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LETTER TO THE EDITOR

A possible cause of cholesterol crystal embolism in a polytrauma patient?

KEYWORDS
ICU;
Cholesterol crystal embolism;
Polytrauma

Introduction

Cholesterol crystal embolization (CCE) is a multiorgan disease that is frequently underdiagnosed. It occurs when an atherosclerotic plaque ruptures and releases cholesterol crystals into the bloodstream resulting in organ damages by mechanical plugging and inflammatory response in small arteries [1]. CCE is a fatal disease that can occur spontaneously or accompany a myriad of endovascular procedures [2]. This entity is not frequently reported in the literature. We report a case of a possible posttraumatic CCE, with atypical symptomatology, affecting a 68-year-old man.

Case report

A 68-year-old male was referred to our emergency department for polytrauma following a high velocity traffic accident. He had a history of hypertension, dyslipidemia, and smoking (50 pack-year). He had never undergone endovascular intervention. He was oriented and conscious and initial hemodynamic and respiratory parameters were normal. Physical examination showed no abnormal findings. Neither blue-colored toes nor livedo reticularis were observed. The computed tomography body scan performed on admission showed multiple fractures. Moreover, the chest CT scan revealed a ground-glass hyperdensity in the right upper lobe of a lung related to a pulmonary contusion and an unstable atheromatous calcium plaque of the aortic isthmus measuring 7 mm in maximum thickness and extending over 18 mm of irregular contours (Fig. 1A). Abdominal CT scan showed an extensive endoluminal defect of 14 mm at the level of the emergence of the celiac trunk (Fig. 1B), foci of hyperacute splenic infarction (multiple infarcts appearing as hypodense non-enhancing lesions during the portal venous phase, associated with areas of hemorrhagic infarction) (Fig. 1C) and an unstable sub-occlusive plaque of the abdominal aorta up stream of its bifurcation extended over 19 mm and 8 mm thick (Fig. 1D). CT scan eliminated the presence of splenic artery dissection or spleen rupture.

The patient was admitted to intensive care unit. Biological findings showed a high level of triglyceride (4.59 mmol/L), HDL-cholesterol at 1.13 mmol/L, LDL-cholesterol at 1.78 mmol/L, normal renal function (urea: 6 mmol/L; creatinine: 90 μmol/L), and a normal serum complement. Laboratory tests showed also an elevated white blood cell count (17,800/μL) and elevated C-reactive protein rate (124 mg/L) but did not show eosinophilia. An ophthalmological examination showed no signs in favor of a cholesterol-embolism. The diagnosis of CCE with spleen infarction was evoked and the patient was put under statin therapy and the preventive anticoagulation therapy was stopped.

Discussion

Cholesterol crystal embolism (CCE) also called arterial embolism of atheromatous origin are observed in patients with diffuse atherosclerosis. The disease usually affects white males, often in their sixties, with a clinical history of arterial hypertension, diabetes mellitus, heavy smoking, and hypercholesterolemia [3].

Although cholesterol crystal embolization may occur spontaneously (in around 20% of the cases) [4], it can follow invasive vascular procedures such as aortic surgical interventions, angiography, and catheterization of the left ventricle [5]. It can also occur after medications targeting the coagulation system [6].

In fact, most cases described in literature were linked to iatrogenic events [7]. In our case, the patient had no history of endovascular catheter manipulation and was not under any type of anticoagulation treatment. The diagnosis of CCE was evoked after a blunt trauma following a road traffic accident.

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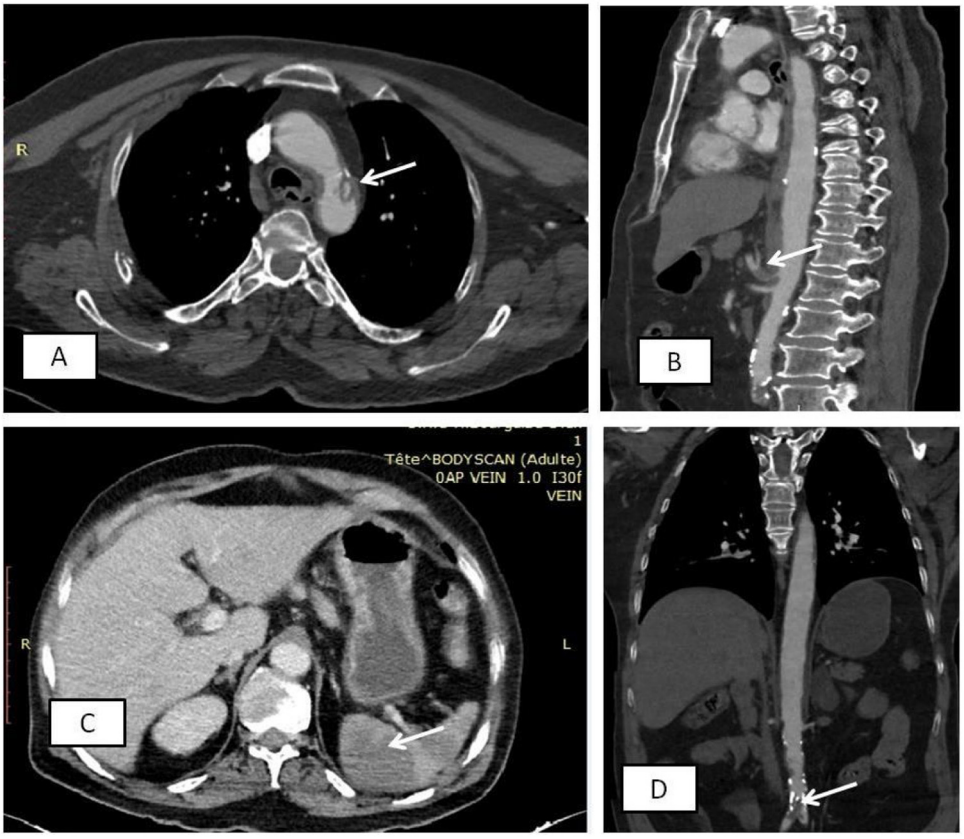

Figure 1 The computed tomography body scan performed on admission showed an unstable atheromatous calcium plaque of the aortic isthmus measuring 7 mm in maximum thickness and extending over 18 mm of irregular contours (arrow image A). It showed again an extensive endoluminal defect of 14 mm at the level of the emergence of the celiac trunk (arrow image B), foci of splenic infarction during the portal venous phase (arrow image C), unstable sub-occlusive plaque of the abdominal aorta up stream of its bifurcation extended over 19 mm and 8 mm thick (arrow image D).

In the present case, CCE could be explained by the severity of the trauma that probably induced plaque erosion and rupture atherosclerotic plaques lining the walls of the aorta exposing the components of the plaque to systemic circulation. This can lead to narrowing or obliteration of small arterial lumens resulting in ischemia or infarction. In addition to mechanical injury, CCE can lead to inflammatory and endothelial vascular reactions [6].

CCE has been considered as a multisystemic disease since it can affect many anatomic sites, including skin, heart, gastrointestinal tract, kidney, retina, and skeletal muscle [1—7]. Clinical presentation depends on the size of cholesterol crystals dislodged and the organ system involved [8].

The gastrointestinal tract is one of the most common organ system affected by cholesterol emboli, second only to kidney and skin [2]. In our case, spleen ischemia was the only organ injury manifested since renal function was normal and the patient did not express skin signs of microcirculatory perfusion's disruption.

In fact, the diagnosis of CCE is often difficult to make due to the polymorphism of the clinical presentation and the subclinical and asymptomatic presentation in some cases. For this reason, the co-occurrence of clinical findings, imaging and laboratory methods are required to establish the diagnosis.

In the present case, diagnosis was evoked on several features. First, our patient had a history of known risk factors for CCE. Second, the patient had multiple atherosclerotic plaques along aorta identified by imaging, associated with the risk of CCE. Particularly, ulcerated atheromatous plaques suggested their vulnerability and were considered to be the source of CCE. Lastly, the presence of acute organ infarction supported the diagnosis.

On the other hand, laboratory tests showed serum inflammation but did not detect eosinophilia. In fact, elevated inflammatory, abnormal renal function tests and eosinophilia are useful but non-specific [6]. In addition, transient eosinophilia tends to occur more in patients with renal involvement which is not present in our case [9,10].

Certainly, the diagnosis of CCE remains mostly a diagnosis of elimination and often refers to clinical, biological, morphological and histologic arguments. The main differential diagnosis is thromboembolism in which thrombus forms on top of atherosclerotic plaque and large emboli break off, causing organ infarctions [1,11]. However, ulcerated plaques aspect in imaging may also support the diagnosis of CCE. Consequently, the decision to stop anticoagulation seemed to be reasonable.

Undoubtedly, it is important to acknowledge the limitations of this case report. First, histopathological

confirmation by biopsy which is the gold standard for diagnosis and the definitive method to prove microvascular obstruction was not accomplished. Nevertheless splenic biopsy procedure may be harmful rather than beneficial [12]. Second, there was a lack of available data to comfort the diagnosis like the rate of LDH or eosinophiluria.

In conclusion, although the limitations of this report, we present a case of possible posttraumatic CCE affecting the spleen. From this interesting observation, several points of interest should be considered. First, CCE could be also a potential complication in polytrauma patients, particularly in patients with severe atherosclerosis. This represents the main importance of the current case since most of reported CCE were usually preceded by either aortic invasive angiographic and surgical interventions or anticoagulant treatment in elderly patients. Another interesting point to consider is the non-specific clinical presentation of the disease (no follow-up of patient is described in this case) Finally, cholesterol embolization syndrome is a manifestation of advanced atherosclerosis that leads to high morbidity and mortality requiring more knowledge to establish guidelines for the diagnosis and therapy of this severe disease.

Human and animal rights

The authors declare that the work described has not involved experimentation on humans or animals.

Informed consent and patient details

The authors declare that this report does not contain any personal information that could lead to the identification of the patient.

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Disclosure of interest

The authors declare that they have no competing interest.

Author contributions

All authors attest that they meet the current International Committee of Medical Journal Editors (ICMJE) criteria for Authorship.

CRediT authorship contribution statement

M. Bahloul: conceptualization, formal analysis, investigation, writing — original draft, validation.

A. Makni: investigation, writing — review and editing.

S. Kharrat: writing — review and editing, validation.

I. Alila: writing — review and editing, validation.

B. Suissi: writing — review and editing, validation.

M. Bouaziz: conceptualization, formal analysis, writing — review, validation.

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M. Bahloul[a,*]
A. Makni[a]
S. Kharrat[a]
I. Alila[a]
B. Suissi[b]
M. Bouaziz[a]

[a] *Department of Intensive Care, Habib Bourguiba University Hospital, Sfax University, Sfax, Tunisia*

[b] *Department of Radiology, Habib Bourguiba Hospital, Sfax University, Sfax, Tunisia*

* Corresponding author. Department of Intensive Care, Habib Bourguiba University Hospital, 3029 Sfax, Tunisia.
E-mail address: bahloulmab@yahoo.fr (M. Bahloul)