

Incidental diagnosis of an acute cerebrovascular event on 99mTc-HMDP bone scintigraphy performed due to suspicion of amyloidosis

Diagnóstico incidental del evento cerebrovascular agudo mediante gammagrafía con 99mTc-HMDP realizada por sospecha de amiloidosis

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Summary
Acute cerebrovascular event (ACE) is a neurological deficit caused by a central nervous system (CNS) injury secondary to insufficient supply of oxygenated blood to the brain. It can be hemorrhagic or ischemic, with the latter being more common. It affects approximately 16 million people worldwide each year, and in Latin America, an incidence of 35-183 per 100,000 habitants has been reported. The main risk factors are smoke, alcohol, hypertension, and family history. The most commonly used diagnostic method is brain computed tomography (CT), and the most accurate one is brain magnetic resonance imaging (MRI). There are few documented cases of increased uptake in bone scintigraphy with meta-stable technetium-99m (99mTc) and hydroxymethylene diphosphonate (HMDP) in the infarct area. The physiopathological mechanism behind this increased uptake observed in infarcts is uncertain, but there are some hypotheses, with the most accepted one being related to intracellular calcium metabolism implicated in ACE. We present the case of a 78-year-old woman with left hemiparesis and central facial paralysis, who underwent brain CT and MRI, leading to the diagnosis of an ACE. Subsequently, a bone scintigraphy was performed

Resumen
El evento cerebrovascular (ECV) agudo es un déficit neurológico causado por una lesión del sistema nervioso central (SNC) secundaria a suministro insuficiente de sangre oxigenada al cerebro. Puede ser hemorrágico o isquémico, que es el más frecuente. Afecta aproximadamente a 16 millones de personas al año en el mundo, y en Latinoamérica se ha descrito una incidencia de 35-183/100.000 habitantes. Los principales factores de riesgo son el tabaquismo, consumo de alcohol, hipertensión arterial y antecedentes familiares. El método diagnóstico más utilizado la es la tomografía de cráneo, y el más preciso, la resonancia magnética cerebral. Hay pocos casos documentados de aumento de la captación en la gammagrafía ósea con tecnecio 99 metaestable (99mTc) e hidroximetilendifosfonato (HMDP) en la zona del infarto. El mecanismo fisiopatológico de esta captación aumentada que se visualiza en los infartos es incierto, pero hay algunas hipótesis, y la más aceptada está relacionada con el metabolismo del calcio intracelular implicado en los ECV. Se presenta el caso de una mujer de 78 años de edad con hemiparesia izquierda y parálisis facial central, a quien se le realizaron tomografía y resonancia cerebral, con las cuales se diagnosticó un ECV. Posteriormente se le realizó una gammagrafía ósea con 99mTc-HMDP, en la cual se evidenció captación aumentada del radiofármaco en la zona del infarto.

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Introduction

Acute cerebrovascular event (CVE) is a neurological deficit due to a lesion of the nervous system caused by an insufficient supply of oxygenated blood to the brain. In Latin America, an incidence of 35-183/100,000 inhabitants has been described (1).

The diagnosis is made with brain computed tomography (CT) or brain magnetic resonance imaging (MRI); however, there are some incidental reports of visualization by bone scintigraphy with 99mTc-HMDP (2).

We present the case of a patient with acute CVE incidentally visualized with 99mTc-HMDP bone scintigraphy; the latter, not included in the diagnostic algorithm, but with characteristic findings.

Clinical case

A 78-year-old woman with a history of coronary artery disease, atrial fibrillation, previous ischemic stroke and recurrent deep vein thrombosis.

She consulted for left hemiplegia and left central facial paralysis since awakening. A simple brain CT scan was performed, in which the right "hyperdense middle cerebral artery sign" was found (Figure 1a), as a hyperacute sign of ischemia, and an area of encephalomalacia in the left inferior frontal gyrus (Figure 1b).

Brain MRI was performed, which reported acute infarction in the territory of the right middle cerebral artery, represented as diffusion restriction in the frontal lobe, parietal, temporal and right insula (figure 2).

Due to recurrent thrombotic events, a transesophageal echocardiogram was performed, which showed hypertrophy of both ventricles and dilatation of both atria; the Cardiology Department ordered a bone scan with 99mTc-HMDP to rule out amyloidosis.

In the bone scan, no signs suggestive of amyloidosis were found; however, a focal increase of enhancement was evidenced in the right region of the skull (Figure 3), which in the fused SPECT CT images correlated with the subacute infarction established in the right inferior frontal gyrus, parietal lobe and insula lobe (Figure 4). Conservative management with neurological rehabilitation was decided.

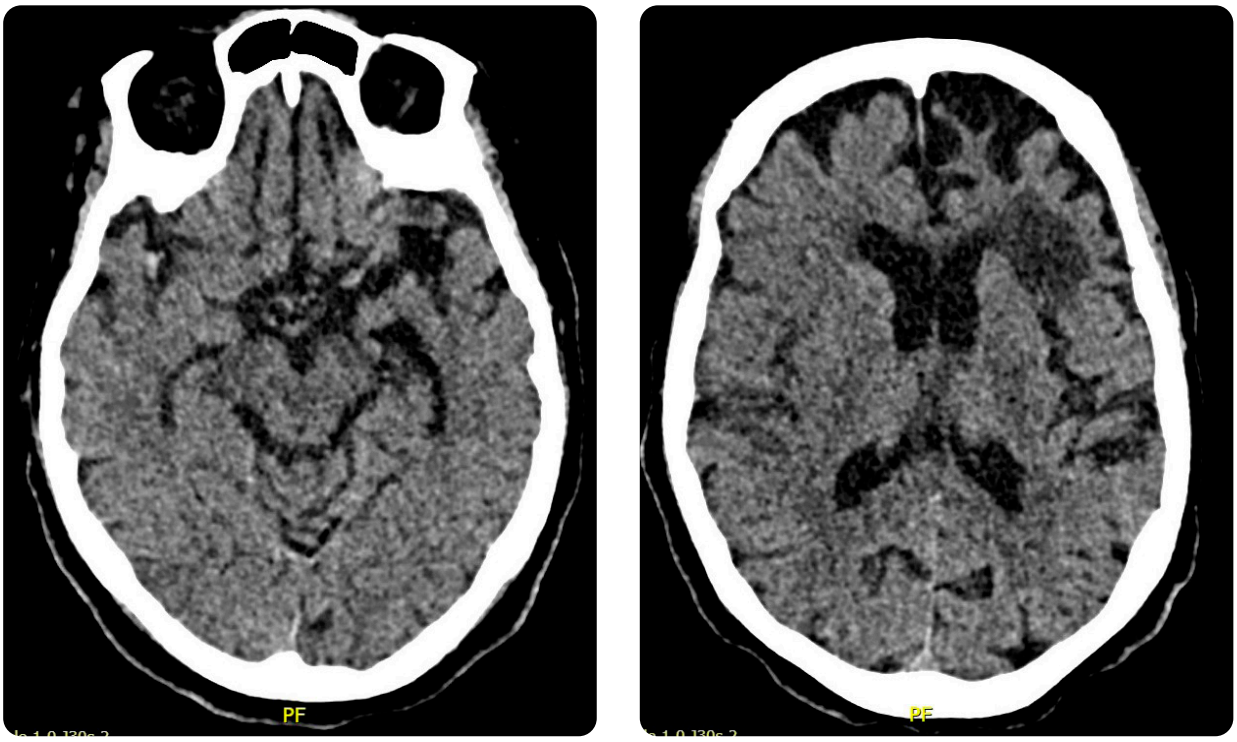


Figure 1. Brain CT, axial. a) Shows the sign of hyperdense right middle cerebral artery, as a sign of hyperacute LCA, and b) area of encephalomalacia in the left inferior frontal gyrus.

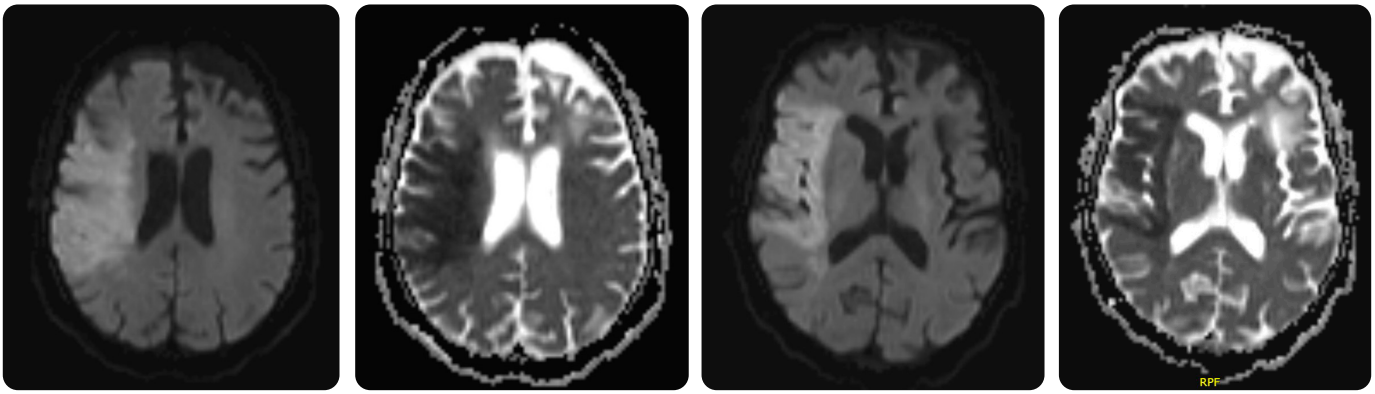


Figure 2. Brain MRI, axial slices in diffusion and ADC sequences. a, c) Areas of diffusion restriction, visualized as high signal in the frontal lobe and right insula; b, d) corresponding to areas of low signal in the ADC map.

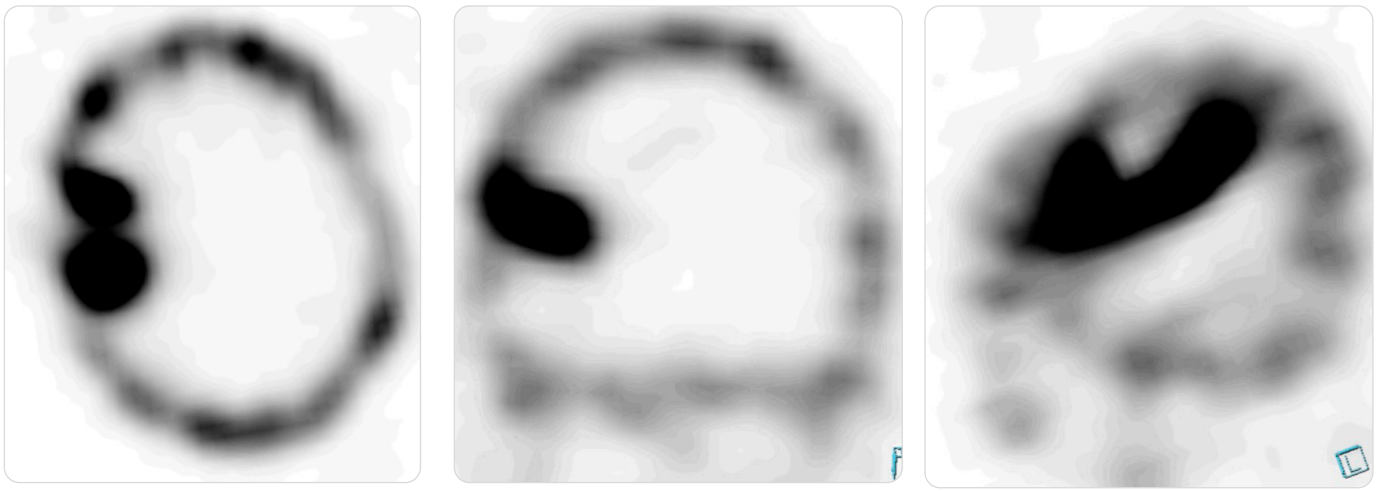


Figure 3. 99mTc-HMDP bone scan: a) axial, b) coronal and c) sagittal, showing focal enhancement in the right region of the cranium.

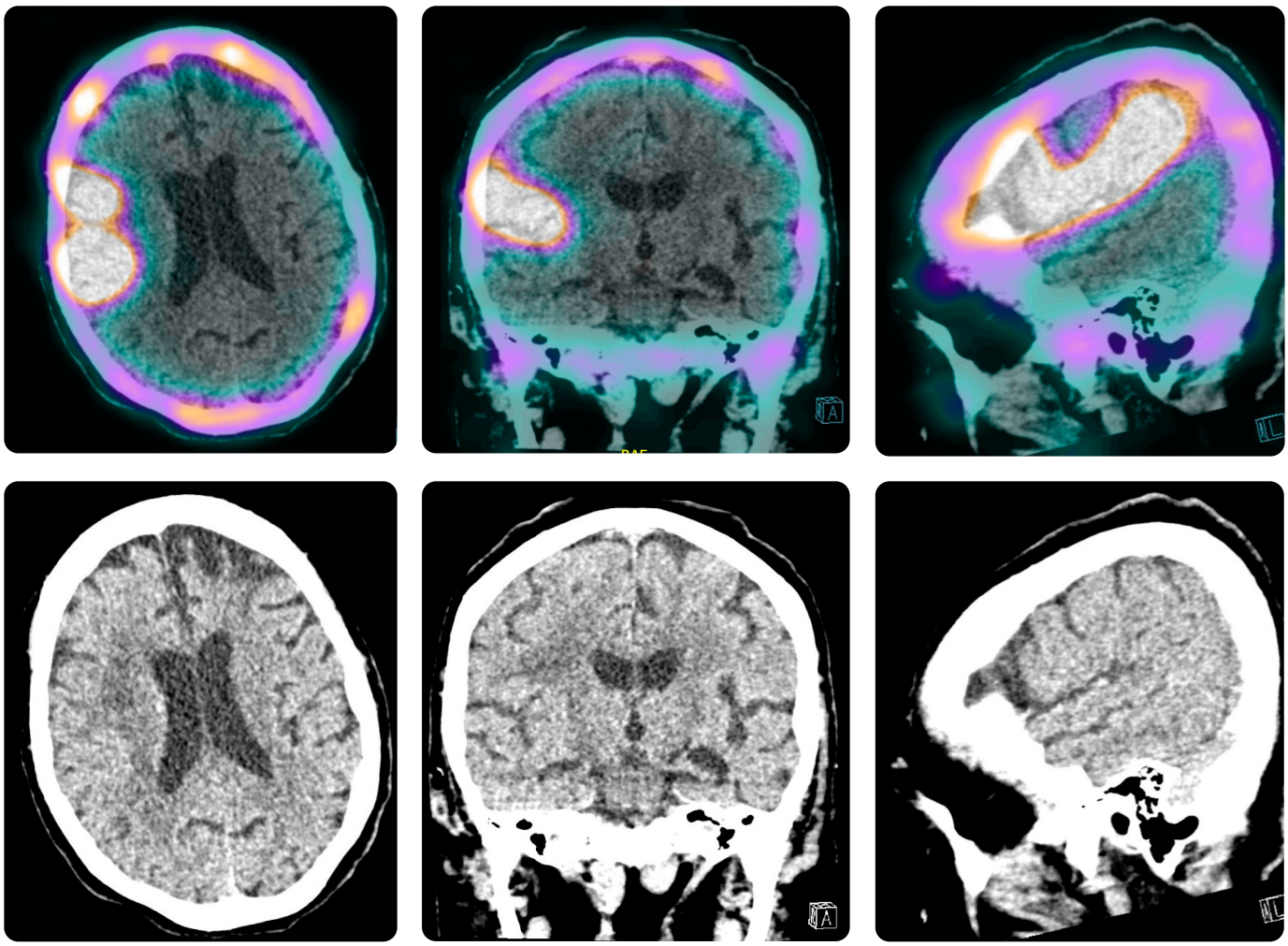


Figure 4. SPECT CT. a) fused in axial, b) coronal and c) sagittal plane, and its correlation with brain CT in d) axial, e) coronal, and f) sagittal plane. Subacute infarction established in the inferior frontal gyrus, parietal lobe and right insula lobe.

Discussion

Acute CVE is defined as a neurological deficit of vascular cause that generates insufficient supply of oxygenated blood to the brain. It can be hemorrhagic or ischemic; the latter accounts for 80% of acute cerebrovascular events (1).

It affects approximately 16 million people per year, of whom about 6 million die and those who survive have frequent functional limitations (3). In Latin America, an incidence of 35-183/100,000 inhabitants has been documented (1).

The main risk factors are smoking, alcohol consumption, arterial hypertension and family history (4).

Several stages have been determined, each with different imaging manifestations: early hyperacute (0 to 6 hours), late hyperacute (6 to 24 hours), acute (24 hours to 1 week), subacute (1 to 2 weeks), chronic (more than 2 weeks) (5).

There are different diagnostic methods, the most widely used is cranial CT, and the most accurate is cerebral MRI (1). Scintigraphy is not a method of choice for the diagnosis of acute CVE, but there are a few reports in the literature on increased radiopharmaceutical enhancement in areas of cerebral infarction (2).

Although there is no clear mechanism for this enhancement, there are some hypotheses, the most accepted is related to intracellular calcium (3, 4, 6). Bone scintigraphy is performed with 99mTc (a radioactive isotope) + HMDP (diphosphonate molecule); diphosphonates have an affinity for calcium, so their main mechanism of action is binding to areas of high metabolic activity in bone, and in areas of increased HMDP accumulation there is detectable gamma radiation emission (7); however, extraosseous enhancement is unusual.

In the pathophysiology of cerebral infarction, initially there is an insufficient supply of oxygen, glucose and nutrients to the brain, which affects oxidative phosphorylation and generates a deficit in the production of adenosine triphosphate (ATP), leading to energy failure and an alteration in the Na+/K+ ATPase pump; Secondary to this, there are alterations in membrane potentials and glutamate release, which activate the N-methyl-D-aspartate (NMDA) receptor, with opening of calcium channels and translocation of calcium to intracellular spaces; finally, there is a mitochondrial dysfunction that releases reactive oxygen species and produces cell death (3, 4, 6).

It is presumed that this intracellular calcium accumulation is what generates the increased radiopharmaceutical enhancement incidentally described in some patients with acute and subacute cerebrovascular events. However, more studies are needed regarding the exact pathophysiology and it should be clear that the technique of choice for the diagnosis of cerebrovascular events is not bone scintigraphy.

Conclusion

Bone scintigraphy is a useful study for the diagnosis of cardiac transthyretin amyloidosis; however, it is not considered in the diagnostic algorithm for cerebrovascular disease.

The most commonly used method in the diagnosis of cerebrovascular disease is cranial CT, and the most accurate is brain MRI.

Incidental enhancement in bone scintigraphy in cerebral infarcts has a poorly understood and hypothesis-based pathophysiology, and more studies are needed.

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