Annales Africaines de Médecine
Cas clinique

Pulmonary embolism discovered after post-traumatic cardiorespiratory arrest complicated by ischaemic stroke in a young footballer: a rare case report

Embolie pulmonaire découverte après un arrêt cardiorespiratoire post-traumatique compliqué d'un accident vasculaire cérébral ischémique chez un jeune footballeur : une rare observation clinique

Thiombiano Lamoudi.Prisca[1], Nacanabo Wendlassida Martin[1], Konaté Lassina[2], Ouedraogo Bertille[3], Lengani Ella[1], Taryètba André Arthur Seghda[1], Anna Tall/Thiam[1], Samadoulougou Koudnoaga André[1]

Corresponding autor
Nacanabo Wendlassida Martin
Courriel : nacmartinwend@gmail.com
Bogodogo University Hospital Cardiology Department, Burkina Faso

Résumé

L'association entre l'embolie pulmonaire (EP) et les accidents vasculaires cérébraux (AVC) continue de poser un défi diagnostique et thérapeutique aux cliniciens. Nous rapportons le cas d'un jeune footballeur présentant une EP concomitante avec un AVC ischémique, compliqué par un remodelage hémorragique. C'était un footballeur professionnel de 22 ans sans antécédents médicaux qui, apparemment, est entré en collision avec un joueur de l'équipe adverse alors qu'il tentait de récupérer le ballon, avant de tomber et de perdre immédiatement connaissance, avec arrêt cardiorespiratoire. Un massage cardiaque immédiat a permis de rétablir l'activité cardiaque et circulatoire. L'examen clinique après la reprise de l'activité cardiorespiratoire a noté un score de 4/15 sur l'échelle de Glasgow, une dyspnée et des signes de motoneurones supérieurs. Le scanner cérébral était normal. Après 2 jours, le scanner thoracique a révélé une embolie pulmonaire. L'imagerie par résonance magnétique (IRM) réalisée 2 semaines après le traumatisme a plutôt conclu à une transformation hémorragique, d'un AVC ischémique temporal droit. Le patient a été traité par héparine de bas poids moléculaire, puis par un antagoniste de la vitamine K, avec un rétablissement quasi normal de la fonction motrice au bout d'un mois. La découverte d'une EP et d'un accident vasculaire cérébral ischémique après un arrêt cardiaque est un événement rare. La survenue d'une transformation hémorragique augmente le risque d'hémorragie intracrânienne et restreignant le recours à l'anticoagulation dans le contexte d'une thrombose évidente.

Mots-clés : arrêt cardiaque, accident vasculaire cérébral ischémique, remodelage hémorragique, embolie pulmonaire, Ouagadougou
Reçu le 11 octobre 2025

Accepté le 25 mars 2026

Summary

The association between pulmonary embolism (PE) and cerebrovascular events continues to pose a diagnostic and therapeutic challenge for clinicians. We report a case of concomitant PE and ischaemic stroke complicated by haemorrhagic remodelling in a young footballer. The patient was a 22-year-old professional footballer with no history of medical problems who apparently collided with a player from the opposing team while attempting to recover the ball, followed by a fall and immediate loss of consciousness with cardiorespiratory arrest. Immediate cardiac massage restored cardiac and circulatory activity. Clinical examination after recovery of cardiorespiratory activity revealed a Glasgow Coma Scale score of 4/15, dyspnoea and upper motor neuron signs. The cerebral scan was normal whereas a CT pulmonary angiography scan carried out 2 days later showed a PE. Magnetic Resonance Imaging (MRI) performed 2 weeks after the trauma revealed hemorrhagic transformation of a right temporal ischaemic stroke. The patient was treated with low-molecular-weight heparin followed by vitamin K antagonist with near-normal recovery of motor function at 1 month. The discovery of a PE and an ischaemic stroke following a cardiac arrest is an uncommon event. The occurrence of haemorrhagic transformation increases the risk of intracranial haemorrhage and complicates anticoagulation in the context of obvious thrombosis.

Keywords: cardiac arrest, ischaemic stroke, haemorrhagic remodelling, pulmonary embolism, Ouagadougou
Received October 11, 2025

Accepted March 13, 2026

https://dx.doi.org/10.4314/aamed.v19i3.16

Annales Africaines de Médecine
Cas clinique
https://dx.doi.org/10.4314/aamed.v19i3.16

1. Bogodogo University Hospital Cardiology Department
2. Tengandogo University Hospital Cardiology Department
3. Medical Imaging Department, Tengandogo University Hospital.

Introduction

Regular physical exercise is highly beneficial for cardiovascular health (1). Physical exercise is thought to reduce the risk of cardiovascular events (2). However, some authors have put forward the hypothesis that chronotropic incompetence during repeated physical exercise increases the risk of cardiovascular events, particularly sudden cardiac arrest, which is responsible for 75% of sudden deaths during physical exercise (3). The exact aetiology of this cardiac arrest is most often not found and requires an autopsy in the event of death (4). Several theories maintain that sudden cardiac arrest is most often the result of an acute rhythm disturbance. However, this hypothesis is not universally accepted. We report a case of post-traumatic cardiorespiratory arrest whose aetiological development led to the discovery of a pulmonary embolism and a stroke.

Case presentation

This case involved a 22-year-old professional footballer with no history of cardiovascular or thromboembolic risk factors. During a match, he was struck in the forehead by a player from the opposing team while attempting to retrieve the ball, followed by a fall with immediate loss of consciousness and cardiorespiratory arrest. He received cardiac massage on the pitch (for approximately 12 minutes), with resumption of cardiorespiratory activity. He was then transferred to the local level 3 health centre. Initial clinical examination revealed a blood pressure of 136/86 mmHg, impaired consciousness (Glasgow score = 4/15) and right hemiplegia. The rest of the physical examination was normal. The initial electrocardiogram and the cerebral scan were normal (figure 1).

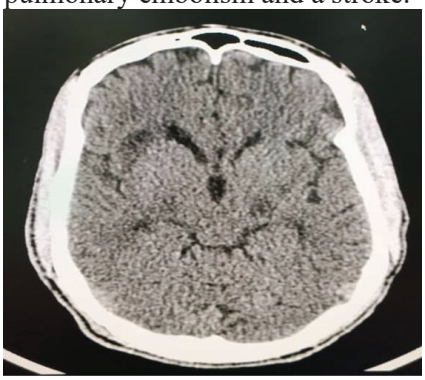

Figure 1. Normal brain scan

Transthoracic ultrasound revealed spontaneous right intracavitary contrast (figure 2). After 2 days, consciousness improved (Glasgow score=12/15), but hemiplegia and dyspnoea persisted. He was referred to the University Hospital for further management.

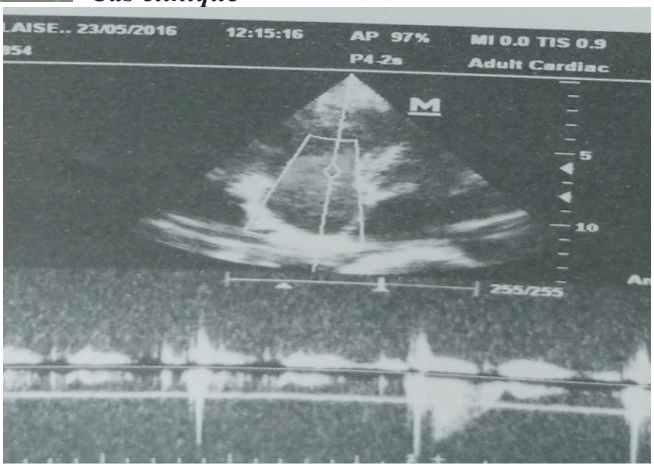

Figure 2. Cardiac ultrasound showing a spontaneous contrast in the right ventricle

A new ECG shows regular sinus tachycardia at 112 bpm with incomplete right bundle branch block. Transthoracic ultrasound confirmed the presence of a right intracavitary thrombus extending to the inferior vena cava (figure 3).

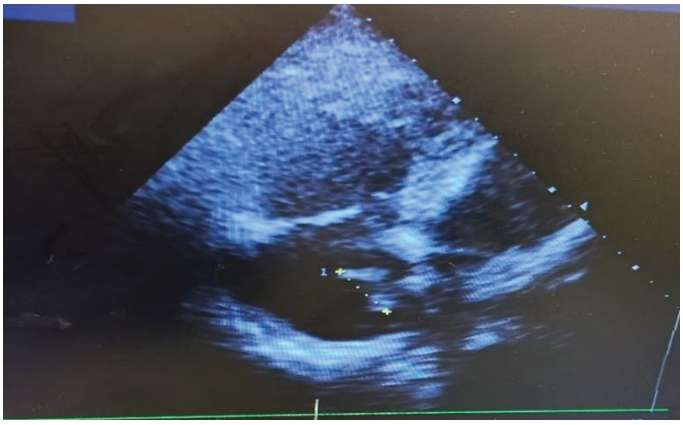

Figure 3. Heart ultrasound showing a thrombus in the right atrium

The CT pulmonary angiography scan showed a gap in the lumen of a segment of the right inferior lobar artery, suggestive of segmental pulmonary embolism (figure 4).

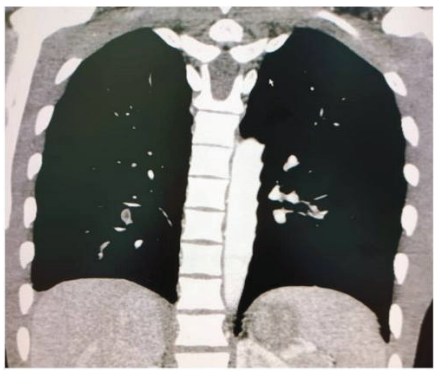

Figure 4. CT pulmonary angiography scan showing pulmonary embolism

Brain MRI (after 2 weeks) showed a circumscribed right insular, oval-center, right fronto-parietal hematoma, with diffuse haemorrhagic suffusions associated with a semi-latero-protuberantial and left mesencephalic infarct extending to the left thalamus. There were no vascular abnormalities (figure 5).

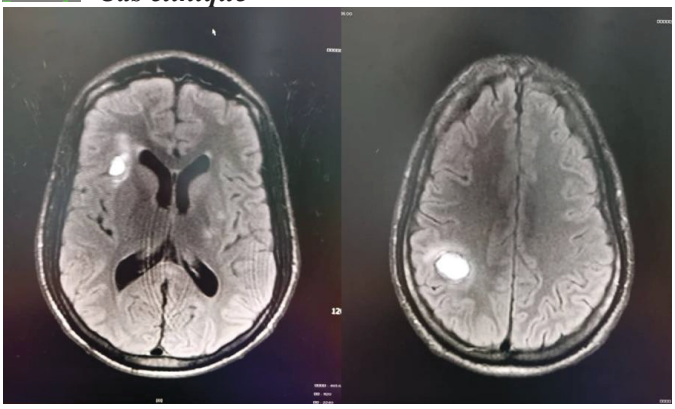

Figure 5. Brain MRI showing AVCI with haemorrhagic remodelling

Doppler echocardiography of the lower limbs to check for venous thrombosis was normal. Biologically, the blood count was normal, and retroviral serology was negative. The etiological work-up for coagulopathy performed remotely from anti-vitamin K treatment was almost normal, with fibrinogen at 3.82g/l (Normal value: 2-4), Protein C at 53% (Normal value: 80-130), low Protein S at 12% (Normal value: 70-130) and no resistance to activated protein C, with a ratio of 4.1 (Normal value: >2.2). There was no factor V Leiden or prothrombin gene mutation, and antiphospholipid antibodies were normal (at 2Uml/L normal <7). The electroencephalogram showed bifocal parieto-temporal and frontal epilepsy, probably lesional.

The diagnosis was segmental PE associated with ischaemic stroke complicated by haemorrhagic denial. The patient was treated with an anticoagulant (low-molecular-weight heparin + vitamin K antagonist), an antidepressant (Paroxetin), a muscle relaxant (Baclofen), an anticonvulsant (Phenobarbital) and physiotherapy. The in-hospital evolution after one month was marked by a stabilization of the Normal International Ratio between 2-3, the occurrence of convulsions, a progressive recovery of motricity, an almost total resorption of bleeding on the CT-scan. Virtually complete recovery of motricity was observed after 12 months.

Discussion

The incidence of sudden cardiac arrest during exercise is 0.6 to 2.1 per 100,000/year (4). Sudden Cardiac Arrest occurs most frequently in young people, due to their hyperactivity. Several previous studies have found that men dominate the ranks of victims of exercise-induced sudden cardiac events (5). The most common aetiologies of sudden cardiac death are ischemic heart disease. Coronary heart diseases are responsible for serious rhythm disorders, which may be accompanied by syncope due to sudden cerebral hypoperfusion. Tokula *et al.* found that over 77% of theses cardiac rythm disorders were non-shockable on arrival at the emergency department (1). In our patient, the electrocardiogram on arrival showed sinus rhythm. The admission Transthoracic Doppler echocardiography revealed spontaneous right intracavitary contrast. No permeable oval foramen was found, which could justify a paradoxical embolic ischemic stroke. This mechanism was found in around 15.30% of cases in the study by Le Moigne *et al.* in 2017 (6). A transoesophageal echocardiogram was not performed because it was not available at our center at the time but could have helped establish the link between the pulmonary embolism and the stroke.

Cardiac massage is thought to be responsible for the stasis and clot formation that cause ischemic stroke (1). This would account for the motor deficit present as soon as cardiorespiratory activity is resumed. Ischemic stroke can then be complicated by haemorrhagic stroke. Indeed, Kyle D. Klingbeil *et al.* found 8.7% cases of haemorrhagic transformation after ischemic stroke in 2021 (7).

Furthermore, cerebral MRI does not reveal any vascular anomalies such as aneurysms that could explain the occurrence of intra-parenchymal hematoma. The hematoma could therefore have been caused by the head injury, especially as it was located in a different territory from the ischaemic stroke. This hypothesis remains unlikely, however, as in this case the haemorrhage would be found on the initial brain scan.

Another pathophysiological hypothesis would be vagal shock secondary to the trauma or related to chronotropic incompetence, which leads to disruption of autonomic control of blood pressure (8). This leads to a significant rise in Blood Pressure, which may be the cause of ischemic stroke. The ensuing bed rest is thought to be at the origin of deep vein thrombosis and pulmonary embolism (8). In this case, at least one hypertension could have been noted. In addition, the patient had low levels of protein C and S, which could explain the occurrence of thrombosis. These abnormalities are rare but can lead to thromboembolic complications (9). These are usually heterozygous deficiencies, with protein S levels generally between 40 and 60% (10). However, clinical expression may vary between carriers of the deficiency in the same family, with asymptomatic cases or, conversely, subjects with recurrent thrombosis.

In our case, the therapeutic challenge is to choose the right treatment for the joint management of ischemic and haemorrhagic stroke associated with pulmonary embolism. To the best of our knowledge, there are few recommendations on the management of this combination. Management in this case is multidisciplinary, and should take place in a neurovascular unit. The prevention of epileptic seizures is also justified by the same recommendations. In the event of thrombus in the right cavities, a number of treatments can be adopted, including surgical thrombectomy, thrombolysis and anticoagulation. However, the therapeutic indication will depend on the underlying aetiology and context (9). In our clinical case, surgical thrombectomy was the most appropriate treatment, as thrombolysis was contraindicated in this context of cerebral haemorrhage. However, as surgical thrombectomy is not currently available in our country, we settled for curative anticoagulation. Anticoagulation in the event of haemorrhage must be carefully guided.

Conclusion

The occurrence of multiple thromboses (PE and ischemic stroke) requires a meticulous etiological search. Although rare in our context, congenital deficiencies (Proteins C; S) should be investigated in families, as their clinical expression varies within the same family, with asymyomatic cases sometimes occurring. This observation shows that, while positive diagnosis is often easy to make, etiological research sometimes comes up against technical difficulties in the context of countries with limited resources.

Conflict of interest

We have no conflict of interest.

Data availability statement

The data that support the findings of this study are available from the corresponding author upon reasonable request.

Consent

We have obtained the patient's consent for publication. Written informed consent was obtained from the patient to publish this report in accordance with the journal's patient consent policy.

Contribution of authors

Thiombiano Lamoudi Prisca: data curation, conceptualisation, manuscript writing, proofreading, submission,
Martin Wendlassida Nacanabo: data curation, conceptualisation, manuscript writing, proofreading, submission
André Arthur Taryètba Seghda: data curation
Konaté Lassina: data curation
Ouedraogo Bertille: data curation
Luingani Ella: data curation
Anna Tall/Thiam: supervision, validation
André Koudnoaga Samadoulougou: supervision, validation
All authors read and approved the final version of the manuscript.

References

1. Tokula TM, Kauppila JP, Pakanen L, Kortelainen M-L, Martikainen M, Huikuri HV, *et al.* Characteristics and Prognosis of Exercise-Related Sudden Cardiac Arrest. *Front Cardiovasc Med.* 2018;**5**:102.
2. Leonard A. Kaminskya B, Charles Germanc, MaryImbodend, CemalOzemekb, James E. Petermana and al. The importance of healthy lifestyle behaviors in the prevention of cardiovascular disease, *Progress in Cardiovascular Diseases* 2022;**70**:8-15.
3. Asatryan B, Vital C, Kellerhals C, Medeiros-Domingo A, Gräni C, Trachsel LD, *et al.* Sports-related sudden cardiac deaths in the young population of Switzerland. *PloS One.* 2017 ;**12** (3) : e0174434.
4. Berdowski J, De Beus MF, Blom M, Bardai A, Bots ML, Doevendans PA, *et al.* Exercise-related out-of-hospital

cardiac arrest in the general population: incidence and prognosis. *Eur Heart J*. 2013;**34** (47):3616-3623.
5. Philipp Bohm, Tim Meyer, Kumar Narayanan, Matthias Schindler, Orianne Weizman, Frankie Beganton, *et al.* Sports-related sudden cardiac arrest in young adults, *Europace* 2023;2**5** (2):627-633.
6. Namaganda P, Nakibuuka J, Kaddumukasa M. Stroke in young adults, stroke types and risk factors: a case control study. *BMC Neurol* 2022;**22**: 335. https://doi.org/10.1186/s12883-022-02853.
7. Klingbeil KD, Koch S, Dave KR. Potential link between post-acute ischemic stroke exposure to hypoglycemia and hemorrhagic transformation. *Int J Stroke Off J Int Stroke Soc*. 2020;**15** (5):477-483.
8. Kim D-I, Tan CO. Alterations in autonomic cerebrovascular control after spinal cord injury. *Auton Neurosci Basic Clin*. 2018;**209**:43-50.
9. Zou C, Li T, Long L, Liu L, Zhu J. Hereditary protein C deficiency with portal vein thrombosis in a Chinese male: A case report. *Exp Ther Med.* 2022;**24** (6):751.
10. Guermazi S, Conard J. Congenital protein S deficiencies; diagnostic difficulties. *Pathol Biol* (Paris). 2009;**57**(6):483-487.

Cite this article as**:** Thiombiano PL, Nacabano MW, Konaté L, Ouedraogo B, Ella L, Taryètba AAS*, et al*. Pulmonary embolism discovered after post-traumatic cardiorespiratory arrest complicated by ischaemic stroke in a young footballer: a rare case report. *Ann Afr Med* 2026; **19** (3): e7173-e7178. https://dx.doi.org/10.4314/aamed.v19i3.16