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CASE REPORT

Pneumomediastinum revisited: we should reconsider usual classification

Melanie Dechamps[a,b], Lieven De Smedt[a] and Said Hachimi Idrissi[a]

[a]Emergency Department, Ghent University Hospital, University of Ghent, Ghent, Belgium; [b]Cardiovascular Intensive Care Unit, Saint Luc University Hospital, Catholic University of Louvain, Brussels, Belgium

ABSTRACT

Pneumomediastinum work-up should focus on differentiating mediastinal organ injuries from alveolar rupture since these two causative mechanisms have a different management and prognosis. After an admission at our Emergency Department, we wanted to challenge the current classification into 'spontaneous' or 'secondary' pneumomediastinum, which reflects the clinical rather than the pathophysiological circumstances and is therefore confusing and inappropriate to our view. We propose a new work-up algorithm based on clinical risks factors and chest CT findings.

KEYWORDS

Pneumomediastinum; chest trauma

Introduction

After a recent admission at our Emergency Department, we wanted to highlight that the usual classification of pneumomediastinum (PM) into 'spontaneous' or 'secondary' is unclear, inducing a misunderstanding of the pathophysiology and therefore leading to inappropriate investigations and management.

Case report

A 19-year-old man presented at the emergency department (ED) after a minor trauma during a football game (fallen on the grass). He complained of a swelling of the throat, mild dyspnea, breath-related chest pain and cough. He had no past medical history and did not take any medication.

Vital signs were within the normal range. Physical examination showed bilateral subcutaneous emphysema in the cervical area, normal bilateral ventilation and a soft abdomen. No visible injury to the thorax could be detected.

Chest X-ray showed a PM and a very small anterior pneumothorax (Figure 1(A) and (B)). Chest CT scan substantiated the PM with signs of Macklin effect (Figure 1(C)). Neither rib fracture nor sign of bronchial or oesophageal perforation were observed. Finally, a flexible laryngoscopy and bronchoscopy were performed which excluded any abnormality.

The patient was observed as an inpatient and was discharged uneventfully 72 h later. Ambulatory follow-up was planned at day 7 with follow-up chest X-ray, substantiating complete recovery.

Discussion

PM work-up aims at differentiating mediastinal organ injuries (MOI) from alveolar rupture (AR), these two underlying mechanisms having a different management and prognosis. Unfortunately, PM is usually classified into two categories according to the clinical circumstances of the PM but not reflecting the etiological process.

A 'secondary' PM refers to a traumatic or iatrogenic origin while a 'spontaneous' PM refers to a PM occurring in other circumstances [1–3]. It must be pointed out that this classification does not reach consensus as for some authors a spontaneous PM only refers to a PM without any identified causative factor [4]. This lack of clarification makes the literature unclear (Figure 2). It is generally admitted that a CT scan and / or an endoscopy looking for MOI must be performed for all traumatic and iatrogenic PM and for spontaneous PM when MOI is suspected but risk factors of MOI are not properly described [4].

From a physiopathological point of view, a PM can be the result of an airway injury, an oesophageal injury, a penetrating trauma or a surgical procedure (air coming from the outside), a mediastinitis (gas producing organism) or an alveolar rupture (with or without a pneumothorax).

The pathophysiology of a PM caused by an alveolar rupture is known as the 'Macklin effect' [5]. It corresponds to a process starting by an overly distended alveolar rupture followed by air dissection along the bronchovascular sheaths towards the hila and into the mediastinum. It can occur in any condition increasing the alveolar pressure.

CONTACT Melanie Dechamps melanie.dechamps@uclouvain.be

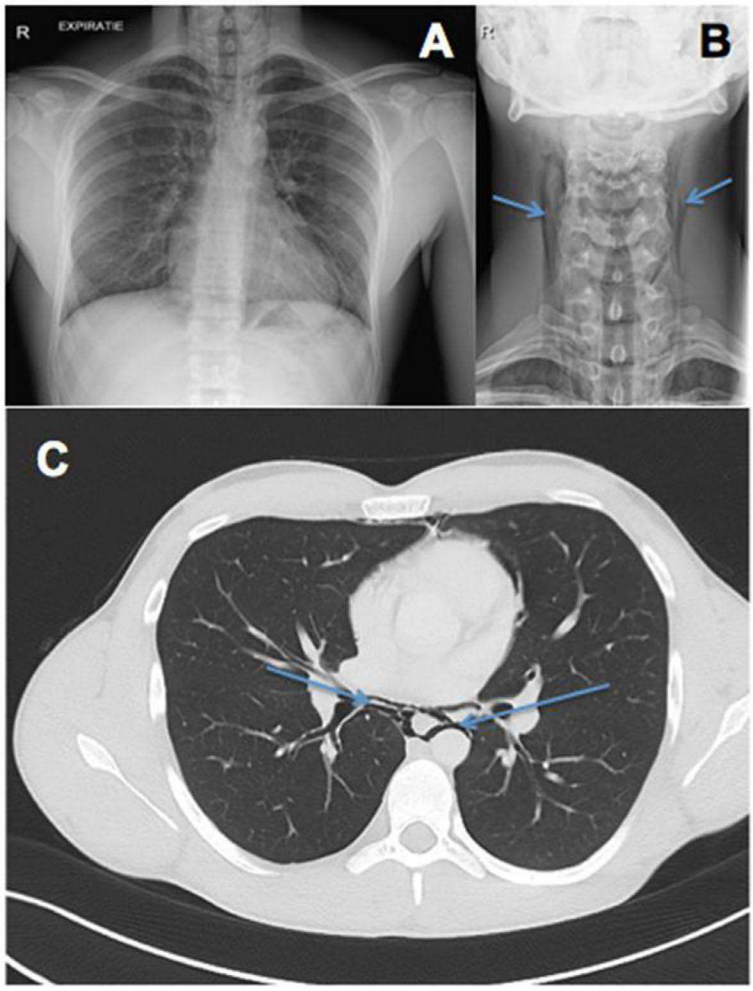

Figure 1. Chest X-ray showing a pneumomediastinum (A), subcutaneous emphysema (B) and chest CT scan showing air dissection along the peribronchovascular sheaths (Macklin effect, C).

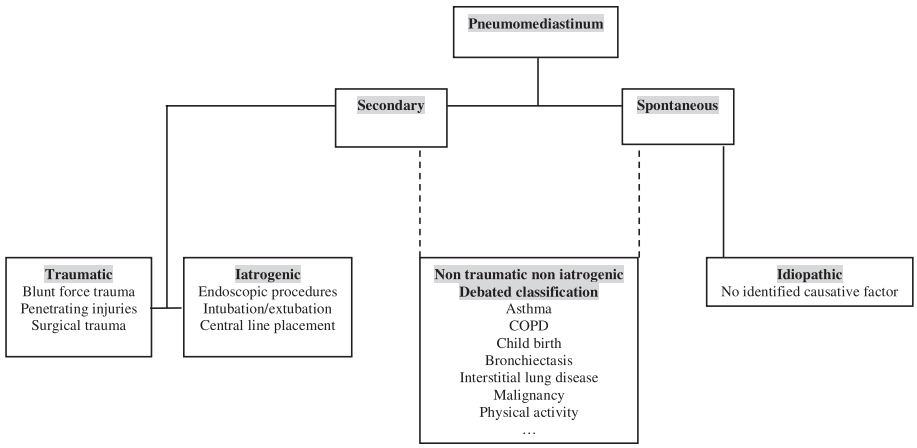

Figure 2. Usual classification of pneumomediastinum reflecting the causative clinical circumstances [3,4].

This mechanism explains most of the cases of 'spontaneous' PM but also a large proportion of the 'secondary' PM leading to the same process, as blunt trauma or mechanical ventilation. Therefore, these 'secondary' PM without MOI should be classified as 'spontaneous' regarding the management and the follow-up. The prognosis of a PM after alveolar rupture is excellent with a complete resolution of the chest X-ray abnormalities within a week and a recurrence rate of 1.2% with very few complications [1]. Management is conservative, antibiotics are not recommended and ambulatory follow-up is applicable for low-risk patients [1,6].

On the contrary, PM caused by an airway or an oesophageal rupture is a serious medical condition requiring admission into hospital, antibiotics administration and in some cases a surgical procedure. Although

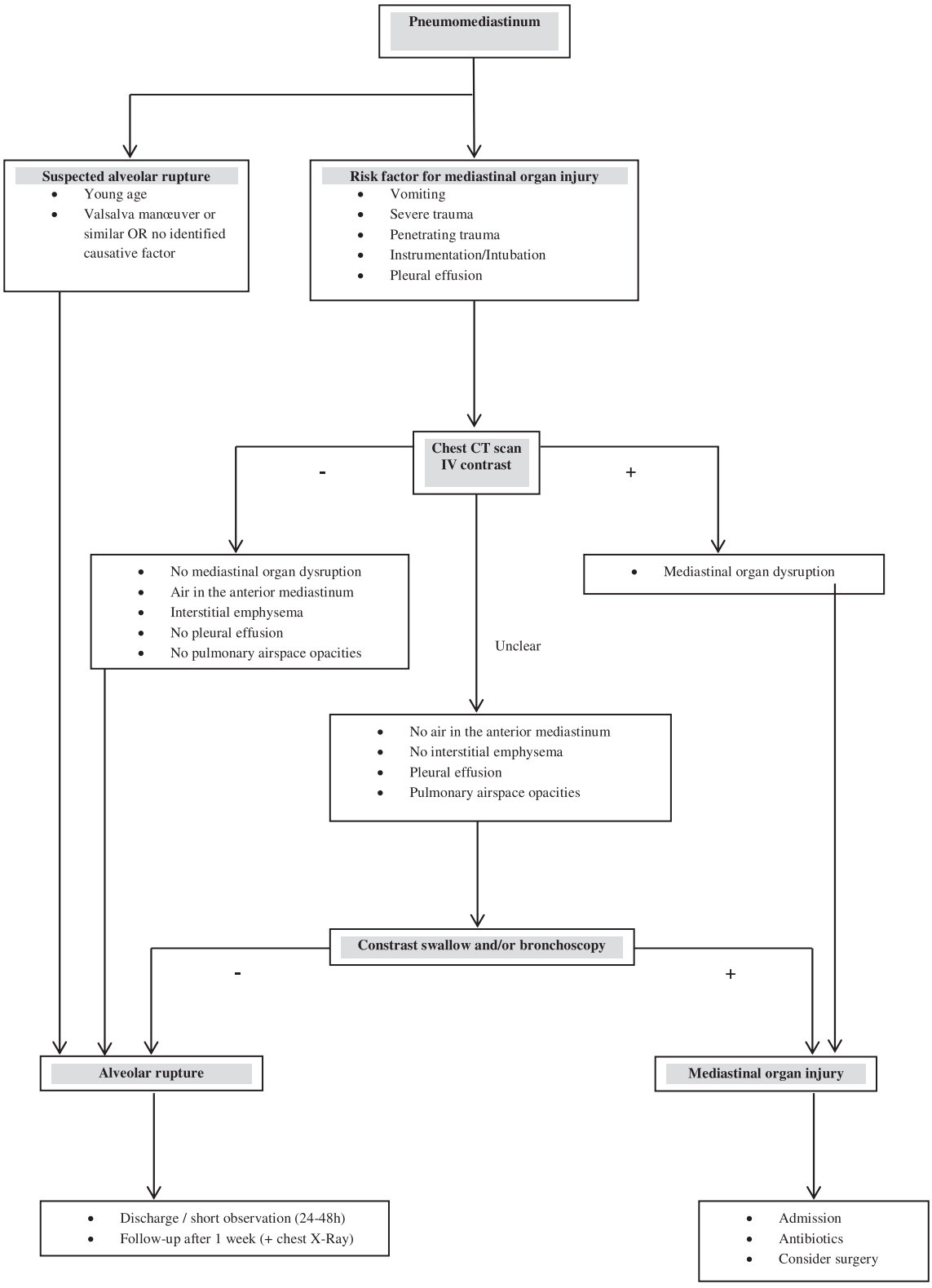

Figure 3. Suggested new classification and work-up algorithm of pneumomediastinum based on risk factors for mediastinal organ injury [6,7].

most of PM described as 'spontaneous' result of an alveolar rupture, some of them are at risk of MOI.

This misclassification made us to review the literature highlighting the epidemiology, risks factors and investigations to rule out MOI.

In the trauma patient population, PM is present in 2.7–10% and MOIs are found in only 0–10.3% of the cases. Risks factors are penetrating injury, severe trauma (high speed road traffic accident) and traumatic intubation. In the non-trauma population, MOIs are much more common with an incidence of 44%. Risks factors for an oesophageal injury are instrumentation, vomiting and pleural effusion and risks factors for an airway injury are oro-tracheal instrumentation,

mechanical ventilation and intubation. In all case, the finding of pneumothorax is a negative predictive factor of MOI [6].

Furthermore, chest CT scan findings are helpful in differentiating AR from MOI. Firstly, CT scan can show obvious signs of airway or oesophageal disruption. Secondly, AR is associated with air in the anterior mediastinum and with pulmonary interstitial emphysema (Macklin effect, Figure 1(C)), while MOI are associated with subdiaphragmatic air, pleural effusion and pulmonary airspace opacities. Invasive investigations are not required when the primary CT scan does not show any sign of MOI [7]. Moreover, chest CT should be used to confirm a suspected PM if chest X-ray is negative as the latter lack of sensitivity [8]

Our case report emphasizes the confusing classification of PM since our patient underwent an endoscopy and a hospital stay of 72 h for a 'secondary PM' despite no risk factor for MOI.

Therefore, we suggest a new work-up algorithm classifying PM between those at risk for MOI or not, properly reflecting the need of invasive investigations (Figure 3). This algorithm, based on retrospective studies [6,7], could be prospectively used for further validation.

In conclusion, PM work-up should be based on risk factors for MOI and chest CT findings to guide the need of invasive investigations rather than on current classification between spontaneous or secondary.

Disclosure statement

No potential conflict of interest was reported by the authors.

ORCID

Melanie Dechamps http://orcid.org/0000-0002-0648-1058
Lieven De Smedt http://orcid.org/0000-0003-0110-4533

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