

Massive Acute Pulmonary Emboly Complicated with a Cardiac Arrest: Diagnostic Difficulty and Management in Tunisian Care Unit

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Introduction

Acute pulmonary embolism (EPA) is a serious complication associated with high morbidity and mortality. We report a case of EPA complicated by sudden cardiac arrest.

Observation

28-year-old patient with no significant pathological antecedents, smoking (30 AP), victim of an aggression causing multiple wounds (fingers, legs). His examination on admission to the orthopedic department: afebrile, morbidly obese, correct cardiorespiratory state, normal cardiopulmonary auscultation. No functional symptoms were reported by the patient. We note, multiple superficial and extended wounds of the two calves. The initial biology was without anomalies. The patient initially received: antibiotic therapy, anti-tetanus serum without thromboprophylaxis (TBP). Surgical exploration (wound suturing) was decided 48 hours after admission. The anesthesiologist on call was then asked to provide anesthesia for the patient. The pre-anesthetic evaluation finds a painful patient (chest pain such as oppression as soon as it is mobilized on the operating table). Clinical examination: conscious GCS at 15, profuse sweating, Pulse Saturation in Oxygen (SpO₂) at ambient air at 97%. TA = 15/08, HR = 100 bpm, no peripheral signs of shock, normal pulmonary cardiopulmonary bypass with apyrexia, foul odor of skin lesions. At the electrocardioscope: under ST shift in the extended anterior, sinus tachycardia. Given this context, we discussed the diagnosis of EPA. We performed an electrocardiogram (ECG): regular sinus rhythm at 120 beats per minute, complete right limb block, under ST extended anterior spread (V1-V6). Troponin levels were negative, Brain Natriuretic peptide (BNP 4.19 ng/l) and D-dimers not measured (reagent not available). Ambient air arterial gas (AA): hypocapnia with hypoxia. Faced with these arguments, the diagnosis of EPA was strongly evoked. Standard heparin with curative dose was administered intravenously with vascular oxygen oxygenation to the mask. Confirmation by thoracic angioscanner could not be performed (by technical failure). An echocardiogram (EC) requested at the bedside of the patient considered non-transportable but not performed due to the lack of an ultrasound system. During the careful mobilization of the patient for transfer to the intensive care unit, abrupt onset of isolated bradycardia at 55 beat/min resolving after a bolus of atropine followed by a 2nd episode of bradycardia with altered state of consciousness and complicated cardiac arrest not recovered after adequate resuscitation. The patient was dead and an autopsy confirmed the diagnosis of massive EPA.

Comment

Confirmation of the diagnosis of EPA can be difficult because of the instability of the patient's condition and/or the unavailability of the key examination. The major role of EC in making the diagnosis and predicting an immediate prognosis that can guide invasive therapy. In our case, the suspicion of EPA was based on several clinical arguments: symptomatology and field but this diagnosis remained hypothetical given the wide diversity of the clinical translation of this pathology. Paraclinical explorations of the first line: ECG, GDS and biological markers: troponins, BNP and D-dimer were collected. From these examinations and from the clinical presentation, we strongly evoked the diagnosis of PE with possible notion of floating thrombus explaining the sudden worsening of the clinical picture during the mobiliza-

tion of the patient. The obstacle of the non-availability of the complementary non-invasive examination (angioscanner) confirming the positive diagnosis and the seriousness of the clinical picture underline the importance of the cardiac ultrasound, anodyne and feasible examination at the bedside of the patient. The contribution can be of great importance because it allows to show signs of right heart failure (dilatation or hypokinesia VD, increase of the ratio of the VD / VG diameters and increase of speed of the tricuspid insufficiency flow, a right-left shunt through a patent foramen ovale (FOP) and presence of thrombus in the right cavities) indicating the immediate initiation of fibrinolytic treatment [1,2]. The absence of a reliable diagnostic tool in our case (mainly EC that can be performed at the bedside of the patient, prompted us to initiate anticoagulants (UFH) in consultation with cardiologists. was not indicated because of the absence of a clinical argument of gravity (state of shock) and because of its own risks. The mobilization of the patient during his transfer to the intensive care unit led to a cardiac arrest which suggests the diagnosis of a floating thrombus at the origin of a massive EP.

Bibliography

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