Case study

Cryptogenic stroke with a patent foramen ovale in a patient withpulmonaryembolism and thrombophilia:a case-based discussion

Abstract :

The patent foramen ovale (PFO) is a commonabnormalitythatisoftenassociatedwithcryptogenic stroke in young people. The management of PFO requiresendovascularclosurewhichpreventsrecurrencebetterthanantithrombotictreatment. In the presence of concomitant thrombophilia, the risk of recurrence of stroke afterclosure of the FOP ? isincreased, and one needs to instituteantithrombotictherapy. The choice and duration of the treatment are controversial. Our studyfocuses on a 37-year-old patient admitted for ischemicvascular accident in whom a patent foramen ovale wasfoundwith an interatrial septal aneurysm on a ground of thrombophiliaassociatedwith a leftpulmonaryembolism and an occlusion. Total leftinternalcarotidartery.

Since at present, thereis no optimal therapeuticstrategy for these patients, the indications must beconsidered on an individual basis. The aim of the clinical case is to relate ourexperience in the management of cryptogenic stroke associatedwithFOP(?) on a ground of thrombophilia.

Keys words :

cryptogenic stroke , patent foramen ovale , hypercoagubality, thrombophilia

Introduction

Patent foramen ovale (PFO) is a congenitalholebetween the right and the leftatria. In 15-30% of the population PFOs tend to persistthroughadulthood. In the majority of the population PFOsremainasymptomatic, but clinical manifestations are believed to beassociatedwithPFOs, such as cryptogenic stroke, especially in patients with atrial fibrillation [8-10]. The increasedrisk of ischemic stroke has been reported in severalstudies in patients withPFOs and acute pulmonaryembolism. The risk of ischemic stroke isshown to behigher in patients with PE and PFO compared to thosewithout PFO, as reported by severalstudieswithsmallnumber of subjects [11]. The present case study highlights the Cryptogenic stroke with a patent foramen ovale in a patient withpulmonaryembolism and thrombophilia.

Clinical case :

A 37-year-old patient with no particularpathologicalhistorypresents for lefthemiplegiawithdysarthria and exertionaldyspneaevolving for 2 months.

The clinicalexaminationfound a patient with a tachycardia at 114 bpm with no otherassociatedabnormalities. Cerebralmagneticresonanceimagingobjectified a deep right SylvianDALY(?)withsigns of hemorrhagicinfarction (1). The electrocardiogramshowed sinus tachycardiawith an S1Q3 pattern and negative T wavesanteriorly.

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Transthoracicultrasoundrevealed an aspect of acute cor pulmonalewith a right ventricledilated at 49 mm and systemicpulmonaryarterial pressures at 73 mmHg (2). Pulmonary CT angiographyconfirmedleftpulmonaryembolism (3). The venousecho-Doppler of the lowerlimbsdid not objectifydeepveinthrombosis. Transesophagealultrasoundrevealed a PFO with an interatrial septum aneurysm (ASIA) of 16x10 mm (4). CT angiography of the supra-aortictrunksshowed total occlusion of the left IC without dissection (5).

The biologicalassessment for thrombophilias found a protein C and antithrombin 3 deficiency. (howwas the assaydone)

The decisionwas to administerput the patient onunfractionatedheparin and then on anticoagulant treatmentbased on acenocoumarol.

Discussion

The management of patients with PFO-related stroke iscomplex. The choice of a treatmentisbased on the followingcriteria: the anatomicalcharacteristics of the severity of the PFO, a high risk of stroke recurrence on the RoPe(full form) score and an unfavorablebenefit/risk ratio of the antithrombotictreatment [2]. Recent trials demonstratedthatclosure of high-risk PFO can reduce the risk of recurrent stroke compared to antithrombotictherapy [3].

Few data are available on the impact of thrombophilia in patients withcryptogenic stroke [4]. Currentstudiesdemonstrate an increasedrisk of cryptogenic stroke in patients withthrombophilia and FOP(?)[5]. In our case, the deficiency in antithrombin 3 and in protein C constitutes a non negligiblesignificantrisk factor.

The origin of the stroke of the patient arises(?) Paraphrase. The first hypothesisis the increasedrisk of venousthrombosiswithparadoxicalembolism, in front of the FOP/ASIA(?) association withonthrombophilia. Closing the PFO isthen an optimal choice. In our case, the high pressures of the right cavitiessecondary to the massive leftpulmonaryembolismmeanthatclosure of the PFO cannotbeconsidered at this phase.

The second hypothesisisthatherembolismwaspurely of arterialorigin, given the total occlusion of the leftCI(?) thatshepresents, or thatherneurologicallesionwasintracerebral. Closing the PFO wouldthenprovide no additionalbenefit.

Somestudiesrecommendthatantithrombotictreatment, despite PFO closure, shouldbeconsidered on an individual basis [4]. There is a significantreduction in stroke recurrence in patients on anticoagulants compared to antiplatelets [6]. The

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American Heart Association recommends anticoagulant therapy for high-risk patients withcoexistingvenousthrombosis [7].

Conclusion

For patients withthrombophilia, with an indication for anticoagulation, closure of the PFO seemsreasonable but prolongedantithrombotictreatmentremains a subject of debate.

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artwork

fig. (1): Brain MRI

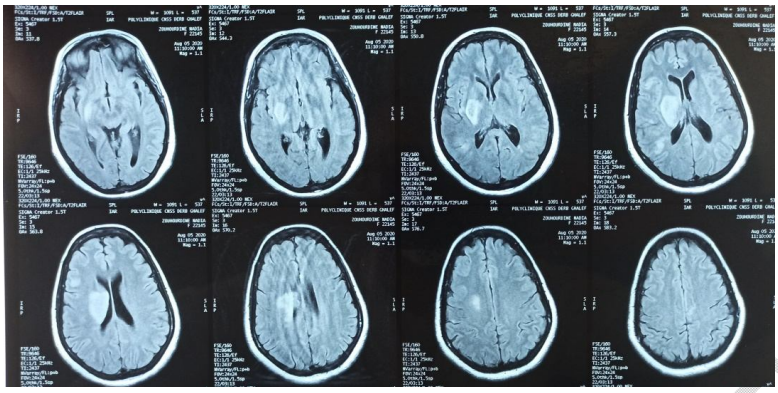

fig.(2) :ETT

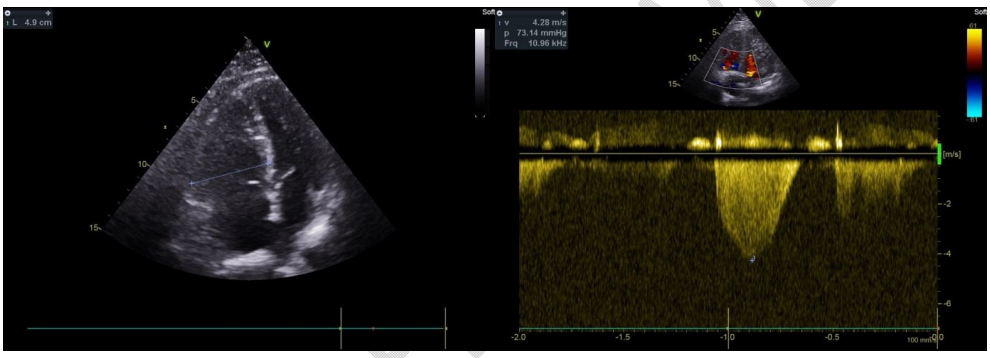

fig.(3): Chest CT angiography

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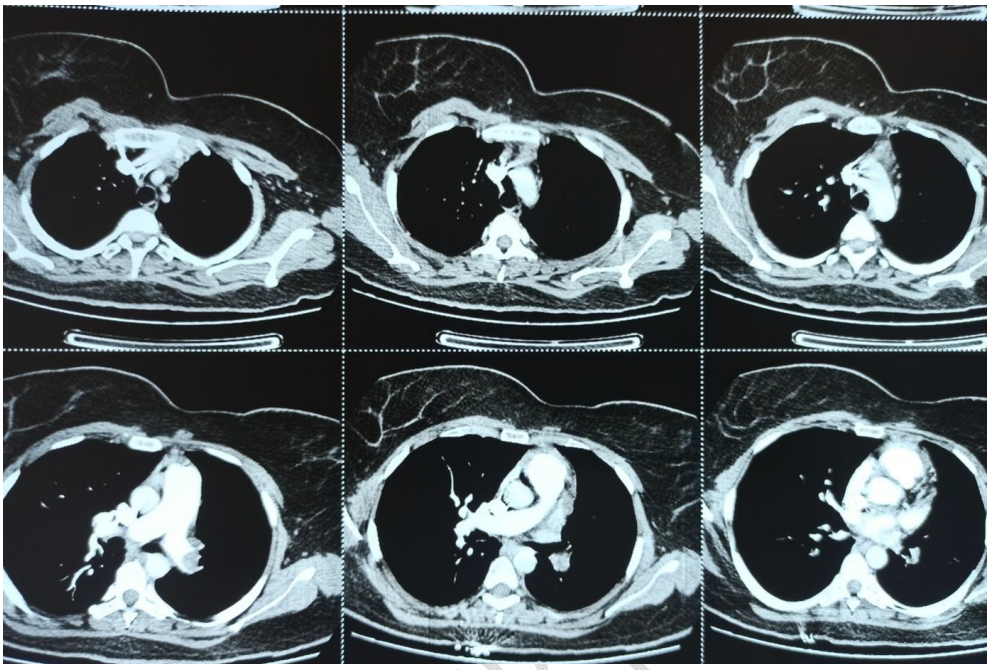


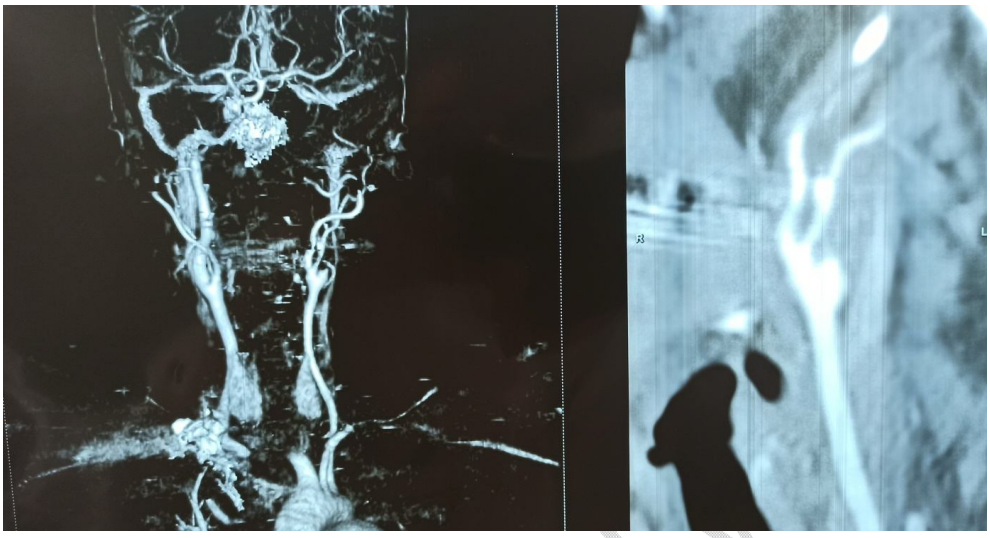
fig.(4): ETO

a: FOP (PFO ?)

b: Bubble test

c: ASIA (full form)

fig.(5): CT angiography of the supra-aortictrunks



“All authorsdeclarethatwritteninformed consent has been obtainedfrom the patient (or otherapproved parties) for publication of this case report and accompanying images. A copy of the written consent isavailable for review. by the editorialboard/editor/editorialboardmembers of this journal."