Spontaneous pneumomediastinum secondary to hyperemesis gravidarum: A case report and principles of recognition and management

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Abstract

Pneumomediastinum is the presence of aberrant air in the mediastinum and is most commonly caused by oesophageal or alveolar rupture. Hyperemesis gravidarum is persistent nausea and vomiting before the 20th week of pregnancy and can increase intrathoracic pressure, precipitating pneumomediastinum.

A 22-year-old patient presented with hyperemesis gravidarum in the 6th week of pregnancy. During her hospital admission, she developed chest pain, and imaging showed pneumomediastinum. Endoscopy excluded oesophageal perforation, a diagnosis of spontaneous pneumomediastinum was made, and her symptoms improved with conservative management.

This case demonstrates how oesophageal perforation and spontaneous mediastinum can present in similar fashion. Oesophageal perforation has high morbidity and mortality and it is vital to identify it early. It is therefore important that clinicians are aware of pneumomediastinum as a potential complication of hyperemesis gravidarum and exclude oesophageal perforation in these individuals.

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1. Introduction

Pneumomediastinum is the presence of aberrant gas within the mediastinum. It is associated with the classic triad of symptoms of chest pain, subcutaneous emphysema and dyspnoea [1]. The gas can originate from the oesophagus, lungs, airways or abdomen, and can track through fascial planes. Pneumomediastinum precipitated by increased intraoesophageal or high intra-alveolar pressures caused by forceful coughing, vomiting or inhalational drug use is well documented [2].

Persistently high intrathoracic pressure can be generated during pregnancy due to hyperemesis gravidarum (HG) or a prolonged second stage of labour [3]. HG is present in 0.3–0.4% of pregnancies and is defined in 0.3–0.4% of pregnancies and is defined as protracted nausea and vomiting with a triad of more than 5% pre-pregnancy weight loss, dehydration and electrolyte imbalance [4].

There is evidence in the literature of rare cases of pneumomediastinum secondary to HG [5,6]. Whilst most examples are due to spontaneous pneumomediastinum, some cases are secondary to oesophageal rupture [7].

In this paper we present a patient with pneumomediastinum secondary to HG, discuss the approach for diagnosis and the importance of ruling out oesophageal rupture.

2. Case Presentation

We report the case of 22-year-old patient of African origin, gravida 3, para 1, at 6 + 2 weeks of pregnancy. She presented to her local maternity unit with intractable vomiting and nausea. Her symptoms persisted despite the use of oral cyclizine. Her medical history included one previous miscarriage and hyperemesis gravidarum during a previous pregnancy. She was admitted and treated with intravenous fluids and intravenous ondansetron, with good response.

Following a two-day admission, she was discharged but unfortunately re-presented 6 days later with recurrence of symptoms and 5 kg weight loss relative to her pre-pregnancy weight. Further supportive treatment was given and the dieticians were involved to optimise her nutrition. Parenteral nutrition was not required at this stage. Transvaginal ultrasound confirmed a viable intrauterine pregnancy. She was discharged 3 days later with oral antiemetics.

The patient was reviewed in the outpatient clinic of the same obstetric unit 10 days after discharge. Symptoms of nausea and vomiting were persistent and further a 4.4 kg of weight loss was measured. The patient was afebrile, with a blood pressure of 116/78 mmHg and heart rate of 92 beats/min. Admission was organised directly from clinic and the patient was treated with intravenous fluids and antiemetics. On day 3 of admission she developed intermittent central chest pain associated with palpitations. Basic observations remained within normal range and serum
troponin was not raised. An electrocardiogram (ECG) showed T wave inversion in leads V1-V5. A chest radiograph (CXR) was performed at this stage (Fig. 1), which excluded pneumothorax but showed demarcation along the left side of the heart, a good indication of pneumomediastinum. A computed tomography (CT) pulmonary angiogram was performed to exclude pulmonary embolus. The CT scan did not show evidence of pulmonary embolism but revealed pneumomediastinum suggestive of spontaneous oesophageal rupture. After discussion with upper gastrointestinal (UGI) specialists she underwent CT examination of the chest and abdomen with oral contrast. The CT scan confirmed pneumomediastinum and also revealed a small left pneumothorax. There was no mediastinal collection and no extravasation of contrast from the oesophagus. The patient was transferred to the UGI unit for oesophago-gastro-duodenoscopy (OGD), which revealed mild lower oesophageal inflammation but excluded oesophageal rupture. A diagnosis of spontaneous pneumomediastinum was made and the patient was discussed in the respiratory multidisciplinary team meeting. The patient was treated conservatively and monitored with serial chest radiographs (2 further radiographs), which showed improvement and then resolution of the pneumomediastinum. The patient was discharged after a 7-day stay in hospital following resolution of symptoms and return to baseline weight. She has since remained clinically well.

3. Discussion

Pneumomediastinum has previously been reported to represent 1 in 25,000–42,000 hospital admissions [8]. Pneumomediastinum can be secondary to alveolar rupture, where it is referred to as spontaneous pneumomediastinum, or can develop secondary to oesophageal rupture, known as Boerhave syndrome. Whilst oesophageal rupture and spontaneous pneumomediastinum may initially present in similar fashions, their mechanisms, management and prognosis are very different.

Alveolar rupture develops due to a sudden rise in the intra-alveolar pressure in the lung periphery, causing an air leak [8]. The air travels centrally to the hilum due to the pressure gradient between the peripheral lung and the mediastinum. Pneumothorax may occur if the pressure rises too quickly [9]. Known precipitating factors include but are not limited to coughing, vomiting, abuse of inhalational drugs or acute asthma attack [10,11]. Spontaneous pneumomediastinum has also been well documented to occur when straining against a closed glottis in a Valsalva-type manoeuvre, such as during labour [1,9].

Oesophageal tears during pregnancy are rare but can be associated with hyperemesis gravidarum [7]. Patients with Boerhave syndrome have the potential to become unwell extremely quickly due to respiratory compromise and septic shock. It is often misdiagnosed as it presents clinically very similarly to tension pneumothorax, cardiac tamponade and dissecting thoracic aorta [12].

Once pneumomediastinum has been identified, the source of air must be determined. In a systemically well patient it can be difficult to distinguish clinically whether the pneumomediastinum is secondary to alveolar or oesophageal rupture. However, this distinction has significant implications for the morbidity and mortality of the patient. Overall, oesophageal rupture has a 30% mortality rate, but effective treatment within 24 h has been shown to decrease this significantly [13]. Complications of oesophageal rupture include mediastinitis, pneumonitis, empyema and pericarditis. Patients who present with pneumomediastinum secondary to oesophageal rupture are more likely to be systemically unwell than those who have suffered alveolar rupture. A previous case series found all but one patient with spontaneous pneumomediastinum were systemically well at presentation [14].

If pneumomediastinum is due to oesophageal perforation, initial treatment should involve fluid resuscitation, broad-spectrum intravenous antibiotics and immediate surgical consultation. This contrasts with the management for spontaneous pneumomediastinum, which is often conservative. Management is with simple analgesia, anti-emetics and supplemental oxygen if required [15,16]. The symptoms and condition usually settle over a few days and it has even been suggested that admission may not be necessary [16]. Recurrence is uncommon, as are complications such as tension pneumothorax, pneumopericardium and air embolism [15,16].

Spontaneous pneumomediastinum secondary to HG is exceedingly rare, with most examples in the literature being in the form of case reports and series. This case demonstrates the clinical presentation,
investigations and management of a patient with pneumomediastinum secondary to the symptoms of HG. Whilst rare, it is important that clinicians are aware of pneumomediastinum as a potential consequence of HG in pregnancy and are able to tailor their management based on its aetiology.

4. Strengths and Limitations

4.1. Strengths

Documentation, patient observations, blood results and imaging were all recorded electronically and so were easily interpretable and readily available. There was little time between case presentation and the write-up, so information and references are all recent.

4.2. Limitations

Whilst our case report highlights the importance of differentiating between spontaneous pneumomediastinum and oesophageal perforation, these are the lessons learnt from our case and may be different for other patients. Generalisations from this case must be interpreted with caution.

Contributors

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Patient Consent

Obtained.

References

[1] J.S. Gorbach, F.L. Counselman, M.H. Mendelson, Spontaneous pneumomediastinum secondary to hyperemesis gravidarum, J. Emerg. Med. 15 (5) (1997 Oct) 639–643.
[2] V.K. Kouritas, K. Papagianopoulos, G. Lazaridis, S. Baka, I. Mpoukonivas, V. Karavasilis, et al., Pneumomediastinum, J. Thorac. Dis. 7 (Suppl. 1) (2015 Feb) 544–549.
[3] A. LoMauro, A. Aliverti, Respiratory physiology of pregnancy: physiology masterclass, Breathe (Sheff) 11 (4) (2015 Dec) 297–301.
[4] Royal College of Obstetrics and Gynaecologists, Green Top Guidelines: The Management of Nausea and Vomiting of Pregnancy and Hyperemesis Gravidarum [Internet], NICE, 2016, [cited 2020 May 29]. Available from: https://www.rcog.org.uk/globalassets/documents/guidelines/green-top-guidelines/gtg69-hyperemesis.pdf.
[5] E.M. Karson, D. Saltzman, M.R. Davis, Pneumomediastinum in pregnancy: two case reports and a review of the literature, pathophysiology, and management, Obstet. Gynecol. 64 (3 Suppl) (1984 Sep) 39S–43S.
[6] T. Yamamoto, Y. Suzuki, K. Kojima, T. Sato, M. Tanemura, M. Kaji, et al., Pneumomediastinum secondary to hyperemesis gravidarum during early pregnancy, Acta Obstet. Gynecol. Scand. 80 (12) (2001 Dec) 1143–1145.
[7] S.-G. Liang, F. Ooka, A. Santo, M. Kaibara, Pneumomediastinum following esophageal rupture associated with hyperemesis gravidarum, J. Obstet. Gynaecol. Res. 28 (3) (2002 Jun) 172–175.
[8] A.C. Ralph-Edwards, F.G. Pearson, Atypical presentation of spontaneous pneumomediastinum, Ann. Thorac. Surg. 58 (6) (1994) 1758–1760.
[9] M. Schwartz, L. Rossoff, Pneumomediastinum and bilateral pneumothoraces in a patient with hyperemesis gravidarum, Chest 106 (6) (1994 Dec) 1904–1906.
[10] S.M. Janes, P.W. Ind, J. Jackson, Images in thorax. Crack inhalation induced pneumomediastinum and bilateral pneumothoraces in a patient with hyperemesis gravidarum, Thorax 59 (4) (2004 Apr) 360.
[11] D.J. McMahon, Spontaneous pneumomediastinum, Ann. J. Surg. 131 (5) (1976 May) 550–551.
[12] G.M. Buchanan, V. Franklin, Hamman and Boerhaave syndromes - diagnostic dilemmas in a patient presenting with hyperemesis gravidarum: a case report, Scott. Med. J. 59 (4) (2014 Nov) e12–e16.

Provenance and Peer Review

This case report was peer reviewed.

Declaration of Competing Interest

The authors declare that they have no conflict of interest regarding the publication of this case report.

Fig. 2. CT examination of the chest and abdomen with oral contrast, confirming pneumomediastinum and also revealing a small left pneumothorax. There is no mediastinal collection and no extravasation of contrast from the oesophagus.
[13] M. Ahadi, N. Masoudifar, Boerhaave syndrome, J. Cardio-Thoracic Med. (2017 Dec) https://doi.org/10.22038/jctm.2017.26492.1143[Internet]. [cited 2020 May 29]; (Online First). Available from:

[14] M.J. Forshaw, A.Z. Khan, D.C. Strauss, A.J. Botha, R.C. Mason, Vomiting-induced pneumomediastinum and subcutaneous emphysema does not always indicate Boerhaave’s syndrome: report of six cases, Surg. Today 37 (10) (2007) 888–892.

[15] G.J. Koullias, D.P. Korkolis, X.J. Wang, G.L. Hammond, Current assessment and management of spontaneous pneumomediastinum: experience in 24 adult patients, Eur. J. Cardiothorac. Surg. 25 (5) (2004 May) 852–855.

[16] J.B. Jougon, M. Ballester, F. Delcambe, T. Mac Bride, C.E.H. Dromer, J.-F. Velly, Assessment of spontaneous pneumomediastinum: experience with 12 patients, Ann. Thorac. Surg. 75 (6) (2003 Jun) 1711–1714.