Journal of Integrative Neuroscience

J. Integr. Neurosci. **2024**; 23(1): 5
https://doi.org/10.31083/j.jin2301005

Review

Posttraumatic Basal Ganglia Infarction by Lenticulostriate Artery Injury in Adult Patients: A Review

Jong Un Moon[1], Younghwan Kim[2], Mina Kim[2], Myoung Soo Kim[1,*]

[1]Department of Neurosurgery, National Medical Center, 04564 Seoul, Republic of Korea
[2]Department of Surgery, Trauma Center, National Medical Center, 04564 Seoul, Republic of Korea
*Correspondence: hanibalkms@hanmail.net (Myoung Soo Kim)
Academic Editor: Gernot Riedel
Submitted: 11 July 2023 Revised: 19 August 2023 Accepted: 31 August 2023 Published: 11 January 2024

Abstract

Post-traumatic striatocapsular infarction (SCI) due to lenticulostriate artery (LSA) damage is rare. Most cases reported are in children. We discuss the pathogenesis and differential diagnosis of this kind of SCI after trauma in adult patients. The most common etiology of non-traumatic SCI are an embolism from the proximal artery, cardiogenic embolism, and atherosclerotic plaque in the proximal middle cerebral artery (MCA). However, injury of the LSA after trauma may lead to hemorrhagic infarction in the basal ganglia (BG). Post-traumatic SCI due to LSA damage might be associated with hemorrhage in the BG. The main locations of these lesions are the distal perfusion area of the LSA, similar to SCI due to intracranial atherosclerotic disease affecting the MCA. Vessel wall imaging, magnetic resonance angiography, and ultrahigh-resolution computed tomography can be used for differentiating the injury mechanism in SCI following a traumatic event.

Keywords: trauma; nervous system; cerebral infarction; cerebral hemorrhage; basal ganglia

1. Introduction

Basal ganglia hemorrhagic lesions following trauma are relatively rare and are reported in 0.9%–3.0% of head traumas [1,2]. Striatocapsular infarction (SCI) after trauma is a rare event [3–5]. Differentiation of traumatic or non-traumatic SCI can be important in forensic medicine. Most cases of ischemic infarction in the basal ganglia (BG) after trauma have been reported in young children [6–8]. We found only five cases of post-traumatic SCI in adult patients [9–13]. In adult patients without any stroke risk factors, trauma must be included in probable etiology [9]. Although some have suggested damage to the lenticulostriate artery (LSA) after trauma [11–13], the mechanism of infarction in the BG following trauma remains unclear. We reviewed articles for post-traumatic SCI caused by damage to multiple LSAs and those with spontaneous SCI. We excluded SCI due to traumatic arterial dissection and thrombus in the middle cerebral artery (MCA).

2. Posttraumatic SCI and Hemorrhage by LSA Damage in Adults without Stroke Risk Factor

Head trauma is rarely cited as an etiology of infarction in the BG. In adults and pediatric patients, arterial dissection can result in ischemic events after trauma [14–17]. Trauma from football and other sports results in arterial dissection with thromboembolism of the brain [14]. Dissection of vessels with cerebral embolism to distal territory can be developed after a traumatic event [18].

However, cases of post-traumatic SCI without vessel dissection have been reported in some adults (Table 1, Ref. [9–13]). The combination of subarachnoid hemorrhage (SAH), hemorrhage, and infarction of the BG due to ruptured LSA after trauma has been reported in only two cases [11,13]. In these two patients, other causes for SAH and ischemia, such as aneurysm or arterial-dissection, were not detected with magnetic resonance (MR) imaging or cerebral angiography. In these two patients, the size of the ischemic lesion in the BG was larger than 1.5 cm [11,13].

The BG and internal capsule are perfused by the LSA. These vessels are end arteries. Therefore, the area they perfuse are at risk for ischemia [19]. Any violent motion of the head may result in damage of the vessel between the subarachnoid part of LSA and the intraparenchymal section of the LSA [20]. Inertia from trauma on the skull can also result in movement of brain tissue and damage to intracranial vessels [6]. This movement leads to a traumatic intimal lesion following the generation of a thrombus. This thrombus causes ischemia in the cerebral tissue. Maki *et al.* [20] suggested that when a stretching force is severe, vessel walls can rupture and lead to cerebral hemorrhage, whereas milder damage may only cause endothelial injury and ischemic stroke. Although the case presented was in a young patient, Ahn *et al.* [21] reported that a punctate high-density lesion detected with a computed tomography (CT) scan 2 h following injury indicates a thrombus in the LSA. In the case presentation by Ahn *et al.* [21], repeat CT after the development of hemiparesis demonstrated a discrete infarction around a focal, high-density region. To

Copyright: © 2024 The Author(s). Published by IMR Press.
This is an open access article under the CC BY 4.0 license.

Publisher's Note: IMR Press stays neutral with regard to jurisdictional claims in published maps and institutional affiliations.

Table 1. Clinical characteristics of five adult patients with posttraumatic striatocapsular infarction.

Sex/age	Mechanism of Trauma	Initial neurological deficit	Image findings	Associated injury	Year	Reference
F/19	Fell off boat	Hemiparesis, facial droop, slurred speech	Acute infarction in internal capsule	No	2014	[9]
M/32	Hit by a truck while driving a bike	Paresis in all four limbs, GCS score 7/15	Right BG infarction and left BG hematoma	External injuries on the right side of body, head and neck	2016	[10]
M/30	Impact to the head during brawl	Disoriented, hemiparesis	SAH, hemorrhage and infarction in BG	Skull base fracture	2012	[11]
F/25	Traffic accident	Mild weakness and numbness in left hand	Hemorrhage and infarction in BG	Fracture in femur, ulnar radius, scaphoid, and hamate	2019	[12]
F/34	Falling backward during a brawl	Hemiparesis, GCS score 12/15	SAH, hemorrhage and infarction in BG	Temporal bone fracture	2023	[13]

F, female; M, male; GCS, Glasgow coma scale; BG, basal ganglia; SAH, subarachnoid hemorrhage.

our knowledge, this description is the first report suggesting a thrombus after trauma. Direct visualization of LSA damage after trauma was reported by Kim *et al*. [12]. An angiographic evaluation of intracranial vessels in four of five cases in adults with post-traumatic BG infarction did not demonstrate damage to the LSA despite CT angiography [9,10], MR angiography [13], or cerebral angiography [11]. Only one of five cases in adults with post-traumatic SCI demonstrated structural damage in the LSA by cerebral angiography and MR angiography [12]. Perhaps the incidence of this mechanism in head trauma is rare and detailed documentation may identify more traumatic causes for stroke [6].

Most cases of post-traumatic SCI due to LSA damage have been reported in children. In the event of a BG infarction in a young child following head trauma, the etiology is often the result of anatomical characteristics at this age [22]. For example, the LSAs form sharp angles with the MCA, which is more acute in children than adults [20,23]. Anatomically, between the intracerebral and the subarachnoid segment of the LSAs, there is a mobile subarachnoid segment, that when stretched and distorted by trauma, results in ischemic lesion in the area of LSAs. Additionally, the sphenoid bone in children is developing and not fully ossified. The brain parenchyma has greater mobility than the skull during violent trauma. Moreover, the subarachnoid space in children is smaller and less protected from trauma than in adults. These factors facilitate the stretching of LSAs by traumatic forces [4,24]. Cerebral blood flow in the first five years increases to twice that of an adult. Thus, children are more susceptible and sensitive to cerebral hypoxia. In young children, even mild head trauma could result in BG infarction [22,25–27].

3. Non-Traumatic SCI

The damage of a deep perforator vessel in the brain can cause a lacunar infarction with a diameter of $<$1.5 cm [28]. Perforating infarcts with a diameter larger than 1.5 cm in the LSA territory can develop from the occlusion of a main stem perforator or simultaneous damage from multiple perforators [29]. Among ischemic lesions in the BG, large subcortical lesions with a size $>$3 cm are classified as a SCI [30]. The clinical presentations of a SCI typically include hemiparesis, language problems, sensory neglect, or apraxia [31].

Following a study conducted by Bladin and Berkovic [32], SCIs were classified as a subtype of ischemia. The ischemic lesions of SCI were located in territories of the LSAs and could extend to territories of the Heubner or anterior choroidal arteries [33–35]. Jose and James [36] proposed classifying ischemic stroke into 10 subtypes. Of the 10 infarction subtypes, SCI is considered a subcortical infarction in the striatocapsular portion, developed following damage to more than one LSA [36]. According to its radiological findings, especially on axial images, these SCIs are lentiform, triangular, or comma-shaped. The size of a SCI is 3–4.5 cm, 1–2 cm wide and 2–4 cm in depth. The impacted infarction regions include the caudate nucleus, putamen, and anterior limb of the internal capsule. The globus pallidus, genu, and posterior limb of the internal capsule are not typically affected. The comma-shaped lesions involve the caudate nucleus along with the anterior limb of the internal capsule and the lentiform nucleus [36]. Cardiac problems, severe carotid artery stenosis, and atherosclerotic disease affecting the proximal MCA are other important causes of SCI [3,32,33,37–40].

Lee *et al*. [41] reported that the dominant portion of SCI on coronal diffusion MR was important for identification of stroke cause. Stroke resulting from a proximal embolism, such as those with cardiac origin, is more preva-

lent in patients with a lesion distributed equally between the distal and proximal perfusion areas of the LSA. By contrast, MCA stenoses resulting in SCI are more common in patients with a lesion distributed predominantly in the distal perfusion area of LSA [42].

Of five adult patients with post-traumatic SCI, we could evaluate the dominant infarction area of the SCI in only three. In these three patients with traumatic SCI due to LSA damage [11–13], the dominant infarcation area was located in the distal perfusion area of the LSA, similar to the SCI associated with symptomatic MCA stenoses.

4. Visualization of an LSA for Identifying Perforator Damage and Vessel Wall Imaging for Detecting Dissection on an Intracranial Vessel

Although direct visualization of damage to the LSAs by trauma is difficult, assessing the injury is important when evaluating SCI after trauma. It is difficult to image the entire proximal-to-distal LSA using angiography. At present, there is no imaging technique that can display the LSAs clearly and directly in clinical settings. Ultrahigh-resolution CT is a useful method for evaluating the LSAs [43]. LSA imaging has clinical implications for differentiating between spontaneous and traumatic origins in patients with BG infarction.

Vessel Wall Imaging (VWI) may provide useful information for differentiating the etiology of SCI. VWI can provide a reliable tool for identifying intracranial vessel plaques *in vivo* [44,45]. Enhancement after gadolinium within an intracranial vessel plaque can suggest a strong association with ischemia [46]. VWI could be used to provide evidence of dissection flap with thrombi [47]. Intramural hematoma is a frequent imaging finding following cervicocerebral artery dissection and can also be imaged using VWI [48].

5. Simultaneous Occurrence of both Spontaneous Infarction and Hemorrhage in Adults

Post-traumatic SCI due to LSA damage in adults could be associated with a hemorrhage in the BG. Therefore, we reviewed the simultaneous occurrence of spontaneous infarction and hemorrhage in adults. Stroke is classified as either an ischemic and hemorrhagic stroke. Usually, a stroke will present as only one type, whereas the simultaneous development of ischemia and hemorrhage is rare. Hypertensive brain hemorrhage and lacunar infarction are developed from common damage to small perforating arteries. Hypertension is a condition commonly underlying primary intracerebral hemorrhage and lacunar infarction. However, the infarction and hemorrhage rarely occur simultaneously [49]. There are a few documented cases of infarction and hemorrhage developed simultaneously in the literature [49–52]. These patients with simultaneous infarction and hemorrhage had no trauma history but had underlying risk factors for cerebral stroke.

We observed simultaneous development of BG infarction and hemorrhage in four of five adult cases of post-traumatic BG infarction [10–13]. In adult patients, the simultaneous development of BG infarction and hemorrhage in the LSAs is important for the differentiation of traumatic or non-traumatic origins.

6. Sequential Spontaneous Occurrence of Hemorrhage and Infarction in the LSA Territory

In case of SCI infarction associated with hemorrhage in LSA territory after trauma, we should distinguish sequential spontaneous occurrence of BG hemorrhage and ischemia in the LSA perfusion area. To our knowledge, only one case of the sequential occurrence of BG hemorrhage and infarction in the LSA territory has been reported [53]. An 82-year-old woman with hypertension and no history of trauma presented with right hemiparesis due to putaminal hemorrhage. The left internal capsule and corona radiata were normal on a an initial CT scan and blood pressure medication was started. On day two, the right hemiparesis progressed to right hemiplegia. CT demonstrated no change of the hematoma volume, but the left corona radiata showed a low-density area. On day six, MR imaging showed corona radiata ischemia. Intensive blood pressure-lowering therapy may result in ischemic damage. Careful observation of neurological status and repeat imaging can suggest differentiation between spontaneous BG hemorrhage followed by infarction in the same LSA territory and trauma associated with the simultaneous development of BG infarction and hemorrhage.

7. Conclusion

Post-traumatic SCI due to LSA damage in adults might be associated with hemorrhage in the BG. Careful observation of neurological status and the use of imaging can be used to differentiate between simultaneous and sequential development of BG infarction and hemorrhage. In cases with no risk factors for stroke, simultaneous post-traumatic development of infarction and hemorrhage in the same LSA territory might suggest a traumatic origin.

Abbreviations

BG, basal ganglia; CT, computed tomography; LSA, lenticulostriate artery; MCA, middle cerebral artery; MR, magnetic resonance; SAH, subarachnoid hemorrhage; SCI, striatocapsular infarction; VWI, vessel wall imaging.

Author Contributions

JUM and MSK designed the research study, collect and sort references, wrote and revised the manuscript. YHK and MK reported similar cases and critically revised the manuscript. MSK designed this review article. All authors

contributed to editorial changes in the manuscript. All authors read and approved the final manuscript. All authors have participated sufficiently in the work and agreed to be accountable for all aspects of the work.

Ethics Approval and Consent to Participate

Not applicable.

Acknowledgment

Thanks to all the peer reviewers for their opinions and suggestions.

Funding

This research received fees for English correction and publication from National Medical Center located in Seoul, Republic of Korea.

Conflict of Interest

The authors declare no conflict of interest.

References

[1] Macpherson P, Teasdale E, Dhaker S, Allerdyce G, Galbraith S. The significance of traumatic haematoma in the region of the basal ganglia. Journal of Neurology, Neurosurgery, and Psychiatry. 1986; 49: 29–34.

[2] Lee JP, Wang AD. Post-traumatic basal ganglia hemorrhage: analysis of 52 patients with emphasis on the final outcome. The Journal of Trauma. 1991; 31: 376–380.

[3] Donnan GA, Bladin PF, Berkovic SF, Longley WA, Saling MM. The stroke syndrome of striatocapsular infarction. Brain. 1991; 114: 51–70.

[4] Dharker SR, Mittal RS, Bhargava N. Ischemic lesions in basal ganglia in children after minor head injury. Neurosurgery. 1993; 33: 863–865.

[5] Ishihara C, Sawada K, Tateno A. Bilateral basal ganglia infarction after mild head trauma. Pediatrics International. 2009; 51: 829–831.

[6] Kieslich M, Fiedler A, Heller C, Kreuz W, Jacobi G. Minor head injury as cause and co-factor in the aetiology of stroke in childhood: a report of eight cases. Journal of Neurology, Neurosurgery, and Psychiatry. 2002; 73: 13–16.

[7] Jauhari P, Sankhyan N, Khandelwal N, Singhi P. Childhood Basal Ganglia Stroke and its Association with Trivial Head Trauma. Journal of Child Neurology. 2016; 31: 738–742.

[8] Yang FH, Wang H, Zhang JM, Liang HY. Cerebral infarction after mild head trauma in children. Indian Pediatrics. 2013; 50: 875–878.

[9] Zwank MD, Dummer BW, Danielson LT, Haake BC. Lacunar stroke in a teenager after minor head trauma: case report and literature review. Journal of Child Neurology. 2014; 29: NP65–NP68.

[10] Behara BR, Mishra S, Tripathy SR, Nath PC, Jena SP, Swarnakar PK, *et al*. An unusual combination of posttrumacit ipsilateral basal ganglia infarction with contralateral hemorrhage: A rare case report and review of literature. Indian Journal of Neurotrauma. 2016; 13: 151–153.

[11] Fung C, Z'Graggen WJ, Beck J, Gralla J, Jakob SM, Schucht P, *et al*. Traumatic subarachnoid hemorrhage, basal ganglia hematoma and ischemic stroke caused by a torn lenticulostriate artery. Acta Neurochirurgica. 2012; 154: 59–62.

[12] Kim E, Kim MS, Kim Y. Vessel Wall Magnetic Resonance Imaging in a Case of Post-traumatic Multifocal Striatocapsular Hemorrhagic Infarction. Neurologia Medico-Chirurgica. 2019; 59: 191–195.

[13] Moon JU, Kim M, Kim MS. Basal ganglia infarction and hemorrhage associated with subarachnoid hemorrhage after trauma in an adult patient: A case report. Interdisciplinary Neurosurgery. 2023; 32: 101719.

[14] Brosch JR, Golomb MR. American childhood football as a possible risk factor for cerebral infarction. Journal of Child Neurology. 2011; 26: 1493–1498.

[15] Sepelyak K, Gailloud P, Jordan LC. Athletics, minor trauma, and pediatric arterial ischemic stroke. European Journal of Pediatrics. 2010; 169: 557–562.

[16] Bogousslavsky J, Regli F. Ischemic stroke in adults younger than 30 years of age. Cause and prognosis. Archives of Neurology. 1987; 44: 479–482.

[17] Baumgartle A, Wolfe L, Puri V, Moeller K, Bertolone S, Raj A. Middle Cerebral Artery Stroke as Amusement Park Injury: Case Report and Review of the Literature. Children. 2017; 4: 64.

[18] Mokri B. Traumatic and spontaneous extracranial internal carotid artery dissections. Journal of Neurology. 1990; 237: 356–361.

[19] Yonas H, Wolfson SK, Jr, Dujovny M, Boehnke M, Cook E. Selective lenticulostriate occlusion in the primate. A highly focal cerebral ischemia model. Stroke. 1981; 12: 567–572.

[20] Maki Y, Akimoto H, Enomoto T. Injuries of basal ganglia following head trauma in children. Child's Brain. 1980; 7: 113–123.

[21] Ahn JY, Han IB, Chung YS, Yoon PH, Kim SH. Posttraumatic infarction in the territory supplied by the lateral lenticulostriate artery after minor head injury. Child's Nervous System. 2006; 22: 1493–1496.

[22] Landi A, Marotta N, Mancarella C, Marruzzo D, Salvati M, Delfini R. Basal ganglia stroke due to mild head trauma in pediatric age - clinical and therapeutic management: a case report and 10 year literature review. Italian Journal of Pediatrics. 2011; 37: 2.

[23] Umansky F, Gomes FB, Dujovny M, Diaz FG, Ausman JI, Mirchandani HG, *et al*. The perforating branches of the middle cerebral artery. A microanatomical study. Journal of Neurosurgery. 1985; 62: 261–268.

[24] Martin NA, Doberstein C, Zane C, Caron MJ, Thomas K, Becker DP. Posttraumatic cerebral arterial spasm: transcranial Doppler ultrasound, cerebral blood flow, and angiographic findings. Journal of Neurosurgery. 1992; 77: 575–583.

[25] Erbayraktar S, Tekinsoy B, Acar F, Acar U. Posttraumatic isolated infarction in the territory of Heubner's and lenticulostriate arteries: case report. The Kobe Journal of Medical Sciences. 2001; 47: 113–121.

[26] Jiang W, Fu J, Chen F, Zhan Q, Wang Y, Wei M, *et al*. Basal ganglia infarction after mild head trauma in pediatric patients with basal ganglia calcification. Clinical Neurology and Neurosurgery. 2020; 192: 105706.

[27] Shaffer L, Rich PM, Pohl KRE, Ganesan V. Can mild head injury cause ischaemic stroke? Archives of Disease in Childhood. 2003; 88: 267–269.

[28] Fisher CM. Lacunar strokes and infarcts: a review. Neurology. 1982; 32: 871–876.

[29] Cho AH, Kang DW, Kwon SU, Kim JS. Is 15 mm size criterion for lacunar infarction still valid? A study on strictly subcortical middle cerebral artery territory infarction using diffusion-weighted MRI. Cerebrovascular Diseases. 2007; 23: 14–19.

[30] Jung S, Hwang SH, Kwon SB, Yu KH, Lee BC. The clinicoradiologic properties of deep small basal ganglia infarction: lacune or small striatocapsular infarction? Journal of the Neurological Sciences. 2005; 238: 47–52.

[31] Croisille P, Turjman F, Croisile B, Tournut P, Laharotte JC, Aimard G, *et al*. Striatocapsular infarction: MRI and MR angiography. Neuroradiology. 1994; 36: 430–431.
[32] Bladin PF, Berkovic SF. Striatocapsular infarction: large infarcts in the lenticulostriate arterial territory. Neurology. 1984; 34: 1423–1430.
[33] Weiller C, Ringelstein EB, Reiche W, Thron A, Buell U. The large striatocapsular infarct. A clinical and pathophysiological entity. Archives of Neurology. 1990; 47: 1085–1091.
[34] Nakano S, Yokogami K, Ohta H, Goya T, Wakisaka S. CT-defined large subcortical infarcts: correlation of location with site of cerebrovascular occlusive disease. AJNR. American Journal of Neuroradiology. 1995; 16: 1581–1585.
[35] Tatu L, Moulin T, Bogousslavsky J, Duvernoy H. Arterial territories of the human brain: cerebral hemispheres. Neurology. 1998; 50: 1699–1708.
[36] Jose J, James J. An MRI Based Ischemic Stroke Classification - A Mechanism Oriented Approach. Annals of Indian Academy of Neurology. 2022; 25: 1019–1028.
[37] Boiten J, Lodder J. Large striatocapsular infarcts: clinical presentation and pathogenesis in comparison with lacunar and cortical infarcts. Acta Neurologica Scandinavica. 1992; 86: 298–303.
[38] Levine RL, Lagreze HL, Dobkin JA, Turski PA. Large subcortical hemispheric infarctions. Presentation and prognosis. Archives of Neurology. 1988; 45: 1074–1077.
[39] Ringelstein EB, Zeumer H, Angelou D. The pathogenesis of strokes from internal carotid artery occlusion. Diagnostic and therapeutical implications. Stroke. 1983; 14: 867–875.
[40] Caplan L, Babikian V, Helgason C, Hier DB, DeWitt D, Patel D, *et al*. Occlusive disease of the middle cerebral artery. Neurology. 1985; 35: 975–982.
[41] Lee KB, Roh H, Park HK, Sung KB, Ahn MY. Analysis of the lesion distributions and mechanism of acute middle cerebral artery infarctions involving the striatocapsular region. Journal of Clinical Neurology. 2006; 2: 171–178.
[42] Bang OY, Heo JH, Kim JY, Park JH, Huh K. Middle cerebral artery stenosis is a major clinical determinant in striatocapsular small, deep infarction. Archives of Neurology. 2002; 59: 259–263.
[43] Murayama K, Suzuki S, Nagata H, Oda J, Nakahara I, Katada K, *et al*. Visualization of Lenticulostriate Arteries on CT Angiography Using Ultra-High-Resolution CT Compared with Conventional-Detector CT. AJNR. American Journal of Neuroradiology. 2020; 41: 219–223.
[44] Yang WJ, Wong KS, Chen XY. Intracranial Atherosclerosis: From Microscopy to High-Resolution Magnetic Resonance Imaging. Journal of Stroke. 2017; 19: 249–260.
[45] Mandell DM, Mossa-Basha M, Qiao Y, Hess CP, Hui F, Matouk C, *et al*. Intracranial Vessel Wall MRI: Principles and Expert Consensus Recommendations of the American Society of Neuroradiology. AJNR. American Journal of Neuroradiology. 2017; 38: 218–229.
[46] Gupta A, Baradaran H, Al-Dasuqi K, Knight-Greenfield A, Giambrone AE, Delgado D, *et al*. Gadolinium Enhancement in Intracranial Atherosclerotic Plaque and Ischemic Stroke: A Systematic Review and Meta-Analysis. Journal of the American Heart Association. 2016; 5: e003816.
[47] Atsina KB, Rothstein A, Messé SR, Song JW. Intracranial vessel wall MR imaging of an intradural vertebral artery dissection. Clinical Imaging. 2020; 68: 108–110.
[48] Ou J, Liao W, Yang S. Characteristics of high-resolution magnetic resonance vessel wall imaging of cervicocerebral artery dissection and the influential factors of vascular recanalization. Journal of Central South University. Medical Sciences. 2021; 46: 467–474. (In English, Chinese)
[49] Pande N, Vivek G, Hande M, Acharya V. Simultaneous occurrence of internal capsule infarct and cerebellar haemorrhage in a patient with hemiplegia. BMJ Case Reports. 2014; 2014: bcr2013201489.
[50] Toyoda K, Kumai Y, Fujii K, Ando T, Ibayashi S. Simultaneous onset of haemorrhagic and ischaemic strokes in a haemodialysis patient. Journal of Neurology, Neurosurgery, and Psychiatry. 2002; 72: 673–674.
[51] Balci K, Utku U, Asil T, Unlu E. Simultaneous onset of hemorrhagic and ischemic strokes. The Neurologist. 2007; 13: 148–149.
[52] Sugiyama H, Tsutsumi S, Watanabe A, Nonaka S, Okura H, Ishii H. Simultaneous presentation of subcortical hemorrhage, subdural hemorrhage, and cerebral infarct in a hemiplegic patient. Radiology Case Reports. 2022; 17: 1376–1379.
[53] Inomata Y, Hanaoka Y, Fujii Y, Ogiwara T, Horiuchi T. Sequential Development of Putaminal Hemorrhage and Corona Radiata Infarction in the Same Lenticulostriate Arterial Territory. Acta Medica Okayama. 2022; 76: 329–332.