

# Case Report: Transient Global Aphasia Due to Pulmonary Embolism

### Vanaken Eric<sup>1\*</sup>, Debain Aziz<sup>1,2</sup> and Lieten Siddhartha<sup>1,2</sup>

<sup>1</sup>Department of Geriatrics, Universitair Ziekenhuis Brussel (UZ Brussel), Laarbeeklaan 101, 1090 Brussels, Belgium.

<sup>2</sup>Frailty in Aging (FRIA) investigation group, Faculty of Medicine & Pharmacy, Vrije Universiteit Brussel (VUB), Laarbeeklaan 103, 1090 Brussels, Belgium

**Corresponding Author:** Vanaken Eric, Department of Geriatrics - Vrije Universiteit Brussel - Laarbeeklaan 101, 1090 Brussels-Belgium, Tel.: +32476539954, Email: Eric.Vanaken@vub.be

**Citation:** Vanaken Eric, Debain Aziz and Lieten Siddhartha (2023). Case Report: Transient Global Aphasia Due to Pulmonary Embolism. J of Aging Res and Gerontol Stud 3: 101

#### Abstract

Background: We describe a case of neurological deficit as a rare presentation of acute pulmonary embolism (PE).

**Case presentation:** A patient, 86-year-old male, who was hospitalized after a fall complicated with fracture of the acetabulum woke up with global aphasia a couple of days after surgery. Stroke protocol was initiated without major deficits on perfusion computerized tomography (CT) of the head. Review of the mediastinal images revealed PE. After optimization of cardiac output with fluids and rate control with digoxin, patient showed a total recovery of speech. Anticoagulation was started and patient showed full recovery.

**Conclusion:** We would like to emphasize that physicians must be vigilant with older patients who fell in unclear circumstances, especially when anamnesis isn't reliable or when diagnosis is unclear. Pulmonary embolism is a life-threatening condition and diagnose in elderly is difficult because symptoms can be absent or unspecific as in this case aphasia was the main symptom. Also, it is important to review imaging as the attending physician has more clinical information and insight of the patient compared to the radiologist who only has limited information.

Keywords: Case Report; Transient global aphasia; atrial fibrillation; pulmonary embolism; fall; elderly

### List of Abbreviations

Pulmonary embolism (PE)

Computerized tomography (CT)

National Institutes of Health Stroke Scale (NIHSS)

#### Intravenous (IV)

North American Symptomatic Carotid Endarterectomy Trial (NASCET)

Activated Partial Thromboplastin Clotting Time (APTT)

Magnetic Resonance Imaging (MRI)

### Introduction

PE has a wide variety of presenting features, ranging from no symptoms to shock or sudden death. The most common presenting symptom is dyspnea followed by chest pain or cough. Many patients present with atypical symptoms but at this moment no literature was found mentioning global aphasia as main symptom. There are some case reports available of patients with PE manifesting with neurological symptoms as a result of hypoxemia or due to cerebral ischemia due to embolism. Some examples are ischemia in fat embolism syndrome after femur fracture [1] or paradoxical embolism in case of deep venous thrombosis combined with a patent foramen ovale [2].

### **Case Presentation**

An 86-year-old male presented at the emergency department after a fall with abdominal pain as a result. He reported to be in good health previously and had no other complaints. No chest pain, no cough or shortness of breath.

Patient was living in a residential care center due to cognitive impairment (last known mini mental state examination 19/30, history of alcohol abuse and Wernicke encephalopathy was not excluded) and reduced mobility. There were no witnesses to the fall, the patient reported falling out of bed and no loss of consciousness, although anamnesis wasn't reliable.

Medical history included arterial hypertension, parkinsonism, ischemic cardiomyopathy with stenting of the left anterior descending coronary artery, duodenal dysplasia and two femoral bypasses. History obtained by the family confirmed no recent falls and no alcohol use in the last years (family bought groceries for the patient and only bought alcohol free beer). Medication taken at home consisted of bisoprolol 2.5 mg once daily, aspirin 80 mg once daily, clopidogrel 75 mg once daily, tamsulosine 0.4 mg once daily, mirtazapine 30 mg once daily, trazodone 100 mg once daily, escitalopram 10 mg once daily, levodopa 200/50 mg 0.25 tablet three times daily, alprazolam retard 0.5 mg once daily and a fentanyl patch of 25 mcg/hour.

Examination showed a pale patient with normal vital signs. Cardiac and pulmonary auscultation was normal. Palpation of the abdomen was painful in the lower portion of the right fossa and abdominal CT scan at the emergency department revealed a complex fracture of the acetabulum with intra-abdominal active bleeding (figure 1 and figure 2).



Figure 1: Fracture of the acetabulum and hematoma.



Figure 2: Blush in the hematoma

Arterial blood gas at room air with pO2 of 91 mmHg (normal 65-80 mmHg), pCO2 of 37 mmHg (normal 36-44 mmHg), normal electrolytes, lactate of 3.6 mmol/L (normal 0.7-2.1 mmol/L) and normocytic anemia with hemoglobin of 9.0 g/dL (normal range 13-16.5 g/dL). Further lab results showed normal platelets, slightly elevated International Normalized Ratio 1.5 (normal range 0.8-1.3) with normal aPTT of 26.5 sec (normal range 22.2-34.4 sec), normal creatine kinase of 154 U/L (normal range < 190 U/L), minimally elevated lactate dehydrogenase of 345 U/L (normal range < 250 U/L) and troponins of 0,08 mcg/dL (normal range < 0,05 mcg/dL). We also noted an elevated N-terminal pro B-type natriuretic peptide of 792 ng/L (normal range < 125 ng/L). Furthermore, there was a normal renal function. A slightly elevated C-reactive protein (13.9 mg/L) with discrete leukocytosis (14900/mL). Thyroid stimulating hormone was normal. Electrocardiogram performed in the Emergency Department was normal. X-ray of the chest showed no abnormalities.

While taking Aspirin 80 mg and Clopidogrel 75 mg daily at home, the patient was administrated tranexamic acid and two units of platelets at the emergency department. Afterwards he was taken to the intensive care for hemodynamic observation.

The next day the patient underwent surgical transcondylar traction of the femur. He was hospitalized on the orthopedics ward for post-operative care. Aspirin was restarted 3 days after the fall and patient started revalidation on the ward.

Five days after the surgery, the patient was woken up in the morning by the nurses and was less reactive. When the doctor arrived in the room the patient was bradykinetic and presenting with a global aphasia (Glasgow Coma Scale 9/15, E3M5V1). Physical examination showed good vital signs with tachycardia: blood pressure 154/97 mmHg, saturation 96% at room air, temperature 37.6°C, heart rhythm was 134 bpm and glycaemia 141 mg/dL. The patient's eyes opened to speech and limbs moved spontaneously, but he did not obey commands and no verbal response could be obtained (National Institutes of Health Stroke Scale (NIHSS) 10). Lung auscultation was normal, cardiac auscultation with tachycardia but no murmur and abdominal examination without abnormalities. The operation wound showed no signs of infection and there were no signs of deep venous thrombosis in the four limbs.

Since the symptoms resembled a wake-up stroke (with global aphasia), stroke protocol was initiated. A non-contrast head CT was immediately obtained, showing no signs of bleeding or setting ischemia. Since there was no clear time of onset, the recent surgery and intra-abdominal bleeding, it was not possible to administer IV thrombolysis. Angio CT did not reveal any large vessel occlusion, thus thrombectomy was not indicated. There were no major perfusion deficits on CT perfusion. However, comparing both hemispheres, there is a global diminished perfusion in the right hemisphere on the Tmax images (figure 3).



Figure 3: Increased Tmax over the right hemisphere, suggestive for global hypoperfusion of the right cerebral hemisphere

We did find calcifications on both carotid arteries (figure 4, 77% stenosis right internal carotid artery and 34% stenosis left internal carotid artery according to NASCET).



Figure 4: Bilateral carotid stenosis

Review of the mediastinal image (taken with the angio-CT of the head to time the delivery of contrast with the imaging of the brain) showed massive pulmonary emboli of the left pulmonal artery and segmental branches (figure 5).



Figure 5: PE as seen on mediastinal image used to time the delivery of contrast to the brain

ECG showed a previously unknown atrial fibrillation. Rate control was obtained with intravenous digoxin 250 mg and oral bisoprolol 5 mg. Patient showed a quick recovery and was able to obey commands and answer questions after obtaining a better cardiac output with adequate rate control one hour after onset (NIHSS 1).

Patient was treated with intravenous heparin (with close monitoring of the aPTT because of the large abdominal hematoma). Transthoracic echocardiography was reassuring without signs of right heart failure. Three days later control CT abdomen showed a decrease of the abdominal hematoma. Anticoagulation was increased to full therapeutic dose without further complications. A follow up in the next two years showed no recurrence of aphasia.

#### Discussion

We presented a case of global aphasia in a hospitalized patient one week upon presentation after a fall. He got diagnosed with PE a week after hospitalization. While the incidence of PE increases significantly with age [3], diagnosis in the elderly is more difficult and often delayed. A recent review associated age (and even disproportionately high proportion with age > 85 years), absence of dyspnea, presence of cardiopulmonary disease and altered mental status (defined by dementia, delirium or syncope) with a delay in diagnosis [4].

Moreover, we would like to emphasize that although Wells score for PE [3], used for determining risk of PE, is more sensitive than the revised Geneva score [5], it is not as reliable with older patients. In this case Wells score was 0 at the presentation of the emergency department, while revised Geneva score was 4, classifying our patient in the moderate risk group: ~20-30% incidence of PE. Where symptoms are non-specific or absent, the diagnosis of PE can be missed.

As a differential diagnosis for global aphasia, we include stroke or transient ischemic attack and epilepsy (e.g., speech arrest). To determine if there were any ischemic lesions, an MRI needed to be done. However, this was not possible in this case due to external fixation materials. Furthermore, because of high age and comorbidities, the results of the MRI would not have had any therapeutic consequences since the patient was already on antiplatelet and anticoagulating therapy. Ischemia of the brain has been described in several cases due to arterial obstruction (e.g., fat embolism syndrome and trombo-embolism from a deep venous thrombosis through a patent foramen ovale as discussed in the introduction). We also could have repeated a CT after a couple of weeks to evaluate if any major infarcts appeared. This was not done, as it would not have changed the therapeutic approach.

Having no prior history of epilepsy and because of spontaneous recovery and lack of recurrence, we didn't perform an electroencephalogram. Since this is a non-invasive procedure, it should have been considered as non-convulsive epilepsy is underdiagnosed in elderly [6].

We did not find any literature specifically mentioning aphasia as main symptom of PE. As a pathophysiological mechanism we propose that this could be a result of relative brain hypoperfusion due to cardiogenic failure as already been described in several studies [7-9]. As the patient was left-handed and global hypoperfusion was seen on the right hemisphere (probably the dominant hemisphere where Broca's and Wernicke's area is located), this could explain the aphasia. Asymptomatic right carotid stenosis could become symptomatic because of cardiogenic failure. Because of the full recovery after improving the cardiac output and the high age of the patient, best medical therapy was advised according to recent European guidelines on endarterectomy [10].

Because of global aphasia, recent surgery and cognitive impairment, elaborate neurological testing proved difficult and more extended deficits could not be excluded in the acute setting.

### Conclusion

- Global aphasia can be a presenting feature of PE.
- PE has a wide variety of presenting features, in elderly diagnosis can be difficult due to variety of symptoms (such as altered mental state or delirium) or the absence of symptoms.
- The need for prompt diagnosis of PE is clear, because with appropriate treatment the majority of patients may survive, but with geriatric patients/ population, the prompt diagnosis is difficult to make.
- In elderly, when a fall is in unclear circumstances and a new cardiac arrhythmia occurs, we should be aware of acute pulmonary embolism.
- The commonly used calculators to predict the risk of PE are not adapted for elderly patients.
- Attending physicians should always review all the imagery themselves as more information and insight is necessary for good diagnosis and the radiologist only has limited information.

### Declaration

Ethics approval and consent to participate: not applicable.

## **Consent for Publication**

Written Informed Consent for publication was obtained from all the patient. All data generated or analyzed during this study are included in this published article [and its supplementary information files].

### **Competing interests**

The authors declare that they have no competing interests.

### Funding

No funding was obtained.

#### Authors' contributions

Case report written and reviewed by authors in collaboration.

### Acknowledgements

Thanks for the department of Geriatrics for making time available to write this case report.

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