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Ultrasonographic evolution of perforator stroke – pictorial essay of medial striate artery infarction in a newborn

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Abstract

Medial striate artery infarction is one of the manifestations of perforator stroke, which poses a risk for basal ganglia destruction. There is no general consensus regarding of origin of the striatal vessels. However, controlling the target brain tissue with available techniques, such as ultrasonography, seems to be the best option for newborns. This pictorial essay presents the ultrasonographic evolution of an infarct in the area of the left head of the caudate nucleus during the early stage and after 4 weeks of observation. Signs of reperfusion, changes in tissue echogenicity, and the occurrence of cystic lesions recorded in lag indicate an alteration. Angiographic magnetic resonance imaging performed at the end of the early follow-up period confirms these observations.

Introduction

Perforator stroke is one of the potential manifestation of perinatal arterial ischemic stroke (PAIS), which is by definition observed between 20 weeks of gestation and 28 days of postnatal life in newborns[1]. It occurs within the territory of the small branches of all arteries in the circle of Willis, especially the anterior (ACA) and middle cerebral arteries (MCA)[2]. These arteries supply blood flow ascending through the anterior perforated substance (APS) to the deep brain structures, including the basal ganglia and internal capsule[3]. Clinical manifestations in newborns may vary, ranging from severe to mild neurological presentations, including asymptomatic cases. They also depend on the phase and morphological stage of infarction[1]. Although MRI is the gold standard technique for the diagnosis and prognosis of stroke, it has some logistical limitations in small patients. This is why cranial ultrasound (CUS) seems to be the best direct tool for evaluating patients' condition. The aim of this pictorial essay is to provide ultrasonographic insights for point-of-care monitoring of medial striate artery stroke.

Ultrasound evaluation of arterial ischemic stroke evolution

A 3-day-old female newborn was admitted to a 3rd-level neonatal intensive care unit from a district hospital due to suspicion of brain hemorrhage on ultrasound. The child was delivered vaginally at term, with a weight of 4090 grams and Apgar scores of 7/7/8/8 at the 1st, 3rd, 5th and 10th minutes of life, as the second baby of a diabetic mother. Hypotonia and desaturation during enteral feeding were observed after birth. A diagnosis of perinatal perforator stroke was established based on CUS. There was no definitive evidence of the cause of the lesion. However, transcardiac paradoxical embolism from the placenta or ductus venosus and maternal diabetes were suspected as potential risk factors. The child did not present any seizures or hemiplegia during hospitalization or during the early follow-up period (Fig. 1).

Within less than an hour after cerebral blood flow stops, cytotoxic oedema with the presentation of stroke occurs. Two major zones in the area of infarction can identified: the core, with already dead tissue, and the penumbra, where hypoperfusion is observed. Ultrasound findings in the early, acute phase of infarction are characterized by mild hyperechogenicity and decreased pulsatility in the affected vessel[4,5].

Subsequently, because of vasogenic edema and coagulation necrosis, tissue echogenicity increased. Reperfusion of the vascular bed may also be observed[5]. This stage is illustrated in Fig. 2 and Video 1 (supplementary material).

The organization of brain tissue ultimately results in cyst formation with gliosis. The process takes approximately 2 months from the onset of the event and ends with tissue loss[5]. Fig. 3, Fig. 4, Fig. 5, and Video 2 (supplementary material) illustrate this stage.

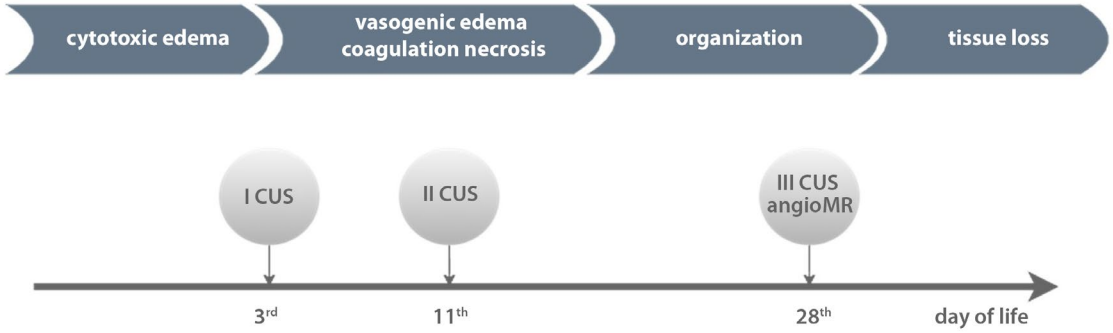

Fig. 1. *Pathophysiological evolution of stroke. Timeline with the patient age at follow-up*

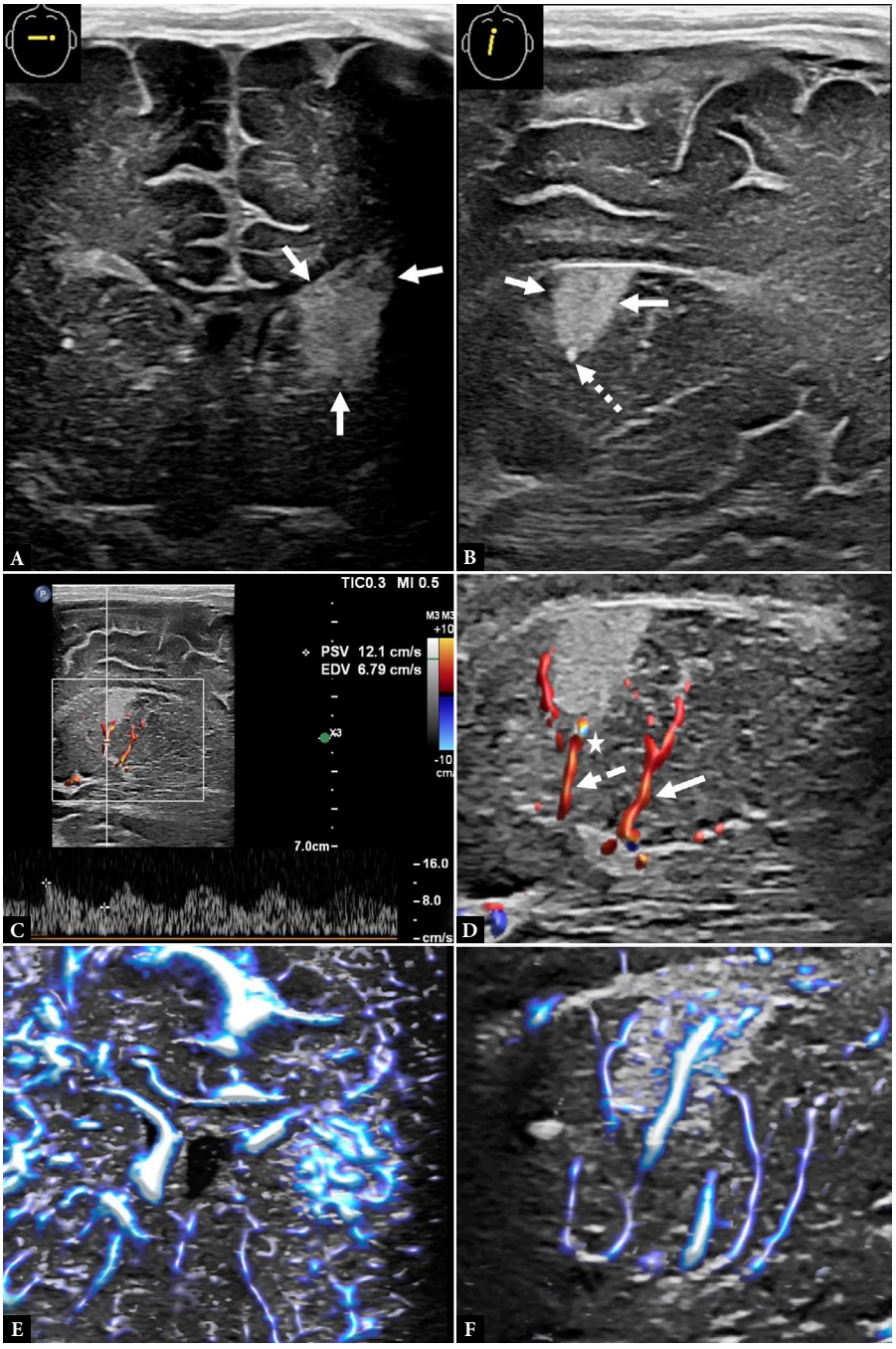

Fig. 2. *First cranial ultrasound (I CUS). Hyperechogenic, heterogeneous area of the left striatum, (arrows, coronal view).* **A.** *Triangular-shaped infarct of the left striatum (arrows), typical for PAIS. The presence of hyperechogenic material within the vessel may suggest embolism (dotted arrow, sagittal view).* **B.** *Arterial slow flow pattern (pulsed wave) registered in the striate artery, with a resistance index of 0.44.* **C.** *Hyperechogenic material blocks the blood flow in the middle striate artery (dotted arrow). There is a twinkling artifact visible in color Doppler, which is typical for hyperechoic, irregular surfaces. The arrow shows the striate vessel with flow visible in color Doppler.* **D.** *Increased blood flow in the area of the penumbra suggests reperfusion, which sustains neuronal demand for oxygen and glucose. Since perforator arteries are terminal vessels, probable flow is maintained with a pressure-passive bed, either through recanalized artery*[5]. **E, F.** *SMI – superb microvascular imaging*

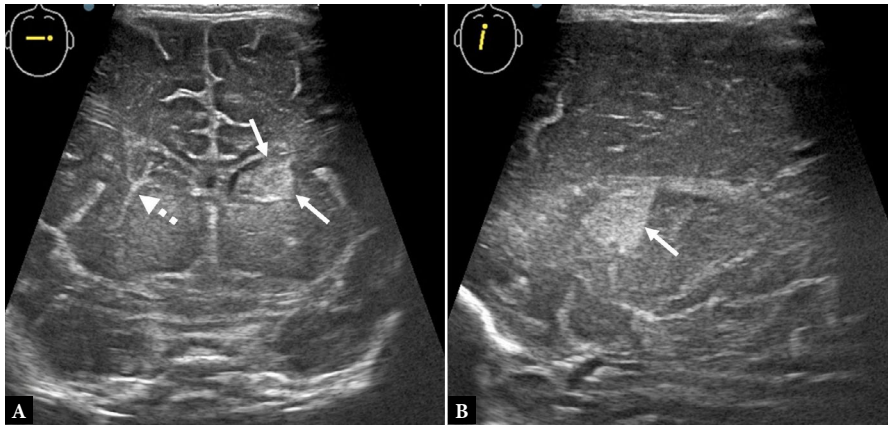

Fig. 3. *Second cranial ultrasound (II CUS). There is lenticulostriate vasculopathy in the right striatum (dotted arrow)* **(A).** *Mild changes in the echogenicity of the infarct (arrows) (***A, B***)*

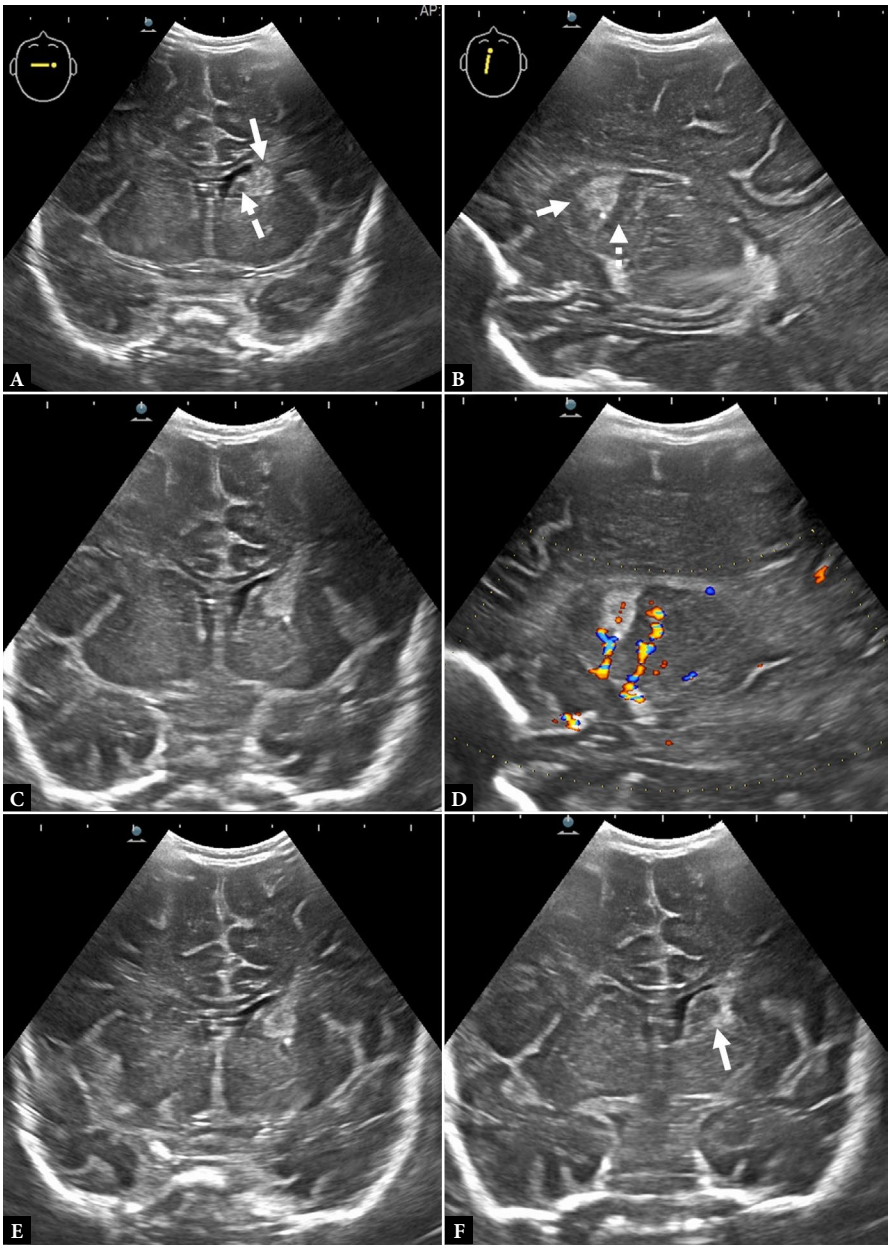

Fig. 4. *Third cranial ultrasound (III CUS). Remodeling of the infarct (arrows,* **A**–**F***) leads to the development of a cyst in the medial part of the head of the caudate nucleus (dotted arrow,* **A***). The cavitation process results in "checkerboard pattern" (***B***) with a characteristic change in the echostructure of the infarct. A marked decrease in the echogenicity of the borderline tissue surrounding the primary lesion is also noted (dotted arrow,* **B***). Lateral ventricle asymmetry with left-side dilatation as a consequence of tissue loss after the event is observed (***A, C, E, F***). Partial resolution of the impairment in blood flow to the affected tissue is visible in color Doppler (***D***)*

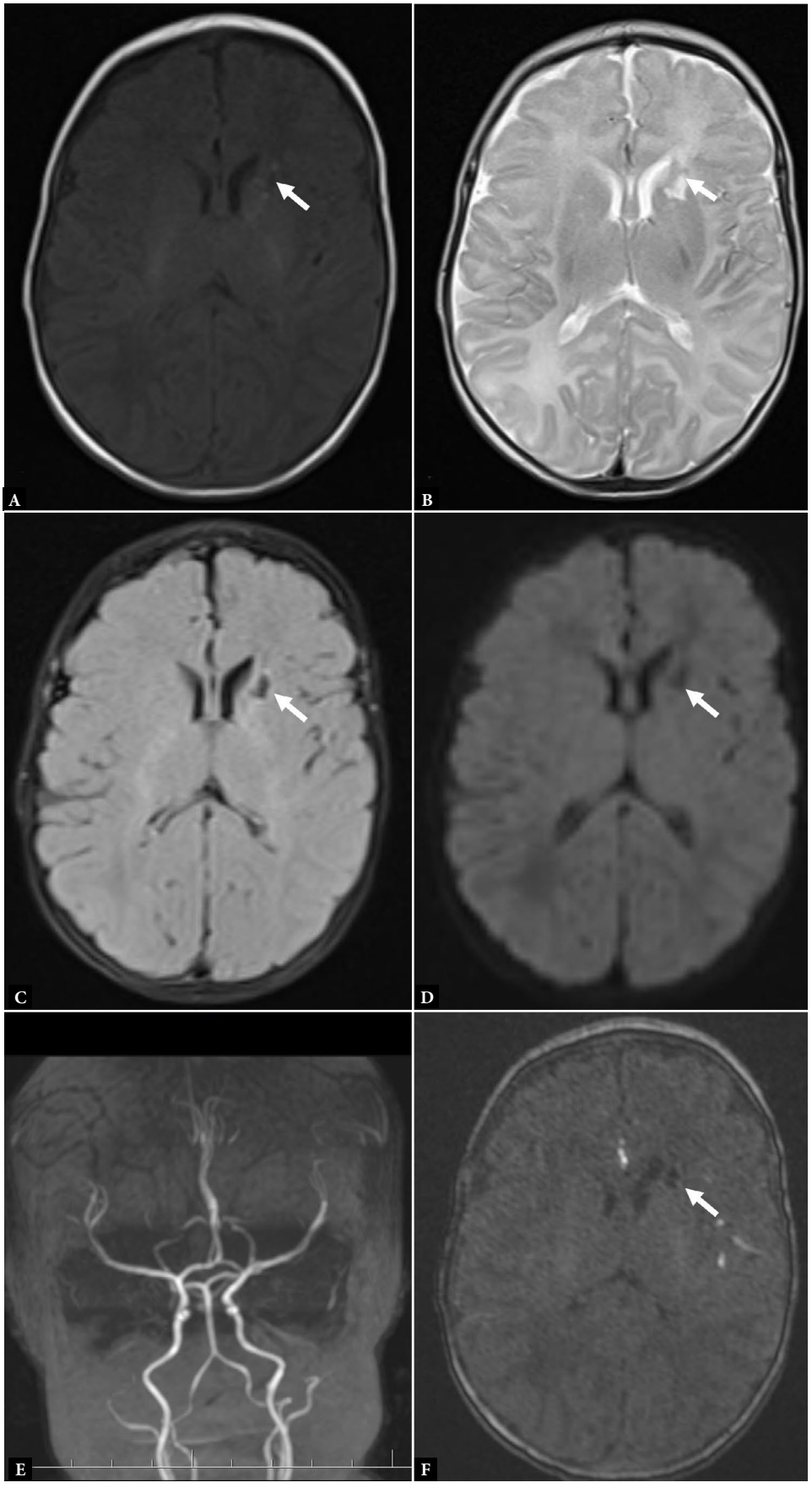

Fig. 5. *Angio MR. Results of magnetic resonance imaging (MRI) on the 28th day after the event (***A–D.** *transaxial views). A centrally located fluid-filled lesion (arrows), surrounded by gliosis, is observed in the left head of the caudate nucleus, with slight invasion into the anterior limb of the internal capsule and putamen (***A.** *T1-weighted sequence;* **B.** *T2-weighted sequence;* **C.** *T2 dark fluid sequence). No restricted diffusion was detected in the RESOLVE (readout segmentation of long variable echo-trains) diffusion-weighted imaging (DWI) (***D**). *There are no signs of malformations in the branches of the circle of Willis in the time-of-flight (TOF) magnetic resonance angiography (MRA) (***E.** *coronal view;* **F.** *transaxial view)*

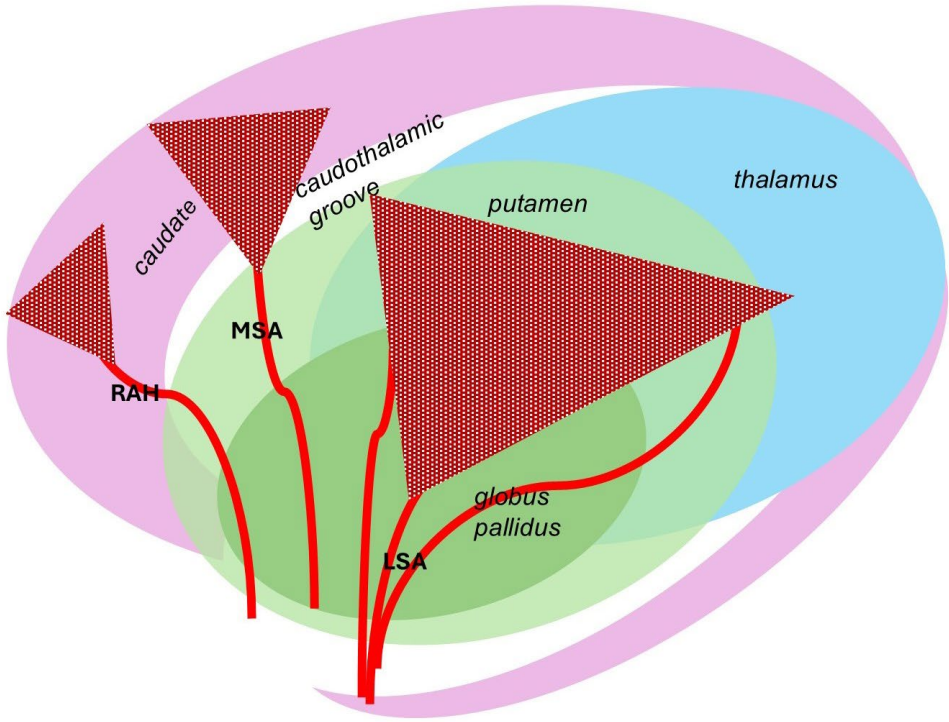

Fig. 6. *Scheme of stroke localization in the ACA and MCA perforator areas based on Abels et al.*[2]. *Lesions are located depending on their position relative to the caudothalamic groove: RAH in front, MSA in the same area or at that anatomical location, and LSA behind*[2]. *The MSA enters the APS and ascends directly through the lentiform nucleus, the caudate nucleus, and the internal capsule. The LSA curves over the lentiform nucleus, ascending the external capsule, transverses the basal ganglia, and finally supplies the upper part of the caudate nucleus, posterior limb, and genu of the internal capsule*[8]. *Lesions of the RAH are typically localized in the antero-inferior part of the caudate head, putamen, and anterior limb of the internal capsule*[6]

Localization of perforator stroke – differentiation based on vascular territory

The nomenclature and anatomical description of the perforators vary among authors. Based on Rosner *et al.*, perforating arteries of the MCA can be classified into medial (medial striatal arteries, MSA) and lateral (lenticulostriate arteries, LSA) groups, using a line that passes posteriorly along the olfactory tract[3,6,7]. Osborn *et al.* classified the distal medial striate artery, called the recurrent artery of Heubner (RAH), as a branch off the ACA A1 segment, whereas the remaining medial and lateral striate arteries as descending from the proximal and distal aspects of the M1 segment of the MCA[8]. Some authors describe all medial striate arteries as arising from the ACA A1 segment[9]. Significant ambiguity remains regarding the origins of the RAH and MSA. Therefore, it is recommended to refer to perforator arteries based on their target tissue rather than their parent trunk[10] (Fig. 6).

Summary

Medial striate artery stroke can be easily monitored in newborns using ultrasonography. Its characteristic shape and localization simplify differential diagnosis. Although most patients do not present any symptoms during early follow-ups, there remains a risk of later manifestations (after 6 months of life) in the domains of gross and fine motor skills. Injury to the area of the caudate nucleus and the anterior limb of the internal capsule may be responsible for pronounced weakness in the arm and face[11]. Therefore, precise monitoring, rehabilitation, and psychological support are recommended for longer follow-up.

Conflict of interest

The authors report no financial or personal relationships with other persons or organizations that could inappropriately influence the content of this publication or claim authorship rights.

Author contributions

Original concept of study: AB, MJ. Writing of manuscript: AB. Analysis and interpretation of data: AB, MJ. Final acceptation of manuscript: MJ. Collection, recording and/or compilation of data: AB, MJ. Critical review of manuscript: MJ.

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