

Intrapartum spontaneous pneumomediastinum: recognising and treating a rare entity

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Abstract

A 20-year-old nulliparous woman presented to our obstetric unit in spontaneous labour at 39+1 weeks gestation following an uncomplicated pregnancy. After a short labour she progressed to a spontaneous vaginal delivery of a live male infant. Immediately after delivery she experienced retrosternal chest pain, facial swelling and dyspnoea. On examination her chest was clear to auscultation, but extensive subcutaneous emphysema was noted over the chest, neck and face. She remained a diagnostic dilemma until a plain chest radiograph revealed a large volume pneumomediastinum and a CT chest and barium swallow excluded an oesophageal perforation leading to the diagnosis of Hamman's syndrome. She responded well to supportive cares and was discharged home on day three postpartum. Spontaneous pneumomediastinum or Hamman's syndrome has been rarely reported in the literature as a complication of labour. Although it runs a benign course it often masquerades as more lethal conditions and clinicians should have a good understanding of its diagnosis and management to ensure good outcomes.

Introduction

Chest pain and acute shortness of breath in the immediate postpartum period can be a sign of multiple life-threatening conditions. A rare cause of these symptoms is spontaneous pneumomediastinum, or Hamman's syndrome.

Pneumomediastinum is defined as the presence of free air in the mediastinal cavity¹. It was first described in association with labour by Hamman in 1945 and is estimated to complicate 1 in 100,000 deliveries^{1,2}. Elevated intrathoracic pressures generated by the Valsalva manoeuvre during labour are postulated to

cause alveolar rupture. Air is then able to track into the mediastinum along bronchovascular connective tissue planes. From there, air enters the subcutaneous tissues causing the subcutaneous emphysema which is pathognomonic of the condition. Although higher pressures can theoretically lead to rupture of the parietal pleura and the development of a tension pneumothorax, the condition generally runs a benign course, resolving spontaneously¹.

The tendency of the syndrome to masquerade as more lethal conditions means that, despite its rarity, Obstetric clinicians should be well equipped to recognise, appropriately manage and investigate Hamman's syndrome

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when it arises. We herein report the case of a spontaneous pneumomediastinum in a low risk primigravida with no significant medical history other than mild asthma. Symptoms developed immediately following an uncomplicated spontaneous vaginal delivery of a live male infant. We discuss in further detail the aetiology of the condition, important differential diagnoses to consider and the recommended management with particular focus on required imaging.

Case report

A 20-year-old nulliparous woman presented to our birth suite at 39+1 weeks gestation in spontaneous labour. She had no significant past medical history other than asthma for which she took regular inhaled long acting beta agonists and steroids. She had never been a smoker. Her antenatal course was similarly uncomplicated. She had initially presented eight hours earlier with prelabour rupture of membranes and was discharged home to await active labour. Following her second presentation her progress was swift and she progressed to a spontaneous vaginal delivery of a live male infant three hours after the onset of active labour with thirty minutes of pushing. She had a postpartum haemorrhage of 770mL which was managed with administration of 10 units of intramuscular oxytocin, an infusion of oxytocin 10 units/h over 4 hours and misoprostol 1000 micrograms, administered rectally. She sustained moderate bilateral labial tears which were sutured. Combined nitrous oxide and oxygen (N₂O:O₂ = 50%:50%) had been used as intrapartum analgesia and was continued during labial suturing.

The patient reported experiencing central chest pain and dyspnoea immediately following delivery however did not voice these concerns until the completion of suturing. The pain was aggravated by inhaling nitrous oxide, movements and lying flat. Increased facial swelling was noted by the patient's partner. The patient denied dry retching or vomiting during the course of her labour and had not experienced recent chest trauma. The obstetric registrar was called to review the patient two hours after delivery. The patient was tachycardic at 105 beats per minute but all other vital signs including respiratory rate and oxygen saturations were within normal range. The patient was speaking in full sentences with no obvious work of breathing. On examination subcutaneous emphysema was noted extending to the chest, neck and face. Nil tracheal deviation was

noted. The chest was clear to auscultation and there were dual heart sounds with no murmurs. An ECG was performed showing normal sinus rhythm. The patient was kept on close monitoring with 1:1 midwifery care.

The patient was reviewed an hour later with worsening painful respiration and chest tightness. The patient had persistent tachycardia at 134 beats per minute and had a new low-grade fever of 37.9°C. The palpable crepitus remained and on repeat auscultation of the chest there was concern about decreased breath sounds in the right upper zone. A plain chest X-ray was requested. This showed extensive pneumomediastinum with subcutaneous emphysema noted at the base of the neck bilaterally. Nil pneumothorax was identified. The findings of the X-ray were discussed with the cardiothoracic team at the nearest tertiary unit. A non-contrast CT chest was recommended to exclude oesophageal tears as well as a barium swallow. Supplemental oxygen via nasal prongs was recommended to help dissipate the emphysema. The CT chest and neck revealed a large volume pneumomediastinum with subcutaneous emphysema over the anterior chest wall and throughout the neck. No obvious oesophageal injury was identified. To complete the investigations, a barium swallow chest X-ray was performed two days postpartum which also revealed no perforation. A diagnosis of Hamman's syndrome was then made.



Figure 1. Chest Xray of the patient demonstrating pneumomediastinum and subcutaneous emphysema over the neck and upper chest.



Figure 2. Three axial slices from the non-contrast CT, again demonstrating extensive pneumomediastinum and subcutaneous emphysema.

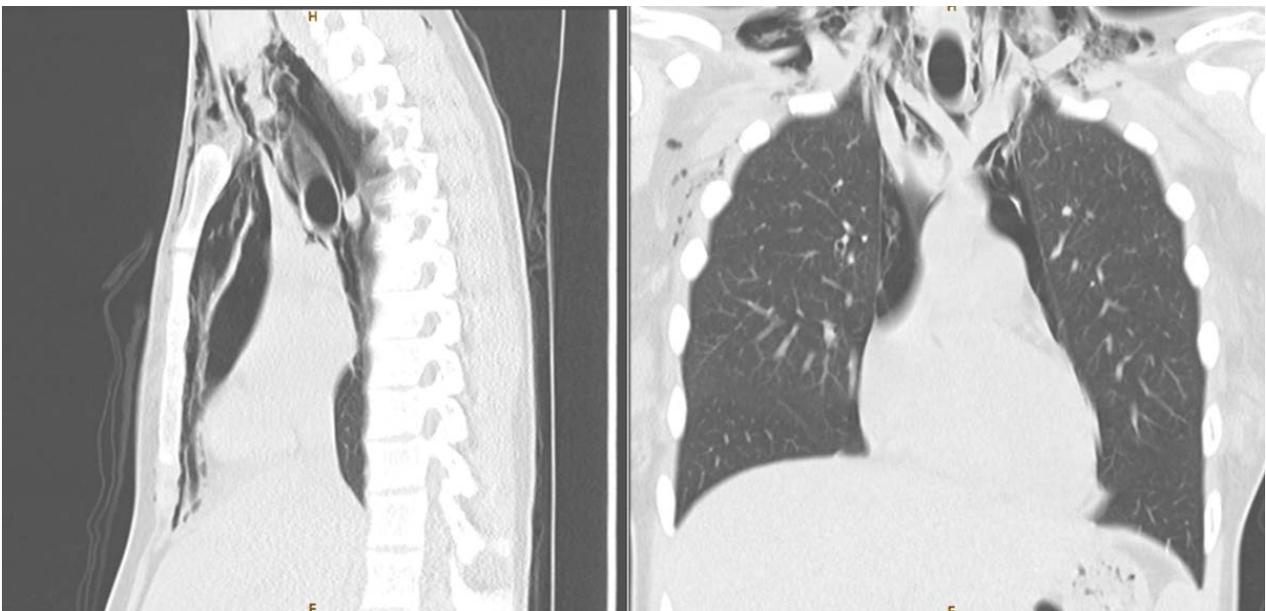


Figure 3. Two coronal and sagittal slices from the non-contrast CT, again demonstrating extensive pneumomediastinum and subcutaneous emphysema.

The patient remained in hospital for observation for three days postpartum. Her tachycardia and fevers normalised within the first 24 hours postpartum. Her symptoms of chest pain and shortness of breath improved dramatically, and she was able to mobilise without significant dyspnoea. She was discharged home on her regular inhalers. No specific follow up was necessary.

Discussion

When it comes to identifying the patient at risk of developing Hamman's syndrome, a specific risk factor profile has not been well defined. Although the phenomenon was not named until 1945, over the past

400 years there have been around 200 cases of Hamman's syndrome reported³. These cases suggest the syndrome is typically associated with young, healthy nulliparous women who experience long labours at term gestations^{1,3,4,5}. The syndrome typically arises during the second stage of labour, though it may not be clinically evident until postpartum^{1,3,5}. A prolonged labour, particularly a lengthy second stage logically increases a woman's risk of developing the syndrome as alveolar rupture is triggered by over-inflation from repeated valsalva, pushing, shouting screaming or vomiting³. However, the association of the syndrome with young women and first pregnancies may simply be coincidental. While asthma has not been well established as a risk factor for Hamman's syndrome

arising intrapartum, a published review of 201 patients in 2013 found that underlying asthma was associated with 25% of cases of spontaneous pneumo-mediastinum arising in non-pregnant individuals, generally following an acute attack⁶.

The most common presenting feature of Hamman's syndrome is chest pain. This is typically retrosternal and may radiate to the neck. Dyspnoea, cough, neck pain and dysphagia are also often present. A variety of other symptoms including odynophagia, light-headedness, numbness, dysphonia and back pain have also been reported⁶. On examination patients may be tachycardic and tachypnoeic with a mild fever⁴. The finding of subcutaneous emphysema is highly indicative of the syndrome as is the presence of Hamman's sign, a harsh sound heard over the praecordium during auscultation⁶. Important differential diagnoses that must be considered and excluded include pulmonary emboli, amniotic fluid emboli, aortic dissection, myocardial infarction, anaphylaxis, toxicity from injectable drugs and mediastinitis from Boerhaave syndrome or oesophageal rupture.

Plain film chest X-rays are the single most important clinical investigation for identifying pneumomediastinum³. Free air in the mediastinum may be indicated on plain film by the classical 'ring sign' caused by air surrounding the intramediastinal portion of the pulmonary artery as it leaves the heart⁶. There is mixed evidence regarding whether a Computerized Tomography (CT) scan of the chest, as performed in our own case, is necessary in addition to a plain film. There are two main reasons why a CT might be considered. Firstly, it has been suggested that extensive subcutaneous emphysema may make X-ray interpretation and hence the detection of a pneumomediastinum more difficult. Secondly and perhaps more crucially, clinicians may be concerned about missing the potentially fatal diagnosis of mediastinitis from oesophageal rupture. In the case report and review of literature by Wozniak and Blackburn in 2011 it was suggested that posterior anterior and lateral X-ray films are the only investigations needed unless there is significant concerns of mediastinitis based on clinical history or examination. Spontaneous oesophageal rupture very rarely occurs in the setting of a labouring woman and is generally preceded by a period of repeated vomiting. If mediastinitis has occurred patients generally become very unwell with ongoing fevers and septic shock in contrast to the benign course of pneumomediastinum alone⁴. A review of 33 cases of

spontaneous pneumomediastinum by Kaneki et al in 2000, found that chest X-ray alone was able to detect pneumomediastinum in over 90% of cases. However, in spite of this finding the authors of this report suggested a liberal use of CT to diagnose pneumomediastinum given in 30% of these cases X-rays had no or only very subtle evidence of pneumomediastinum. It was felt that omitting a CT scan may lead to a missed diagnosis by a less experienced clinician⁷. Barium swallows were recommended only if there is abnormality detected on CT⁶.

Hamman's syndrome generally runs a benign course and once the diagnosis is confirmed supportive therapy is typically the only treatment necessary. Oxygen and bed rest are recommended to facilitate absorption of the free air and resolution of the pneumomediastinum⁶. Recovery generally occurs within 3-14 days⁸. Given the increased alveolar pressures needed to generate a pneumothorax, these occur extremely infrequently. However, signs of tension pneumothorax should prompt emergency decompression⁶. If Hamman's syndrome is detected intrapartum the use of inhaled nitrous oxide is not recommended as it leads to an increase in air trapping. Similarly, positive pressure ventilation should be avoided if a caesarean delivery is required. Liberal use of regional anaesthesia is encouraged to allow increased passive descent and decreased maternal pushing effort. Outlet instrumental deliveries may be also be employed to reduce maternal valsalva manoeuvre⁹. Recurrence has been reported in two cases only, both occurring before 1900⁸. Use of outlet forceps as prophylaxis against Hamman's syndrome in subsequent deliveries has been suggested but is largely considered to be unnecessary due to the low likelihood of recurrence⁸.

We hope that our report and discussion is informative for other Obstetric units about the signs, investigations and management of this rare and interesting condition. While, given the benign nature of Hamman's syndrome, no lasting harm came to our patient, upon reflection of our management it is evident how our approach was skewed by our lack of experience with the syndrome. Despite our patient having key predisposing factors and classical signs and symptoms arising almost immediately postpartum, Hamman's syndrome was not diagnosed until nearly 48 hours following delivery. Given the rarity of the condition and the broad ranging risk factors it would be ineffective to identify at risk women antenatally. However, knowledge of Hamman's syndrome and its predisposing factors might have helped our team to identify the diagnosis sooner.

On review of the literature we can also conclude that while chest radiographs are the most important investigation in cases of Hamman's syndrome the use of chest CT should be encouraged if there is clinical suspicion of mediastinitis. Although our patient did not have any significant vomiting during her labour and pneumomediastinum was easily detected on chest radiograph, her persistent low-grade fever and tachycardia during the first 24 hours postpartum justified the use of CT to exclude oesophageal injury. While it is ideal to minimise unnecessary exposure to radiation, especially in a population of generally young and otherwise healthy women it is equally important not to miss or delay treatment for a more lethal condition such as mediastinitis. Knowledge of the existence of and presenting symptoms of Hamman's syndrome is recommended for all clinicians tending to labouring mothers, not only to avoid excessive imaging when it is not indicated, but also to reduce stress caused to both clinician and patient about a more sinister underlying pathology.

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