Spontaneous pneumomediastinum after uncomplicated vaginal delivery

Introduction

Hamman's syndrome is a rare condition characterized by spontaneous pneumomediastinum with subcutaneous emphysema, classically seen in the postpartum period in young, previously healthy primiparous women. It is thought that the prolonged Valsalva effects of pushing in labour can lead to this phenomenon. The incidence is roughly 1 in 100 000 deliveries. Hamman's syndrome has been reported in all stages of labour, but it is most commonly diagnosed postnatally.

Discussion

The diagnosis of Hamman's syndrome was made based on the history, clinical findings and chest radiograph. Spontaneous postnatal pneumomediastinum associated with subcutaneous emphysema, also known as Hamman's syndrome, was first described by Louis Virgil Hamman in 1945. Hamman's syndrome has been reported in all stages of labour (Dudley and Patten, 1988); however, it is most commonly diagnosed postnatally but is thought to occur during the second stage of labour (Majer and Graber, 2007). The use of inhalational drugs is also suspected to be contributory (Cho et al, 2015). When diagnosed during labour there is a school of thought that the delivery should be 'accelerated' via instrumental delivery to prevent progression of mediastinal emphysema.

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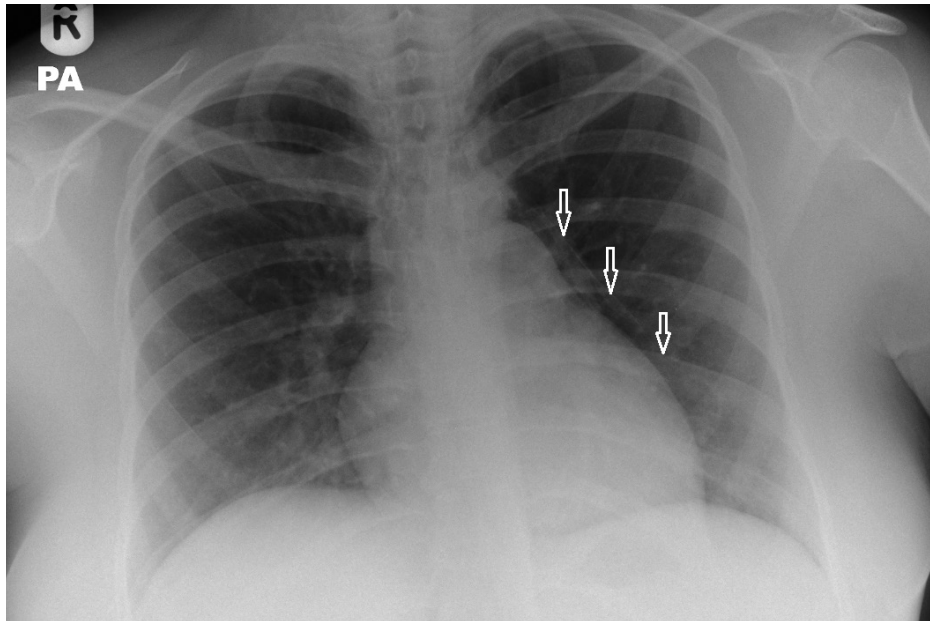
Figure 1. Chest radiograph showing an unusual oblique line (white arrows) running parallel to the left upper heart border.



CASE REPORT

A 21-year-old primiparous woman presented to triage at 38+ weeks gestation complaining of spontaneous rupture of membrane but no contractions. Speculum examination confirmed leaking pink liquor but the os was long and closed. She was monitored and transferred to labour ward where a healthy male infant weighing 2816 g was spontaneously delivered after 6 hours and 58 minutes of labour. The patient required Entonox and pethidine as pain relief in labour.

She had had an uncomplicated pregnancy, estimated as low risk at booking which took place at 12+3 weeks and there were no antenatal admissions. Past medical and surgical histories were both unremarkable.

She was not on any regular medications other than prenatal vitamins. She was a non-smoker, teetotal and lived at home with her husband as a housewife.

Three hours after delivery and now on the postnatal ward she complained of acute shortness of breath, pain in her neck and shoulder, and reported that her skin felt 'bubbly'. Her observations were stable with a blood pressure of 111/64 mmHg and oxygen saturations of 97% on room air with a respiratory rate of 16 breaths/min. On physical examination the chest was clear with bilateral air entry but there was marked crepitus felt on the anterior chest wall indicating subcutaneous emphysema. Crepitus was not audible with the heartbeat (Hamman's sign).

An arterial blood gas showed a pH of 7.453 (normal range (NR) 7.350–7.450), pCO_2 of 4.10 kPa (NR 4.67–6.40 kPa), pO_2 of 13.5 kPa (NR 11.1–14.4 kPa), HCO_3 of 22.7 mmol/litre (NR 22–28 mmol/litre) and lactate of 2.9 mmol/litre (NR 0.5–1.6 mmol/litre). All other blood results were unremarkable and an electrocardiogram demonstrated normal sinus rhythm. A chest radiograph (*Figure 1*) was reported as 'lungs clear, with no evidence of surgical emphysema. A thin linear opacity, adjacent and parallel to the left upper heart border, is unusual and pneumomediastinum should be considered.'

The patient in this case remained in hospital for 24 hours of observation and conservative management, remaining haemodynamically stable throughout admission. She was discharged home once the discomfort had settled and subcutaneous emphysema had decreased.

The most recognized theory currently is that the act of labour and prolonged Valsalva manoeuvres produced during labour from pushing leads to the rupture of marginal alveoli (Kouki and Fares, 2013). The increase in thoracic pressure causes a pressure gradient and thus movement of air into the mediastinum. Once in the mediastinum, the air can migrate to the subcutaneous tissues leading to subcutaneous emphysema.

Shortness of breath and chest pain post-partum is a worrying sign and it is important to consider and exclude several life-threatening differentials, including pulmonary embolism, myocardial infarction, pneumothorax, aortic dissection and tamponade. However, Hamman's syndrome is usually a benign self-limiting condition, treated with supportive management, oxygen and analgesia. A rare and sinister complication is cardiac tamponade caused by development of a pneumopericardium. There is very little evidence as to what role this diagnosis plays in subsequent pregnancy and whether women diagnosed with Hamman's syndrome should be managed differently. **BJHM**

Cho C, Parratt JR, Smith S, Patel R (2015) Spontaneous pneumomediastinum (Hamman's syndrome): a rare cause of postpartum chest pain. *BMJ Case Rep* https://doi.org/10.1136/bcr-12-2010-3603

LEARNING POINTS

- Chest radiography is a vital diagnostic tool in the diagnosis of Hamman's syndrome.
- This case highlights the need to consider important differential diagnoses in post-partum patients with chest pain and shortness of breath.

Dudley DK, Patten DE (1988) Intrapartum pneumomediastinum associated with subcutaneous emphysema. *CMAJ* **139**(7): 641–642.
Kouki S, Fares AF (2013) Postpartum spontaneous pneumomediastinum 'Hamman's syndrome'. *BMJ Case Rep* https://doi.org/10.1136/bcr-2013-010354
Majer S, Graber P (2007) Postpartum pneumomediastinum (Hamman's syndrome). *CMAJ* **177**(1): 32. https://doi.org/10.1503/cmaj.061581

Two cases of cocaine nasal insufflation-induced pneumomediastinum

Two isolated, yet almost identical cases of young and previously healthy adults presented to the emergency department with pneumomediastinum on the same day (*Figures 1* and *2*). Both presented with dyspnoea, one with some associated chest tightness (patient A) and the other with associated neck tightness (patient B). Patient B had clinical and radiological evidence of surgical emphysema (*Figure 2*). Both admitted to 'snorting' cocaine before symptom onset.

Spontaneous pneumomediastinum is recognized in cocaine use (Morris and Shuck, 1985), and is a result of increased alveolar pressures during snorting, leading to rupture and air escaping into interstitial tissues. The locally damaging effect of cocaine on alveoli can further breach alveolar integrity causing pneumomediastinum (Maunder et al, 1984; Gotway et al, 2002).

A review of 25 case studies of spontaneous pneumomediastinum highlighted it as a largely benign condition that can be managed conservatively, so further investigation, e.g. with computed tomography, is unwarranted. Symptoms resolved in a median of 24 hours (Alnas et al, 2010). Both patients were managed conservatively and made a full, uncomplicated recovery. **BJHM**

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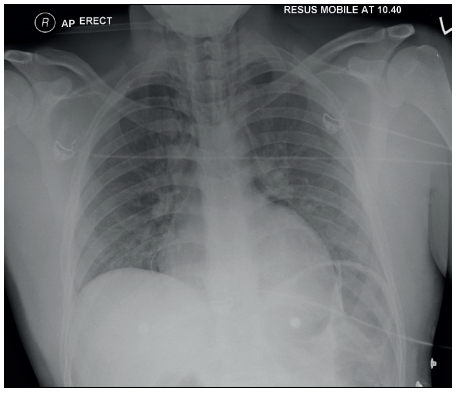
Figure 1. Patient A – comparison lines are seen surrounding the heart and upper mediastinum, consistent with a pneumomediastinum.



Alnas M, Altayeh A, Zaman M (2010) Clinical course and outcome of cocaine-induced pneumomediastinum. *Am J Med Sci* **339**(1): 65–67. https://doi.org/10.1097/MAJ.0b013e3181c371da
Gotway MB, Marder SR, Hanks DK et al (2002) Thoracic complications of illicit drug use: An organ system approach. *Radiographics* **22** Spec No: S119–S135. https://doi.org/10.1148/radiographics.22.suppl_1.g02oc01s119
Maunder RJ, Pierson DJ, Hudson LD (1984) Subcutaneous and mediastinal emphysema, pathophysiology, diagnosis and management. *Arch Intern Med* **144**: 1447–1453. https://doi.org/10.1001/archinte.1984.00350190143024
Morris JB, Shuck JM (1985) Pneumomediastinum in a young male cocaine user. *Ann Emerg Med* **14**(2): 194–196. https://doi.org/10.1016/S0196-0644(85)81104-5

Figure 2. Patient B – there is a pneumomediastinum with extensive gas in the soft tissues of the neck.