Cureus
Part of SPRINGER NATURE

Open Access Case Report

Acute Contralateral Hemibody Hyperhidrosis in Gasperini Syndrome Associated With Intracranial Vertebral Artery Dissection: A Case Report

David Fabian Ramirez Moreno [1, 2], Fabian Erazo-Amariles [1, 2], Alberto Masaru Shinchi [1, 2]

1. Neurology, Fundación Valle del Lili, Cali, COL 2. Faculty of Health Sciences, Icesi University, Cali, COL

Corresponding author: Fabian Erazo-Amariles, fabianerazo1234@gmail.com

Review began 09/18/2026
Review ended 10/24/2026
Published 10/29/2026

© Copyright 2026
Ramirez Moreno et al. This is an open access article distributed under the terms of the Creative Commons Attribution License CC-BY 4.0., which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

DOI: 10.7759/cureus.118777

Abstract

Gasperini syndrome is a rare crossed brainstem syndrome caused by lesions of the caudal pontine tegmentum, characterised by ipsilateral cranial nerve (CN) V-VIII dysfunction and contralateral hemibody hypoalgesia; fewer than 20 cases have been documented since its original description in 1912. We present a 57-year-old man who developed acute left peripheral facial palsy, left abducens palsy, left facial hypoaesthesia, left (clinically assessed) hypoacusis, and gait instability, with clinically complete ipsilateral CN V-VIII involvement. Contralateral pain and temperature and touch sensation were formally tested and intact. From the initial presentation, he exhibited prominent diaphoresis restricted to the contralateral (right) hemibody, without ipsilateral anhidrosis or Horner syndrome, and remained afebrile throughout admission. Brain MRI demonstrated an acute ischaemic infarct at the medial posterior left pontine tegmentum. Susceptibility-weighted imaging showed an eccentric crescent-shaped intramural haematoma at the left intracranial vertebral artery (V4), confirming vertebral artery dissection; digital subtraction angiography showed a long intracranial stenosis of the left vertebral artery and the right vertebral artery terminating in the posterior inferior cerebellar artery (PICA), a recognised anatomical variant. To our knowledge, contralateral hemibody hyperhidrosis has not previously been reported in Gasperini syndrome. We propose, as an anatomical hypothesis rather than a demonstrated mechanism, that it reflects disruption of crossed sympatho-inhibitory sudomotor fibres in the medial/paramedian pontine tegmentum, with sparing of the dorsolateral excitatory and oculosympathetic pathways, concordant with the medial lesion topography and the absence of Horner syndrome.

Categories: Neurology, Radiology, Emergency Medicine
Keywords: abducens nerve palsy, autonomic nervous system dysfunction, brainstem syndrome, contralateral hyperhidrosis, facial colliculus, gasperini syndrome, intracranial vertebral artery dissection, pontine infarct, posterior circulation stroke, sudomotor dysfunction

Introduction

Gasperini syndrome (GS) is one of the rarest crossed brainstem syndromes in clinical neurology, with fewer than 20 cases documented in the indexed literature. Described by the Italian internist Ubaldo Gasperini in 1912, it results from a lesion of the caudal pontine tegmentum and is classically defined by ipsilateral cranial nerve (CN) V, VI, VII, and VIII dysfunction combined with contralateral hemibody hypoalgesia. Bhagat et al. reviewed the published cases and proposed a revised, more inclusive definition requiring ipsilateral CN VI and VII palsies plus at least one additional core feature [1,2].

The lesion disrupts multiple structures packed within the caudal pontine tegmentum: the facial nucleus and its genu coursing around the abducens nucleus, the spinal trigeminal nucleus and tract, the lateral lemniscus, and the spinothalamic tract conveying contralateral pain and temperature sensation [2,3].

The syndrome may be accompanied by autonomic phenomena - Vesza et al. reported a case associated with ipsilateral trigemino-autonomic headache, attributed to trigeminovascular activation with secondary autonomic accompaniments - but, to our knowledge, sudomotor dysfunction has not previously been described as a feature of GS [4].

We report a case of GS associated with left intracranial vertebral artery dissection occurring on a background of a contralateral posterior inferior cerebellar artery (PICA)-terminating vertebral artery variant, with the novel finding of prominent contralateral hemibody hyperhidrosis as an acute autonomic manifestation, expanding the known clinical spectrum of this syndrome.

Case Presentation

A 57-year-old man with no prior medical history and no known vascular risk factors presented to the emergency department after acute-onset left peripheral facial palsy and gait instability beginning the previous evening. He reported a morning headache and elevated blood pressure the preceding day. On arrival, blood pressure was 183/95 mmHg, heart rate 95 bpm, oxygen saturation 97%, and temperature

36.0°C; he remained afebrile throughout admission (recorded temperatures 35.5-36.7°C).

Neurological examination revealed left abducens palsy (CN VI), left peripheral facial palsy (CN VII) with lagophthalmos, left facial hypoaesthesia (CN V), left clinically assessed hypoacusis (CN VIII), and mild dysarthria. Limb strength was 5/5 in all four limbs with no long-tract motor deficit; the Romberg sign was positive with mild dysmetria (side not specified in the record). Pain, temperature, and touch were formally tested over the trunk and limbs bilaterally and were symmetric and intact.

At initial examination, the patient exhibited prominent diaphoresis restricted to the right (contralateral) hemibody, involving the face, neck, trunk, and limbs, without ipsilateral anhidrosis, ptosis, or miosis. The asymmetry was identified at the bedside by inspection and palpation - the right hemibody was visibly and palpably diaphoretic while the left was dry - was documented by the emergency physician and by the treating team and had improved by the sixth day. As the patient remained afebrile, thermoregulatory (febrile) sweating was excluded; secondary causes of unilateral hyperhidrosis were considered unlikely given the hemibody (non-segmental) distribution, the absence of Horner syndrome, and the absence of any local cutaneous or structural cause.

The combination of clinically complete ipsilateral CN V-VIII involvement satisfied the revised diagnostic criteria for GS proposed by Bhagat et al.; contralateral hemibody hypoalgesia, part of the original description, was absent [1]. Among the differential diagnoses, Foville and Millard-Gubler syndromes characteristically extend ventrally into the basis pontis and produce contralateral hemiparesis, which was absent here, while lateral inferior pontine (anterior inferior cerebellar artery, AICA) syndrome overlaps clinically. These entities overlap, and lateral pontine infarction can produce variable abducens/facial combinations depending on lesion extent; the diagnosis therefore rested on lesion localisation and the overall clinical-anatomical constellation rather than on any single discriminating sign.

Investigations

Emergency brain MRI showed a focus of restricted diffusion at the medial posterior left pontine tegmentum, consistent with an acute ischaemic infarct (Figure *1A*); the lesion topography was medial rather than the classic lateral tegmental location of AICA-territory infarction. Susceptibility-weighted imaging demonstrated an eccentric, crescent-shaped intramural haematoma within the wall of the left V4 segment, reviewed and confirmed by neuroradiology - a specific sign of intracranial vertebral artery dissection (Figure *1B*). Initial time-of-flight MR angiography did not identify the vertebral lesion.

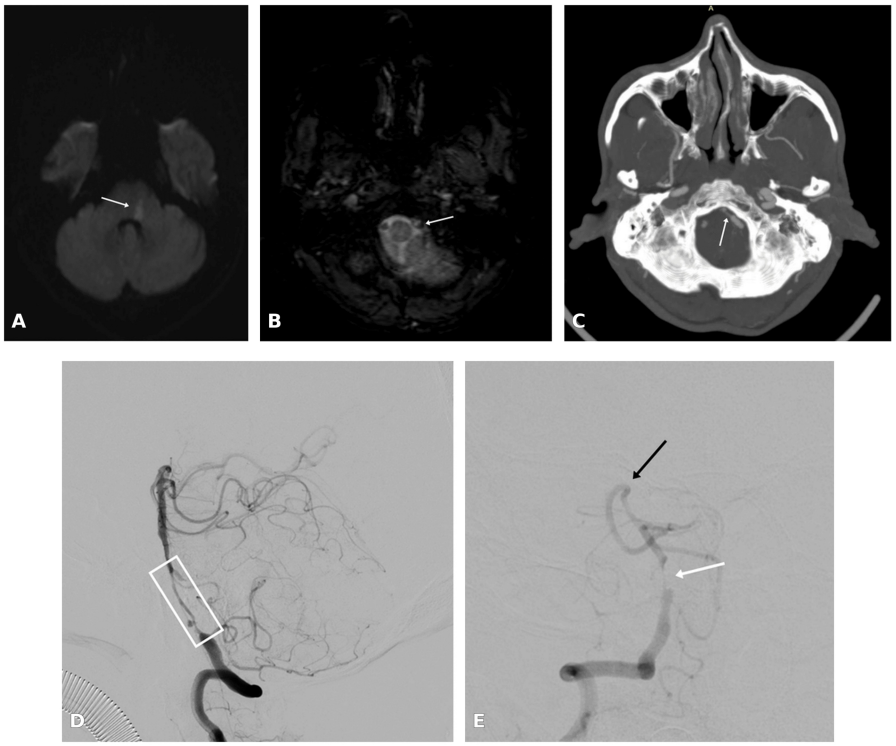


FIGURE 1: Neuroimaging in Gasperini syndrome with contralateral hemibody hyperhidrosis

(A) Axial diffusion-weighted MRI: focal restricted diffusion in the medial posterior left pontine tegmentum (arrow), consistent with an acute ischaemic infarct. (B) Axial susceptibility-weighted MRI: eccentric, crescent-shaped intramural haematoma within the wall of the left intracranial vertebral artery (V4 segment) (arrow), confirmed by neuroradiology—a specific sign of intracranial vertebral artery dissection. (C) Axial CT angiography: wall irregularity of the left vertebral artery at the V4 segment (arrow). (D) Digital subtraction angiography, left vertebral artery injection: long intracranial stenosis of the V4 segment proximal to the vertebrobasilar junction (box). (E) Digital subtraction angiography, right vertebral artery injection: the right vertebral artery terminates in the posterior inferior cerebellar artery (white arrow) without continuation to the basilar artery (black arrow), consistent with a PICA-terminating vertebral artery variant.

PICA: posterior inferior cerebellar artery

CT angiography showed wall irregularity of the left vertebral artery at the intracranial V4 segment, measuring approximately 2 cm on source images (Figure *1C*), and described the right vertebral artery as terminating in the PICA. Only non-stenosing fibrocalcific plaque was present at the left carotid bulb, with no significant stenosis of the extracranial vertebral arteries; a small incidental saccular aneurysm (dome 1 mm, neck 2 mm) at the clinoid segment of the left internal carotid artery was considered an unrelated incidental finding.

Digital subtraction angiography demonstrated a long intracranial stenosis of the left vertebral artery (V4) proximal to the vertebrobasilar junction (Figure *1D*), with the vertebrobasilar system otherwise normal and the posterior cerebral arteries filling through the posterior communicating arteries. The right vertebral artery terminated in PICA without continuation to the basilar artery, consistent with a PICA-terminating vertebral artery variant rather than an acquired occlusion (Figure *1E*). The diagnosis of intracranial vertebral artery dissection was based on the intramural haematoma (crescent sign) on susceptibility-weighted imaging together with the V4 luminal abnormality, in a patient without atherosclerotic risk factors. Transoesophageal echocardiography showed no cardioembolic source and an interatrial septal aneurysm without interatrial shunt on agitated-saline contrast. Laboratory investigations were unremarkable.

Management and outcome

Given the absence of a large-vessel occlusion amenable to reperfusion, thrombolysis and thrombectomy were not indicated. Secondary prevention was initiated with dual antiplatelet therapy (aspirin 100 mg and clopidogrel 75 mg daily for 21 days) and high-intensity statin therapy (atorvastatin 40 mg), alongside antihypertensive treatment (valsartan 80 mg twice daily); the patient was admitted to the intensive care unit for haemodynamic and neurological monitoring. After a six-day hospital stay, he was discharged for

multidisciplinary rehabilitation, with an outpatient neurology review scheduled at one month; at discharge, the facial and abducens palsies persisted, while the contralateral hemibody diaphoresis had improved. No follow-up vascular imaging was obtained.

Discussion

This case presents three features that, to our knowledge, have not previously been described together in GS: (1) contralateral hemibody hyperhidrosis as an acute autonomic finding; (2) a medial pontine tegmentum topography; and (3) its occurrence in the setting of intracranial vertebral artery dissection with a contralateral PICA-terminating vertebral artery variant.

Contralateral hyperhidrosis: a new autonomic dimension of GS

The most distinctive finding is contralateral hemibody hyperhidrosis, an autonomic manifestation not previously documented in GS. Contralateral hyperhidrosis has been reported after cerebral infarction and in other brainstem lesions [5]: Pellecchia et al. described pure contralateral hemihyperhidrosis from a lacunar upper pontine infarct, but as an isolated chronic complaint without acute deficits [6]; Rousseaux et al. described it after lateral medullary infarction, but delayed by six to eight months [7]; and Sato and Nitta reported it in Foville syndrome after a lower pontine haemorrhage, appearing about one month after onset and resolving within a week [8]. In contrast, in our patient, the sudomotor asymmetry was present from the initial presentation as part of a complete acute pontine syndrome. The defensible novelty is therefore its occurrence within GS specifically and its acute onset, rather than the phenomenon of contralateral hyperhidrosis itself.

Two descending sudomotor pathways have been characterised: a primary ipsilateral excitatory hypothalamospinal tract in the dorsolateral tegmentum, and a secondary crossed sympatho-inhibitory pathway that descends through the brainstem and is thought to decussate in the medulla, though it remains incompletely characterised [5,9,10]. In our patient, the medial posterior pontine lesion is anatomically consistent with disruption of this crossed inhibitory pathway while sparing the dorsolateral excitatory and oculosympathetic pathways, concordant with the absence of ipsilateral anhidrosis and Horner syndrome. We emphasise that no objective sudomotor testing (thermoregulatory sweat test or quantitative sudomotor axon reflex test) was performed; the proposed mechanism therefore remains an anatomical inference rather than a physiologically demonstrated finding. We further note that the preserved contralateral pain and temperature sensation is compatible with, but does not prove, anatomical separability of the sudomotor and spinothalamic fibres, since the infarct may have been small enough to spare the spinothalamic tract entirely.

Medial topography: an atypical lesion site

Classical GS involves the lateral pontine tegmentum supplied by the long circumferential branch of AICA, yet our patient showed a medial posterior lesion. This topography is most consistent with a paramedian or median basilar perforating branch; this culprit-branch localisation is an anatomical inference based on lesion location and the known perforator classification, not an established clinical frequency in GS [11]. It demonstrates that medially situated infarcts can produce the full GS, consistent with the spectrum described by Bhagat et al. [1].

Vascular aetiology: dissection on a background of a vertebral variant

Spontaneous cervical (extracranial) artery dissection accounts for 10-25% of ischaemic strokes in young and middle-aged adults; these figures pertain to cervical/extracranial dissection, whereas isolated intracranial V4 dissection, as in our patient, is considerably less common and less well characterised [12]. The medial posterior pontine infarct lay within a basilar perforator territory; in arterial dissection both haemodynamic compromise and artery-to-artery embolism are recognised mechanisms of ischaemic stroke [13,14]. On digital subtraction angiography, the stenosis lay in the distal left V4 proximal to the vertebrobasilar junction with the vertebrobasilar system otherwise normal; we, therefore, regard the link between the vertebral lesion and the infarct as probable, with haemodynamic compromise, artery-to-artery embolism and intrinsic basilar perforator disease all possible and not distinguishable on the available imaging. The dissection occurred on a background of a contralateral PICA-terminating vertebral artery, a recognised anatomical variant that reduces basilar inflow reserve.

Limitations

This report has several limitations. Objective sudomotor testing and formal mapping of the midline sweating border were not performed; hearing loss was assessed clinically without audiometry; and House-Brackmann grading, taste, lacrimation, and Weber/Rinne testing were not formally documented in the acute setting. The stroke mechanism could not be established with certainty, and no follow-up vascular imaging was obtained. As a single case, these observations are hypothesis-generating.

In any acute pontine syndrome, bedside examination should actively seek sudomotor asymmetry; acute contralateral hemibody hyperhidrosis without ipsilateral Horner syndrome may be a useful localising sign that should prompt comprehensive neurovascular imaging, including evaluation for vertebral artery

dissection even in the absence of cervical pain.

Conclusions

This case broadens the recognised clinical spectrum of GS. The observed findings were acute contralateral hemibody hyperhidrosis without ipsilateral Horner syndrome in a patient meeting the revised diagnostic criteria, a medial posterior pontine infarct, a left V4 vertebral artery dissection confirmed by an intramural haematoma on susceptibility-weighted imaging, and a contralateral PICA-terminating vertebral artery variant. As a proposed rather than demonstrated mechanism, the hyperhidrosis is anatomically concordant with disruption of crossed sympatho-inhibitory sudomotor fibres in the medial/paramedian tegmentum, with sparing of the dorsolateral excitatory and oculosympathetic pathways. Medially situated pontine infarcts can produce the Gasperini phenotype, extending its topographic spectrum beyond the classical lateral AICA territory.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Fabian Erazo-Amariles, Alberto Masaru Shinchi

Acquisition, analysis, or interpretation of data: Fabian Erazo-Amariles, David Fabian Ramirez Moreno

Drafting of the manuscript: Fabian Erazo-Amariles, David Fabian Ramirez Moreno

Critical review of the manuscript for important intellectual content: Alberto Masaru Shinchi, David Fabian Ramirez Moreno

Supervision: Alberto Masaru Shinchi

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

Acknowledgements

The Claude language model (Anthropic, San Francisco, California) was used to assist with literature verification, critical revision, and language editing during the preparation of this manuscript. All content was reviewed and verified against primary sources by the authors, who take full responsibility for its accuracy. No AI tool was used to generate or modify any image.

References

1. Bhagat R, Narayanan S, Elnazeir M, et al.: Gasperini syndrome: a case report and systematic review and proposing a new definition. Case Rep Neurol. 2020, 12:433-9. 10.1159/000510845
2. Hayashi-Hayata M, Nakayasu H, Doi M, Fukada Y, Murakami T, Nakashima K: Gasperini syndrome, a report of two cases. Intern Med. 2007, 46:129-33. 10.2169/internalmedicine.46.1714
3. Stenitzer BS, Fruehwirth-Kaspar D, Brandner M: Gasperini syndrome: a rare brainstem syndrome - a case report. Case Rep Ophthalmol. 2026, 17:402-7. 10.1159/000551798
4. Vesza Z, Várallyay G, Szoke K, et al.: Trigemino-autonomic headache related to Gasperini syndrome . J Headache Pain. 2010, 11:535-8. 10.1007/s10194-010-0251-y
5. Kim BS, Kim YI, Lee KS: Contralateral hyperhidrosis after cerebral infarction: clinicoanatomic correlations in five cases. Stroke. 1995, 26:896-9. 10.1161/01.str.26.5.896
6. Pellecchia MT, Criscuolo C, De Joanna G, D'Amico A, Santoro L, Barone P: Pure unilateral hyperhidrosis after pontine infarct. Neurology. 2003, 61:1305. 10.1212/wnl.61.9.1305
7. Rousseaux M, Hurtevent JF, Benaim C, Cassim F: Late contralateral hyperhidrosis in lateral medullary infarcts. Stroke. 1996, 27:991-5. 10.1161/01.str.27.5.991
8. Sato K, Nitta E: Pontine hemorrhage presenting with Foville syndrome and transient contralateral hyperhidrosis (Article in Japanese). Rinsho Shinkeigaku. 2000, 40:271-3.
9. Minota K, Coon EA, Benarroch EE: Neurologic aspects of sweating and its disorders . Neurology. 2019, 92:999-1005. 10.1212/WNL.0000000000007540
10. Saito H: The location and characteristics of the thermal sudomotor pathways in the human brainstem: a

reappraisal. Auton Neurosci. 2019, 217:80-90. 10.1016/j.autneu.2019.01.006
11. Kwiatkowska M, Rzepliński R, Ciszek B: Anatomy of the pontine arteries and perforators of the basilar artery in humans. J Anat. 2023, 243:997-1006. 10.1111/joa.13927
12. Schievink WI: Spontaneous dissection of the carotid and vertebral arteries . N Engl J Med. 2001, 344:898-906. 10.1056/NEJM200103223441206
13. Dejakum B, Kiechl S, Knoflach M, Mayer-Suess L: A narrative review on cervical artery dissection-related cranial nerve palsies. Front Neurol. 2024, 15:1364218. 10.3389/fneur.2024.1364218
14. Debette S, Mazighi M, Bijlenga P, et al.: ESO guideline for the management of extracranial and intracranial artery dissection. Eur Stroke J. 2021, 6:39-88. 10.1177/23969873211046475