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Atrial Fibrillation Induced By A Brain Tumor In A Patient With No Medical History: A Rare Case Report

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Abstract:
Atrial fibrillation (AF), typically linked to cardiovascular risk factors, is rarely associated with primary brain tumors without thoracic involvement. This case report describes a 46-year-old man with no prior medical history who presented with palpitations and a syncopal episode. Initial evaluation revealed rapid AF, which was managed with electrical cardioversion. Normal echocardiography ruled out intracardiac thrombus. However, a brain CT uncovered a left frontoparietal tumor, indicating a neurocardiogenic mechanism for AF. This rare case highlights the need to consider unusual causes, like neurological conditions, for AF in patients without cardiovascular risk factors. It also emphasizes the challenges in anticoagulation due to the tumor's hemorrhagic risks, underscoring the importance of a multidisciplinary and personalized treatment approach.

Keywords: *atrial fibrillation, brain tumor, arrhythmia, neurocardiogenic mechanism, anticoagulation, syncope.*

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I. Introduction

Atrial fibrillation (AF) is the most common supraventricular arrhythmia and usually occurs in the presence of well-established cardiovascular risk factors[1]. Its association with central nervous system pathologies, notably brain tumors, is rare and poorly documented[2]. We report here an unusual case of inaugural AF revealing a primary brain tumor, in the absence of thoracic involvement or cardiovascular history, suggesting a possible implication of neurocardiogenic mechanisms.

II. Case Description

A 46-year-old man with no significant medical history presented to the emergency department with sudden- onset palpitations that had started 24 hours prior to consultation and had progressively increased in intensity. The patient also reported a single episode of syncope that had occurred 48 hours earlier.

At the time of admission, his vital signs were generally stable, except for an irregular tachycardia. The initial neurological examination was unremarkable. The electrocardiogram (ECG) revealed atrial fibrillation with a ventricular rate fluctuating between 160–180 bpm (Figure 1). His laboratory tests returned normal. The patient subsequently underwent external electrical cardioversion (EEC) after receiving 6000 IU of subcutaneous enoxaparin and no signs of intracardiac thrombus at the transthoracic echocardiography (TTE). Immediate evolution was favorable, with restoration of sinus rhythm and a heart rate of 80 bpm (Figure 2). TTE post EEC showed normal valves morphology, non-dilated atrias, normal-sized ventricles, and preserved systolic function. In the absence of an obvious cardiac cause and due to the inaugural nature of the presentation, a brain computed tomography (CT) scan was performed. It revealed a left fronto-parietal intra-axial suspect lesion exerting mass effect on the ipsilateral lateral ventricle, with perilesional edema in a finger-like pattern (Figure 3). Magnetic resonance imaging (MRI) confirmed the presence of a tumoral process centered on the left lateral ventricular trigone extending beyond the ventricular wall with invasion of the corpus callosum, without hydrocephalus (Figure 4).

The diagnosis of a tumoral process revealed by inaugural rapid atrial fibrillation was retained. The patient was then transferred to the neurosurgery department for specialized care.

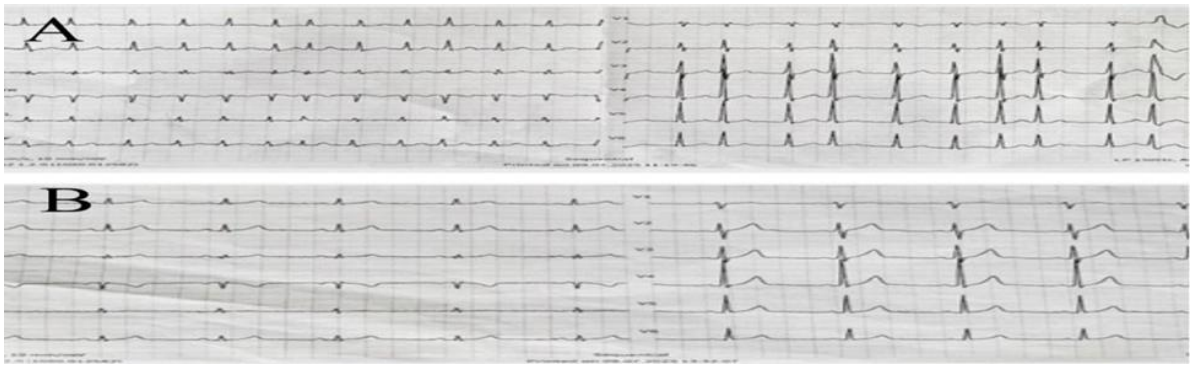

Figure 1. Electrocardiograms: (A) on admission showing atrial fibrillation, and (B) after external electrical cardioversion showing restauration of a sinus rhythm.

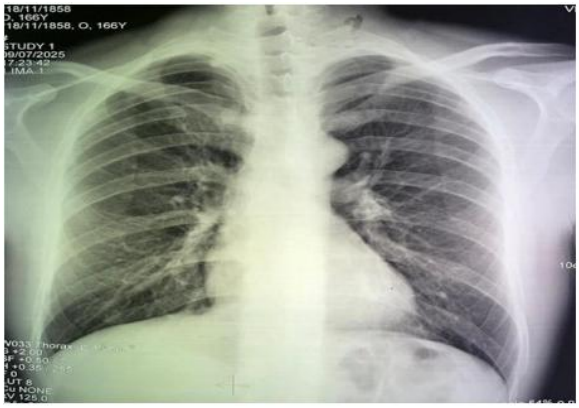

Figure 2. Normal Chest X-ray.

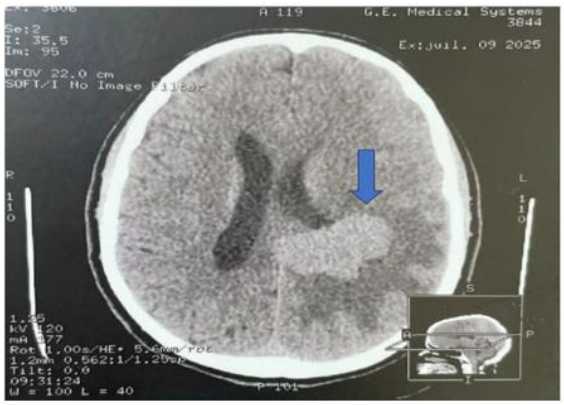

Figure 3. Brain CT scan showing a left fronto-parietal intra-axial lesion with mass effect on the ipsilateral ventricle (arrow) and perilesional edema.

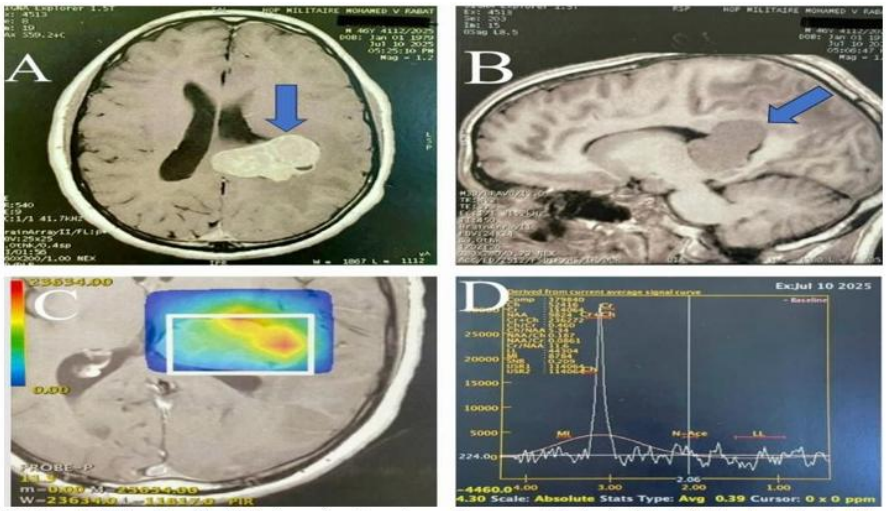

Figure 4. Brain MRI: axial (A) and sagittal (B) views showing a tumoral process at the level of the left lateral ventricular trigone, measuring 52×36×30 mm, extending beyond the lateral ventricular wall with invasion of the corpus callosum, without hydrocephalus (arrows). The spectroscopic profile (C) and (D) showing a choline peak and a drop in N-Acetylaspartate, a moderate myo-inositol peak and no lactate or lipid peaks.

III. Discussion

Brain tumors typically present with focal neurological signs, cognitive disturbances, or seizures, depending on their location, size, or mass effect. More rarely, they are discovered incidentally during the workup of an endocrinopathy, a primary cancer, or a systemic disorder[3]. In the case reported, the patient had no neurological symptoms at the time of admission but presented with atrial fibrillation (AF) and a recent history of syncope, which prompted brain imaging. Initially aimed at ruling out a thromboembolic complication, the cerebral imaging unexpectedly revealed a brain tumor.

Several studies have confirmed a higher prevalence of AF in cancer patients, although figures vary depending on the studied populations and methodologies used[4,5,6]. Cancers and AF share several common risk factors such as age, hypertension, obesity, and diabetes. Furthermore, the chronic systemic inflammation associated with the tumor microenvironment promotes atrial structural and electrical remodeling, facilitates the formation of ectopic foci, and induces conduction abnormalities. All of which contribute to the onset and maintenance of AF[4,5,6]. Our patient had none of the risk factors described in the literature[1], and his blood test showed no signs of inflammation.

Moreover, the occurrence of AF can be triggered by anticancer treatments, especially certain chemotherapies (anthracyclines, tyrosine kinase inhibitors, alkylating agents) and immunotherapies, which can cause direct cardiotoxicity, disrupt the cardiac conduction system, or promote a pro-arrhythmic state through indirect mechanisms (myocardial ischemia, electrolyte imbalances, systemic inflammation)[7]. Likewise, thoracic radiotherapy is a recognized risk factor for arrhythmias through fibrosis, inflammation, and myocardial remodeling, especially when it affects the atria or pulmonary veins[8]. Finally, oncologic surgery, through the inflammatory, metabolic, and hemodynamic stress it induces, can be a triggering factor for transient postoperative AF[9]. Our patient had not been diagnosed with a brain tumor previously and was not receiving any related treatment.

However, in rare cases, such as the one reported here, the brain tumor itself may be the origin of AF, through a neurocardiogenic mechanism. Certain brain regions — notably the insula, brainstem, and hypothalamus — are involved in the autonomic regulation of heart rhythm. The left insula is classically associated with parasympathetic modulation, while the right insula is involved in sympathetic modulation. Even indirect involvement of the insula can result in sympathetic hyperactivation, leading to arrhythmias or even sudden death. Brainstem lesions can affect cardio-respiratory centers and induce bradycardia, apnea, or cardiac arrest. Hypothalamic involvement (e.g., craniopharyngioma, glioma) can lead to sympathetic overactivation, causing severe hypertension, Takotsubo syndrome, and arrhythmias. Finally, a seizure related to a temporal tumor may provoke tachyarrhythmias or ictal asystole, occasionally responsible for unexplained sudden death in epileptic patients[10]. In our case, the left fronto- parietal intra-axial mass, although not invading the insula, exerted a significant mass effect that could interfere with autonomic circuits, possibly explaining the initial arrhythmia. The absence of any cardiac cause on echocardiography and the absence of cardiovascular risk factors supports this hypothesis.

The coexistence of cancer and AF increases the risk of thromboembolic events, due to both the atrial stasis induced by AF and the prothrombotic state associated with malignancy, characterized by platelet activation, secretion of pro-thrombotic cytokines and tissue factors, and local venous compression. Studies have shown that patients with both cancer and AF have a higher risk of stroke than non-cancer patients with similar CHA2DS2-VASc score[11,12]. In addition, brain tumors increase the risk of bleeding[12]. Therefore, the decision to initiate anticoagulation in patients with brain tumors and AF presents a true clinical dilemma, as one must balance thromboembolic risk with the potentially catastrophic risk of intracranial hemorrhage. The decision should be based on a careful, individualized assessment of thrombotic risk (CHA2DS2-VASc) and bleeding risk (HAS-BLED)[13]. In our case, the patient had a CHA2DS2-VASc score of 0, which did not justify curative anticoagulation initially. A single dose of 6000 IU of enoxaparin was administered prior to cardioversion followed by prophylactic anticoagulation at 4000 IU/day.

IV. Conclusion

This case highlights an episode of atrial fibrillation secondary to a primary brain tumor, in the absence of thoracic involvement or known cardiovascular disease. It suggests a neurocardiogenic mechanism, likely related to disruption of the autonomic regulation of cardiac rhythm. This observation emphasizes the need to consider a neurological origin in cases of unexplained atrial fibrillation, particularly in young patients without cardiovascular risk factors. Anticoagulation in this context remains a challenge due to the risk of bleeding and requires a personalized approach.

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