed dissecting cavernous internal cerebral artery aneurysm (Fig 2A) with right direct carotid cavernous fistula. Antiplatelets (aspirin and prasugrel) were given two hours prior to flow diverter. Surpass (4.5 x 20 mm, Stryker neurovascular, Michigan) was placed across the fistula along with multiple detachable coils within the fistula sac and complete occlusion was obtained (Fig 2B, 2C). Post procedure CT showed high convexity subarachnoid hemorrhage (Fig 2D,2E). Angiogram showed no evidence of arterial leak (Fig 2F,2G,2H). Blood pressure was reduced and a repeat CT after six hours showed no increase in bleed. She was discharged on dual antiplatelets.

DISCUSSION

Isolated high convexity subarachnoid hemorrhage following flow diverter is a rare known complication. White et al. has reported six cases of post procedure delayed hemorrhage with four of them manifesting as convexity subarachnoid hemorrhage following flow diverter. They hypothesized possibility of antiplatelets drugs and cerebral hyperperfusion as a cause of bleed with further investigation required.[4] Here we discuss the probable mechanisms underlying this phenomenon.

Intraprocedural inadvertent arterial wire perforation is a commonly considered etiology; however, in this instance, the procedure was done under biplane fluoroscopy and the distal end of wire was never navigated beyond M2 middle cerebral artery. There was no arterial leak observed in per-operative/ post-operative angiographic runs.

Secondly, the possibility of a focal cortical infarct with hemorrhagic transformation as a causative mechanism was examined. The use of prasugrel, a drug with lower probability of platelet resistance, therapeutic intravenous anticoagulation, no evidence of embolism on angiographic runs and no infarct on follow up CT make this mechanism less likely.

Thirdly, could the antiplatelets and anticoagulants used have resulted in hemorrhage? There are isolated reports of trend towards higher rates of intracranial hemorrhage with prasugrel; however, the sample size is small and no definitive conclusion can be drawn from them. Further, the authors have reported on the safety of prasugrel in ten patients with a blister aneurysm treated with a single flow diverter.[5] Finally, none of the studies with newer antiplatelets have reported on high convexity hemorrhage. Therefore, one may conclude that antiplatelets cannot be solely held responsible.

The fourth possibility was aneurysm rupture. The lack of imaging evidence of blood in the basal cisterns pointed to an alternate mechanism other than aneurysm rupture.

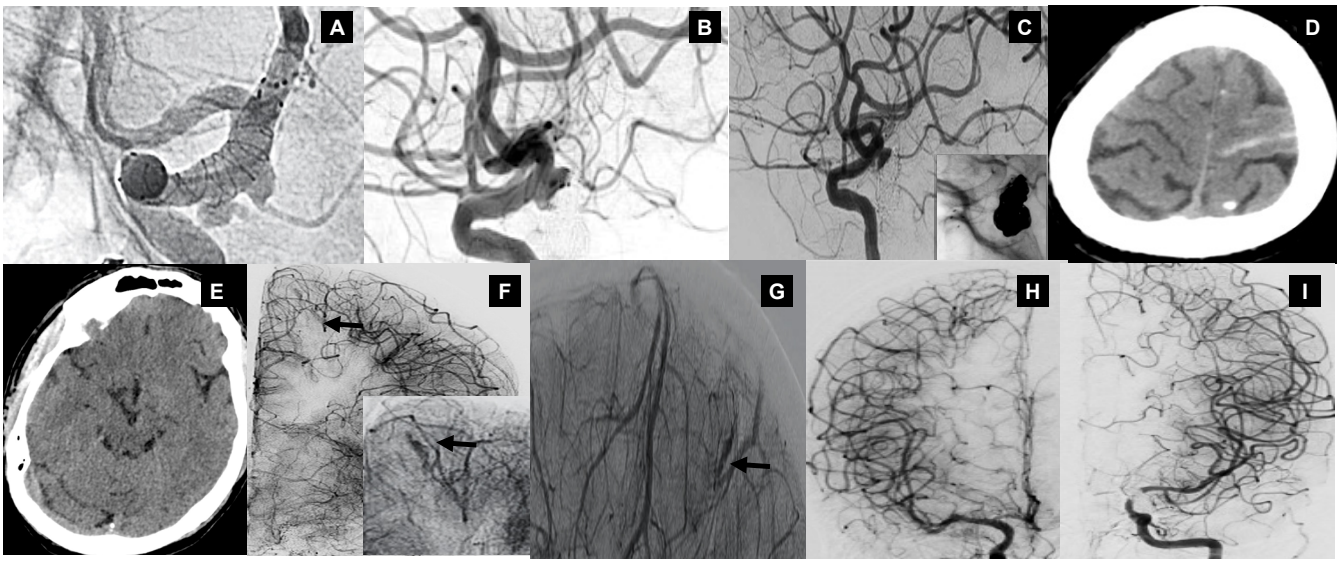

FIGURE 1: (A) Post FD for right PCOM and AChA aneurysm covering ACA (B)Left recanalized AChA aneurysm. (C) Post FD for left AChA aneurysm showing good wall opposition and stasis. (D,E) CT brain showed HCS with no blood in basal cisterna and sylvian fissure. (F) Watershed shift with pial collaterals from MCA and focal caliber change in distal collateral channels with enlarged AP view. (G) Focal caliber change in distal collateral channels with enlarged lateral view. (H) Right ICA DSA after 6 months showing filling of right ACA and left A2 ACA. (I) left ICA showing occlusion of left A1 ACA with pial collaterals from MCA.

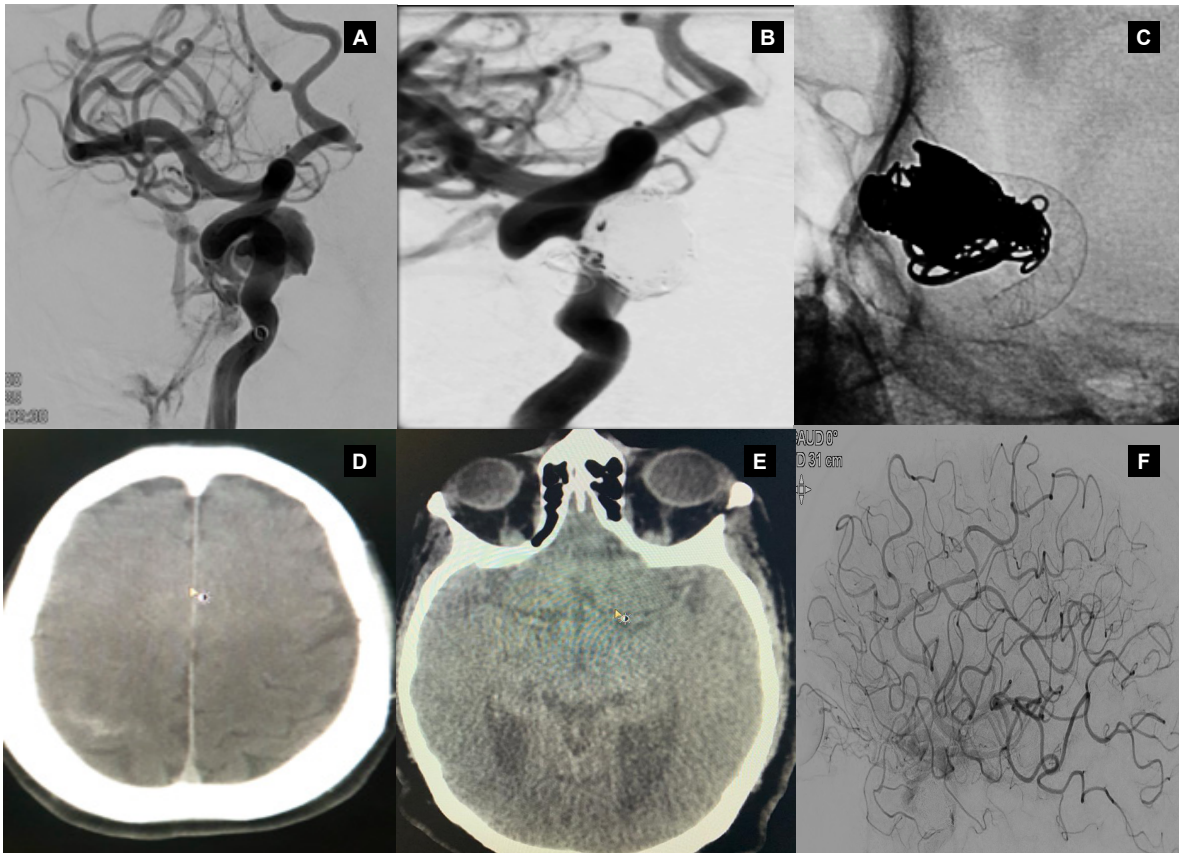

FIGURE 2: (A) DSA showing dissecting cavernous ICA aneurysm with right direct carotid cavernous fistula. (B,C) Post FD and coiling showing complete occlusion with patent flow diverter. (D,E) CT brain showed evidence of HCS with no blood in sylvian fissue. (F) Post DSA showing no arterial leak- AP and lateral view.

Angiography in first patient showed watershed shift towards anterior cerebral artery with pial collateral from middle cerebral artery post flow diverter. The jailing of left A1 anterior cerebral artery caused flow diversion away from ipsilateral anterior cerebral artery territory and an inadequate anterior communicating artery resulted in pial collaterals from middle cerebral artery sub-serving the anterior cerebral artery territory. Therefore, we hypothesize the probability of rupture of pial collateral as the mechanism underlying this bleed. The smooth muscle cells of collateral channels lack myogenic responsiveness and therefore have less tone. They are subject to a higher circumferential wall stress.[6] Multiple case studies have cited collateral vessels as potential source of hemorrhage following large vessel occlusion from atherosclerosis and moyamoya disease.[7,8] In the first case the right anterior cerebral artery origin was covered by flow diverter leading to relative lack of collateral support through anterior communicating artery and consequently greater reliance and more hemodynamic change in leptomeningeal collaterals from middle cerebral artery. The reliance on pial

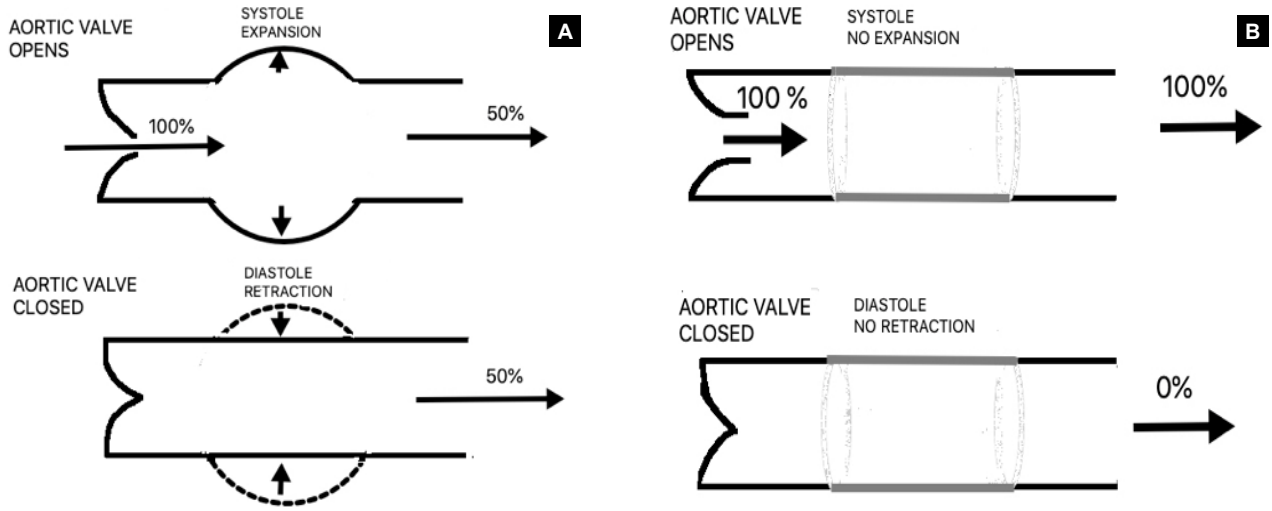

FIGURE 2: A) Windkessel effect - maintained pulse pressure during cardiac cycle leading to continuous flow during both systole and diastole. B) At the site of flow diverter there is loss of elasticity of blood vessels which will alter pressure transmission leading to augmentation in peak systolic flow with low or no diastolic flow.

collaterals was apparent on six month angiography. Further, the loss of windkessel effect (Fig-3), a phenomenon that maintains pulse pressure during cardiac cycle, could have potentially augmented the hemodynamic stress/challenge at the level of the collateral conduits. The loss of elasticity at the flow diverter segment alters pressure transmission in distal circulation leading to augmentation in peak systolic flow with low or no diastolic flow. Thus, we suspect that one of collateral channel ruptured owing to the elevated hemodynamic stress attributable to watershed shift and augmented systolic flow from loss of windkessel effect. In the second patient, a fistula at the cavernous segment resulted in reduced antegrade flow. Therefore, impaired vasomotor reserve due to arteriolar dilatation to maintain perfusion in territory distal to the fistula associated with loss of windkessel effect could have contributed to hemorrhage. Aggressive blood pressure management prevented expansion of bleed and both patients did clinically well.

The key limitation to our study is that we have not performed perfusion studies, MRI diffusion/ Flair or transcranial doppler flow assessments to support our hypothesis.

CONCLUSION

High convexity subarachnoid hemorrhage following flow diverter placement is a rare complication. Sudden hemodynamic change/ stress at the level of collateral conduits due to watershed shift, augmented systolic flow and in certain instances impaired vasomotor reserve could have resulted in collateral rupture. Flow studies are necessary to confirm our hypothesis.

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