

Simultaneous Occurrence of a Deep Ischemic Stroke and Pontine Intracerebral Hemorrhage: A Case Report and Literature Review

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Abstract:

Background: The simultaneous occurrence of ischemic stroke and spontaneous intracerebral hemorrhage is an exceptionally rare clinical presentation that poses significant diagnostic and therapeutic challenges. Although both conditions share several vascular risk factors, their coexistence in distinct vascular territories is uncommon and remains poorly understood.

Case Presentation: We report the case of a 54-year-old woman with poorly controlled arterial hypertension who presented with sudden-onset severe headache, tetraparesis, and altered alertness. Brain imaging revealed a deep left capsulolenticular acute ischemic infarction in the territory of the middle cerebral artery, associated with an acute pontine intraparenchymal hemorrhage. The diagnostic workup did not identify a cardioembolic source or another secondary cause. The simultaneous involvement of two territories supplied by perforating arteries, in the setting of severe hypertension and multiple cardiovascular risk factors, strongly suggested diffuse hypertensive cerebral small vessel disease as the underlying mechanism. Management consisted of strict blood pressure control, intensive cardiovascular risk-factor management, individualized secondary prevention, and early rehabilitation. The clinical outcome was favorable, with complete neurological recovery.

Conclusion: The coexistence of a deep ischemic infarction and a deep intracerebral hemorrhage is a rare manifestation of cerebral small vessel disease and represents a major diagnostic and therapeutic challenge. Multimodal brain imaging is essential for identifying both ischemic and hemorrhagic lesions and for guiding the etiological assessment. In the absence of specific management guidelines, treatment should be individualized according to the respective ischemic and hemorrhagic risks, with particular emphasis on strict blood pressure control and comprehensive management of vascular risk factors.

Key Word: Ischemic stroke; Intracerebral hemorrhage; Pontine hemorrhage; Cerebral small vessel disease; Hypertension; Lacunar infarction; Cerebral microangiopathy.

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I. Introduction

Stroke is one of the leading causes of mortality and disability worldwide. It can be broadly classified into two major entities: ischemic stroke, which accounts for approximately 80–85% of cases, and spontaneous intracerebral hemorrhage (ICH), which represents approximately 10–15%. Although these two forms of stroke share several cardiovascular risk factors, they differ substantially in their underlying pathophysiological mechanisms, therapeutic management, and prognosis.

The concomitant occurrence of ischemic stroke and primary intracerebral hemorrhage in the same patient is an exceptionally rare clinical situation. This association poses major diagnostic and therapeutic challenges, particularly regarding the initiation of antithrombotic therapy, which may increase the risk of hematoma expansion while being essential for the prevention of recurrent ischemic events. The underlying pathophysiological mechanisms remain incompletely understood and most commonly involve severe arterial hypertension, cerebral small vessel disease, certain forms of vasculitis, or disorders of hemostasis.

We report the case of a patient presenting with a simultaneous deep ischemic infarction in the territory of the left middle cerebral artery and a pontine intraparenchymal hemorrhage, in the setting of poorly controlled arterial hypertension. Through this case and a review of the literature, we discuss the potential pathophysiological mechanisms underlying this rare association, as well as the main diagnostic and therapeutic challenges involved in its management.

II. Case Presentation

A 54-year-old right-handed woman was admitted for a sudden-onset neurological deficit characterized by weakness of all four limbs and severe headache. Her medical history was notable for arterial hypertension diagnosed several years earlier and treated with amlodipine (5 mg/day), with poor treatment adherence. She had no history of stroke, known diabetes mellitus, or use of combined estrogen–progestin contraception. Her obstetric history included two full-term pregnancies (G2P2).

A few hours prior to admission, the patient developed sudden-onset diffuse, severe headache, associated with a sensation of heaviness in all four limbs and altered alertness. An emergency non-contrast brain computed tomography (CT) scan prompted her transfer to a university hospital. On arrival, the clinical presentation was dominated by severe hypertension requiring initial management in the intensive care unit before neurological assessment.

At admission, the patient was conscious, with a Glasgow Coma Scale score of 15/15. Her blood pressure was 170/90 mmHg, heart rate was 80 beats per minute, and oxygen saturation was 96% on room air. Her body mass index was 40 kg/m². Neurological examination revealed mild tetraparesis, more pronounced on the right side, associated with ataxia, with no impairment of higher cortical functions. The National Institutes of Health Stroke Scale (NIHSS) score was 6.

Brain magnetic resonance imaging (MRI) demonstrated an acute ischemic infarction involving the left capsulolenticular region within the deep territory of the left middle cerebral artery (MCA), appearing hyperintense on fluid-attenuated inversion recovery (FLAIR) sequences, with restricted diffusion and a decreased apparent diffusion coefficient (ADC). Brain CT also revealed a spontaneously hyperdense pontine lesion, corresponding on MRI to a T2-hypointense lesion, consistent with an acute pontine intraparenchymal hemorrhage (Figure 1A, B, C).

Laboratory investigations revealed fasting hyperglycemia of 1.65 g/L and a glycated hemoglobin (HbA1c) level of 7.5%, consistent with newly diagnosed diabetes mellitus. The lipid profile showed dyslipidemia, with a total cholesterol level of 2.37 g/L, low-density lipoprotein cholesterol (LDL-C) of 1.46 g/L, and triglycerides of 2 g/L. Renal function was preserved (urea: 0.39 g/L; creatinine: 9.68 mg/L). Electrocardiography showed regular sinus rhythm. Transthoracic echocardiography and evaluation of the supra-aortic trunks revealed no abnormalities that could account for the cerebrovascular event.

Management consisted of strict blood pressure control, secondary prevention of ischemic stroke, and intensive management of cardiovascular risk factors, particularly newly diagnosed diabetes mellitus, dyslipidemia, and obesity. Early functional rehabilitation was initiated. The clinical course was favorable, with complete neurological recovery.



Figure 1A. Axial non-contrast brain computed tomography (CT) scan showing a spontaneously hyperdense pontine lesion, consistent with an acute intracerebral hemorrhage.

Figure 1B. Axial T2-weighted brain magnetic resonance imaging (MRI) showing a hypointense pontine lesion corresponding to the acute intracerebral hemorrhage.

Figure 1C. Axial fluid-attenuated inversion recovery (FLAIR) brain MRI showing left capsulolenticular hyperintensity, consistent with an acute cerebral infarction involving the deep territory of the middle cerebral artery.

III. Discussion

The simultaneous coexistence of ischemic stroke (IS) and spontaneous intracerebral hemorrhage (ICH) represents an exceptionally rare clinical presentation. Unlike hemorrhagic transformation of a cerebral infarction, a relatively common complication of ischemic stroke, this association reflects two primary cerebrovascular events occurring concomitantly in distinct anatomical territories. Since the first case was reported in the literature, only a limited number of isolated cases and small case series have been published, highlighting the rarity of this entity and the lack of specific recommendations regarding its diagnostic and therapeutic management (1–4) (Table 1).

Table 1. Summary of published cases of concomitant ischemic stroke and spontaneous intracerebral hemorrhage.

Author	Risk factors	Ischemic stroke location	ICH location	Presumed etiology	Treatment	Outcome
Quintas et al.	Mechanical heart valve on anticoagulation	MCA occlusion	Concomitant intracerebral hemorrhage	—	Anticoagulation; mechanical thrombectomy	Favorable
Nalumansi et al.	Hypertension, atrial fibrillation	Frontoparietal infarction	Cerebellar hemorrhage	Hypertensive disease + atrial fibrillation	Conservative management	Favorable
Our case	Poorly controlled hypertension, obesity, diabetes mellitus, dyslipidemia	Left capsulolenticular infarction	Pontine hemorrhage	Probable hypertensive small vessel disease	Blood pressure control, individualized secondary prevention, rehabilitation	Complete neurological recovery

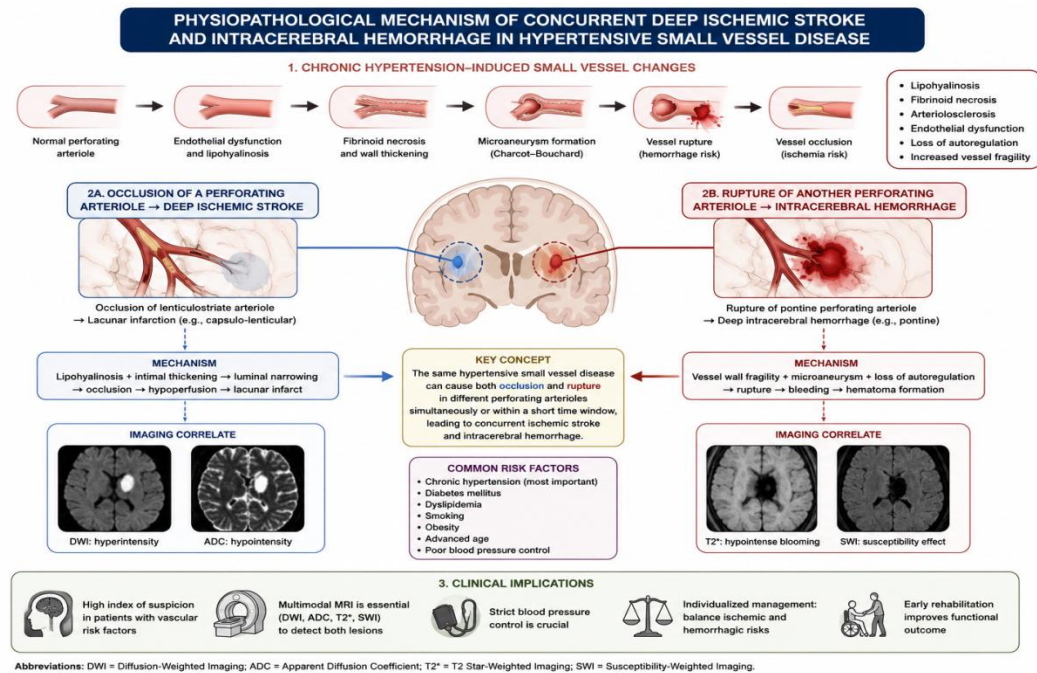
Our case describes a 54-year-old woman presenting simultaneously with a left capsulolenticular infarction in the deep territory of the middle cerebral artery and a pontine hemorrhage in the setting of severe hypertension. This presentation is particularly noteworthy because of the deep location of both lesions, which exclusively involved territories supplied by perforating arteries. The absence of evidence supporting a cardioembolic origin or another secondary etiology makes diffuse hypertensive cerebral small vessel disease particularly likely.

Chronic arterial hypertension is the main shared risk factor for lacunar infarctions and deep intracerebral hemorrhages. The histopathological changes it induces in perforating arterioles—including lipohyalinosis, fibrinoid necrosis, arteriolosclerosis, and endothelial dysfunction—result in diffuse weakening of the cerebral microcirculation (5–7). This small vessel disease may evolve through two distinct but complementary mechanisms: occlusion of a perforating artery leading to a lacunar infarction, or rupture of another structurally compromised artery resulting in intracerebral hemorrhage. Thus, ischemic and hemorrhagic manifestations may be regarded as two different expressions of the same underlying diffuse pathological process.

This hypothesis is further supported by the anatomical distribution of the lesions observed in our patient. The infarction involved the territory of the lenticulostriate arteries, whereas the hemorrhage was located in the pons, which is supplied by pontine perforating arteries. Both territories belong to the network of small perforating arteries and are particularly vulnerable to the deleterious effects of chronic hypertension. This anatomical concordance, in our view, provides an important argument in favor of diffuse hypertensive small vessel disease rather than two independent vascular events.

Nevertheless, several pathophysiological mechanisms have been proposed to explain this unusual association. In addition to hypertensive small vessel disease, central nervous system vasculitis, antiphospholipid syndrome, certain thrombophilic disorders, hemostatic abnormalities, reversible cerebral vasoconstriction syndrome (RCVS), posterior reversible encephalopathy syndrome (PRES), and cerebral amyloid angiopathy have been reported (8–11). However, in our case, the absence of clinical or laboratory evidence supporting these etiologies, together with the deep location of the lesions and the setting of severe hypertension, makes these diagnoses less likely. In particular, cerebral amyloid angiopathy predominantly affects older individuals and typically manifests with recurrent lobar intracerebral hemorrhages (Figure 1).

Figure 1. Proposed pathophysiological mechanism underlying the simultaneous occurrence of a deep cerebral infarction and deep intracerebral hemorrhage in the setting of hypertensive cerebral small vessel disease.



Our case also highlights the diagnostic challenges associated with this clinical presentation. The detection of an intracerebral hemorrhage may lead to underrecognition of a concomitant ischemic infarction, whereas identification of an acute infarction may divert attention from a concurrent hemorrhage. In this context, multimodal neuroimaging plays a crucial role. Diffusion-weighted imaging (DWI) and apparent diffusion coefficient (ADC) maps allow confirmation of the acute nature of ischemic lesions, whereas T2*-weighted or susceptibility-weighted imaging (SWI) sequences are highly sensitive for detecting blood degradation products and cerebral microbleeds (12–14). The combination of these sequences enables accurate characterization of the lesions and helps guide the etiological workup.

The therapeutic management of this association represents a major challenge. Recommendations for ischemic stroke and intracerebral hemorrhage may indeed be potentially conflicting. Secondary prevention of ischemic stroke relies on antithrombotic therapy and intensive management of vascular risk factors, whereas the presence of intracerebral hemorrhage requires particular caution regarding these treatments in order to minimize the risk of hematoma expansion or recurrent bleeding (15,16). In the absence of specific recommendations for this exceptional clinical situation, the therapeutic strategy should be individualized, taking into account the size and location of the hemorrhage, the presumed underlying mechanism, and the respective ischemic and hemorrhagic risks. In our patient, strict blood pressure control, intensive management of cardiovascular risk factors, and early rehabilitation resulted in complete neurological recovery.

Compared with the few cases previously reported in the literature, our case presents several distinctive features. First, it combines a deep capsulolenticular infarction with a pontine hemorrhage, involving two territories supplied exclusively by perforating arteries. Second, the underlying pathophysiological mechanism strongly favors diffuse hypertensive small vessel disease, with no evidence of another underlying cause. Finally, the favorable clinical outcome contrasts with the often guarded prognosis reported in previous cases, possibly owing to early recognition of both lesions and prompt management of cardiovascular risk factors.

Nevertheless, our case has several limitations. The absence of advanced vascular imaging specifically assessing the arterial wall, comprehensive thrombophilia testing, and histopathological confirmation precludes definitive establishment of the proposed mechanism. However, the consistency between the clinical context, vascular risk factors, lesion distribution, and available literature strongly supports the hypothesis of diffuse hypertensive cerebral small vessel disease.

In conclusion, the coexistence of a deep cerebral infarction and a deep intracerebral hemorrhage represents a rare clinical presentation that should raise suspicion of diffuse small vessel involvement, particularly in patients with poorly controlled arterial hypertension. This case highlights the importance of comprehensive analysis of brain imaging, a rigorous etiological workup, and an individualized therapeutic

approach. It also provides further support for the concept that deep ischemic and hemorrhagic manifestations may represent two complementary expressions of the same underlying hypertensive small vessel disease.

IV. Conclusion

The simultaneous occurrence of a deep ischemic stroke and spontaneous intracerebral hemorrhage represents an exceptionally rare clinical presentation and a major diagnostic and therapeutic challenge. This case highlights the central role of hypertensive cerebral small vessel disease, which may give rise to concomitant ischemic and hemorrhagic manifestations in distinct perforating vascular territories. It also emphasizes the value of multimodal brain imaging for identifying all lesions and guiding the etiological workup.

In the absence of specific recommendations, management should be individualized, taking into consideration the risk–benefit profile of antithrombotic therapy and rigorous control of cardiovascular risk factors, particularly arterial hypertension. Further studies are needed to better elucidate the pathophysiological mechanisms underlying this rare association and to establish appropriate therapeutic recommendations.

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