

BRIEF COMMUNICATION

KISA RAPOR

**BASILAR ARTERY PATHOLOGIES PRESENTED WITH ISCHEMIC STROKE;
MANAGEMENT AND COURSE CASE SERIES**

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ABSTRACT

Basilar artery occlusive disease has heterogenous clinical presentations and carries a high risk of morbidity and mortality. Treatment strategies have not been proved by large prospective randomised clinical trials due to infrequency and various presentations. Management of the patient can be designated based on clinical and radiological findings and underlying etiologies.

Keywords: Reversible cerebral vasoconstriction syndrome, reversible splenial lesion, postpartum psychosis.

İSKEMİK İNME İLE BAŞVURAN BAZİLER ARTER PATOLOJİLERİ; YÖNETİM VE SÜREÇ VAKA SERİSİ

ÖZ

Baziler arter oklüzif hastalığı heterojen klinik bulgularla ortaya çıkar ve yüksek morbidite ve mortalite riski taşır. Sıklığının az olması ve farklı başvuru bulguları nedeniyle yönetim ve tedavi stratejileri büyük prospektif randomize klinik çalışmalarla kanıtlanmamıştır. Hastanın yönetimi klinik ve radyolojik bulgular ve altta yatan etyolojiler değerlendirilerek belirlenebilir.

Anahtar Sözcükler: Baziler arter, iskemik inme, endovasküler müdahale.

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INTRODUCTION

Ischemic stroke caused by basilar artery occlusive disease has high mortality and dependence rates even with best medical therapy and/or endovascular treatment (EVT) (1-3). Basilar artery pathologies may present with various clinical and radiological pictures (4). Previous randomized studies dealing with anterior circulation acute ischemic stroke with large vessel occlusion repeatedly showed a net superiority of EVT (5) but basilar artery occlusion trials could not achieve the same clear net success (1). Rareness and heterogeneity of basilar artery pathologies create difficulties for randomized studies.

PATIENTS AND METHODS

In our short report we retrospectively evaluated consecutive 20 patients managed and treated at our center and followed up at 3rd month between May/2017-May/2020 with posterior ischemic stroke and detected basilar artery vascular pathologies. We evaluated the severity of clinical findings based on NIH stroke scale score (6); lesion localizations, TOAST classifications (7), distal flow and presence of posterior communicating artery. Informed consent form was signed by all patients for this report.

RESULTS

Early treatment strategies are mechanical thrombectomy (MT) (n=10), intravenous (iv) antiplatelet and/or anticoagulant infusion (n=3), dual antiplatelet therapy (DAPT) (n=4), endovascular stenting and angioplasty (n=1), anticoagulant infusion with MT (n=1) (Table). In our report all patients with stenosis had lesions at proximal and/or mid basilar segments (Table). 3 patients died; all were admitted with NIHSS 27 and treated with MT.

SELECTED CASE SUMMARIES

CASE 1

39 years old male patient admitted to our hospital with imbalance started a few days ago. Diffusion MRI showed right cerebellar hemispheric acute infarction (Figure 1a). Neurological examination was normal. There was a partial thrombus at distal segment of basilar

artery allowing distal flow seen at admission head and neck computed tomography (CT) angiography (Figure 1b,c,d). There was no other large artery disease at CT angiography (CTA). Tirofiban intravenous (iv) infusion was started and given for 8 hrs. Transcranial doppler (TCD) bubble test revealed a right to left shunt then heparin iv infusion was continued as treatment of choice. Transesophageal echocardiography (TEE) proved patent foramen ovale (PFO). At follow-up CTA thrombus was completely resolved. There was no other etiologic cause so PFO was closed. Patient was discharged with no neurological deficit.

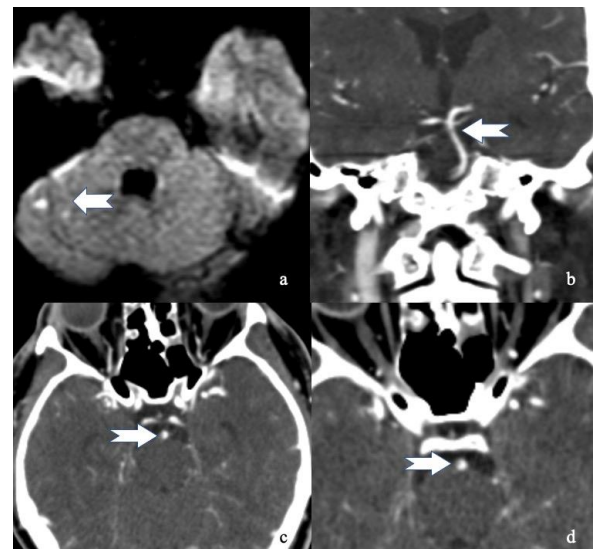


Figure 1a. Diffusion-weighted imaging (DWI) at admission of Case 1. Arrow show the acute ischemic lesion at cerebellum, **b.** CTA coronal plane, **c.** CTA axial plane, **d.** CTA axial plane with magnification, arrows showing partial thrombus.

CASE 15

62 years old female presented with right hemiparesis, NIHSS score 6. Diffusion MRI showed left pontine and peduncle infarction (Figure 2a). CTA showed a significant stenosis (Figure 2b,c) which was regressed at follow-up CTA (Figure 2d) after heparin infusion for 24 hours and dual antiplatelet treatment. Patient was discharged with mRS score 4

CASE 8

68 years old male patient was evaluated for imbalance and nausea-vomiting started two days

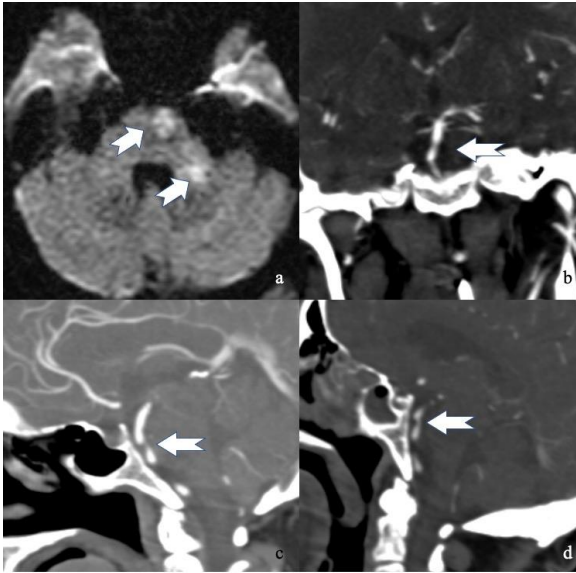


Figure 2a. DWI at admission of Case 15, arrows show the acute ischemic lesions, **b.** CTA at admission at coronal plane, **c.** Sagittal plane CTA at admission, **d.** Sagittal plane CTA at follow up, arrows showing basilar artery stenosis.

ago, he had ataxia and nystagmus at neurological examination. There was cerebellar infarction at diffusion MRI (Figure 3a) and preocclusive stenosis in proximal basilar artery was detected at CTA (Figure 3b). Clopidogrel 75 mg/day and acetylsalicylic acid 100 mg/day were given. During preparation for digital subtraction angiography (DSA) for diagnosis at angiography table, patient had bradycardia and confusion, intubation and emergent angiography were performed under general anesthesia. Preocclusive stenosis causing distal hypoperfusion was confirmed at proximal basilar artery (Figure 3c,d) then stenting and angioplasty were successfully performed at stenosis and a significant and effective dilatation and reperfusion were reached (Figure 3e,f). After operation patient without any new neurological findings and discharged with baseline NIHSS score 2, mRS score was 0 at 3rd month.

CASE 7

32 years old male patient presented with mild left hemiparesis, facial asymmetry and dysarthria; was last seen well 5,5 hours ago. There were cerebellar and occipital ischemic lesions at diffusion MRI (Figure 4 a,b). He has been admitted to neurology service under oral acetylsalicylic acid treatment. He had a generalized tonic clonic seizure 15 hours after last seen well and

transferred to neuro intensive care unit (ICU). CTA was taken at ICU showing midbasilar thrombus with distal complete flow (Figure 4c), right vertebral artery intracranial occlusion before basilar artery junction and left vertebral artery irregularities at junction. He had no new neurological deficit other than postictal confusion. Antiepileptic therapy and heparin infusion was added to acetylsalicylic acid. Under heparin infusion 23 hours after last seen well patient had respiratory arrest, decerebrate posture and left hemiplegia. At 24th hour of last seen well he has been taken to angiography room for EVT. DSA showed proximal basilar and distal left vertebral artery occlusion (Figure 4d). Mechanical thrombectomy was performed with stent retriever (Figure 4e). Complete recanalization was succeeded and final angiography showed bilateral vertebral artery dissection (Figure 4f). Early neurological improvement was achieved at 9th hour of EVT. DAPT was started at follow up and mRS was 0 at 3rd month.

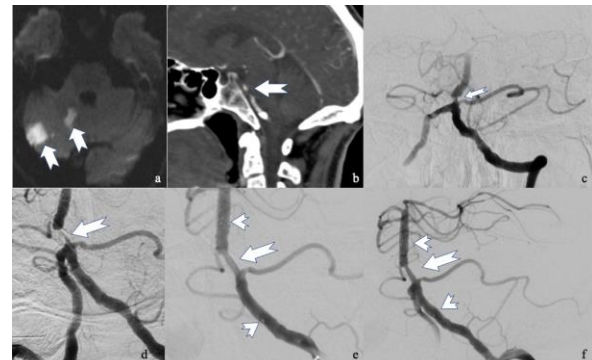


Figure 3a. DWI at admission of Case 8, arrows show the acute ischemic lesions, **b.** Sagittal plane CTA at admission arrow showing critical stenosis at basilar artery, **c-d.** DSA at coronal plane arrows show critical stenosis at basilar artery, **d.** DSA after placement stent at coronal plane, **e.** DSA after post-stent angioplasty, arrows show the opening of stenosis, arrow-heads show stent markers.

DISCUSSION AND CONCLUSION

In our report we presented 20 ischemic stroke patients with basilar artery vascular pathologies. Five of these patients admitted with NIHSS score >20 and only one of them achieved functional independence with EVT. Three patients (cases 13-14-15) with admission NIHSS score <10 had 3rd month mRS \geq 2 showing inconsistency of mild clinical presentations and poor outcomes. Our patients have different aspects

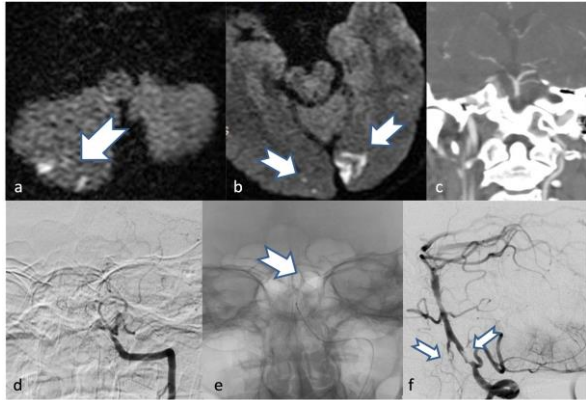


Figure 4a-b. DWI at admission of Case 7 arrows showing the acute ischemic lesions, **c.** Coronal plane CTA showing intact distal flow, **d.** DSA showing proximal basilar and distal left vertebral artery occlusion, **e.** DSA showing stent retriever (arrow) at left PCA, **f.** Dissection at bilateral vertebral arteries (arrows).

for collaterals and distal flow; seven of nine patients with complete distal flow had functional independence compatible with the importance of distal flow.

BEST and BASICS studies did not show a distinct efficacy of endovascular treatment (EVT) in acute basilar artery occlusions (BAO) (8). A multicenter registry trial ATTENTION study has been recently published and demonstrated a clinical benefit of EVT in comparison with best medical management in BAO within 24 hours (3). Better outcomes with EVT in ATTENTION study were clearly significant in patients with baseline NIHSS score ≥ 10 representing a moderate to severe onset (3). The predictors for good outcomes for EVT in BAOs were time to recanalization, early neurological improvement, good collaterals and distal BAO (8). Atherosclerosis at basilar artery was reported to be usually at proximal and mid basilar segments while embolic thrombi usually are found at distal segments (2,9). Collateral scoring is critical for prognosis of basilar artery occlusion patients (10). MT for acute basilar artery occlusions is still the matter of question for benefits, timing and exclusion criteria (11-13).

Each basilar artery ischemic stroke patient should be evaluated carefully and management strategies should depend on clinical and radiological findings.

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Table. Clinical and radiological characteristics of cases.

| Case No | Sex | Age | Basilar artery Pathology | Early Treatment | Lesion | Admission NIHSS Score | Pcomm Presence | Vascular Pathology Localization | Distal Flow | TOAST classification | Treatment at Discharge | 3 rd mo mRS score |
|---------|-----|-----|------------------------------------|---|---|-----------------------|----------------|---------------------------------|-------------|----------------------|------------------------|------------------------------|
| 1 | M | 39 | Thrombus | Tirofiban iv. inf. for 8hr then heparin iv inf. | Unilateral Cerebellar Infarction | 0 | Bilateral | Distal basilar | Complete | Cardioembolic | DAPT | 0 |
| 2 | M | 61 | Stenosis | Oral Antiplatelet | Unilateral Cerebellar Infarction | 0 | Unilateral | Midbasilar | Complete | LAA | DAPT | 0 |
| 3 | F | 72 | Stenosis | Oral Antiplatelet | No Lesion | 2 | Fetal PCA | Midbasilar | Complete | LAA | DAPT | 0 |
| 4 | M | 69 | Athero | Oral Antiplatelet | Bilateral Cerebellar Infarction | 2 | None | Midbasilar | Complete | LAA | DAPT | 0 |
| 5 | M | 71 | Thrombus | MT | Unilateral Cerebellar and Bilateral Occipital Infarction | 4 | None | Proximal and Midbasilar | Complete | Cardioembolic | MAPT, OAC | 0 |
| 6 | M | 53 | Stenosis | Oral Antiplatelet | Unilateral Cerebellar and Pontine Infarction | 4 | Fetal PCA | Midbasilar | Complete | LAA | DAPT | 0 |
| 7 | M | 32 | Thrombus (Vertebral a. Dissection) | Heparin iv inf. for 24hr then MT | Bilateral Occipital Infarction | 16 | None | Proximal Basilar | Absent | Other | DAPT | 0 |
| 8 | M | 68 | Stenosis | Endovascular stenting and angioplasty | Unilateral Cerebellar Infarction | 2 | Unilateral | Proximal Basilar | Complete | LAA | DAPT | 0 |
| 9 | M | 62 | Thrombus | Heparin iv inf. for 48hr | Unilateral Occipital and Mesencephalic Infarction | 4 | None | Midbasilar | Complete | Undetermined | DAPT | 1 |
| 10 | F | 78 | Thrombus | MT | Bilateral Thalamic Infarction | 6 | Fetal PCA | Distal basilar | Absent | Cardioembolic | OAC | 1 |
| 11 | F | 38 | Thrombus | MT | Bilateral Cerebellar Infarction | 10 | Unilateral | Midbasilar | Partial | LAA | MAPT | 1 |
| 12 | M | 56 | Thrombus | MT | Unilateral Cerebellar and Occipital Infarction | 27 | None | Distal basilar | Partial | LAA | MAPT | 1 |
| 13 | M | 86 | Thrombus | MT | Bilateral Cerebellar and Thalamic Infarction | 2 | None | Distal basilar | Absent | Cardioembolic | OAC | 2 |
| 14 | M | 70 | Thrombus | MT | Bilateral Cerebellar Infarction | 8 | Unilateral | Proximal Basilar | Absent | Undetermined | MAPT | 3 |
| 15 | F | 62 | Stenosis | Heparin iv inf. for 24hr | Pontine Infarction | 6 | Bilateral | Proximal and midbasilar | Complete | Undetermined | DAPT | 4 |
| 16 | F | 45 | Thrombus | MT | Unilateral Cerebellar and Occipital Infarction | 27 | None | Midbasilar | Absent | Undetermined | OAC | 5 |
| 17 | M | 39 | Thrombus | MT | Bilateral Cerebellar Infarction and Unilateral Thalamic Hematom | 27 | Unilateral | Distal basilar | Complete | Undetermined | Exitus | 6 |
| 18 | M | 55 | Thrombus | MT (PICA perforation during MT) | SAH | 27 | None | Proximal Basilar | Absent | LAA | Exitus | 6 |
| 19 | M | 37 | Thrombus | MT | Bilateral Occipital Infarction | 27 | Unilateral | Proximal Basilar | Absent | Undetermined | xitus | 6 |
| 20 | M | 65 | Thrombus | Heparin iv inf. for 48hr | Bilateral Cerebellar Infarction | 10 | Unilateral | Distal basilar | Complete | Undetermined | DAPT | 1 |

NIHSS: National Institutes of Health Stroke Scale; Pcomm: Posterior communicating artery; mRS: Modified Rankin Scale iv: intravenous; inf: infusion; DAPT: dual antiplatelet therapy; MAPT: mono antiplatelet therapy; OAC: oral anticoagulant; LAA: large artery atherosclerosis; MT: mechanical thrombectomy; PICA: posterior inferior cerebellar artery; SAH: subarachnoid hemorrhage.

Ethics

Informed Consent: The authors declared that informed consent form was signed by the patients.

Copyright Transfer Form: Copyright Transfer Form was signed by the authors.

Peer-review: Internally peer-reviewed.

Authorship Contributions: Surgical and Medical Practices: DFB, ZY, TDÖ, OS, AEÇ, UŞ. Concept: DFB, ZY, TDÖ, OS, AEÇ, UŞ. Design: DFB, ZY, TDÖ, OS, AEÇ, UŞ. Data Collection or Processing: DFB, ZY, TDÖ, OS, AEÇ, UŞ. Analysis or Interpretation: DFB, ZY, TDÖ, OS, AEÇ, UŞ. Literature Search: DFB, ZY, TDÖ, OS, AEÇ, UŞ. Writing: DFB, ZY, TDÖ, OS, AEÇ, UŞ.

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