

Bullous emphysema — an incidental observation during dissection*

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Brandon J. CRIVELLO Anita JOY +	During routine dissection, a pulmonary bulla was observed on the diaphragmatic surface of the right lung of an 87 year old male donor. Examination revealed a possible case of bullous emphysema. Bullous emphysema results when pulmonary bullae, defined as an air-filled space >1cm diameter and with a wall thickness <1mm, form as a consequence of alveolar breakdown in a normal lung, or as a sequela of chronic obstructive pulmonary disease (COPD) with resultant emphysema. The dissection team documented this anatomical variation and used this as a learning experience to conduct a detailed literature review of the pathogenesis, mechanisms, treatment options and clinical management of these patients in a dental setting.
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Introduction

Cystic spaces observed in chest radiographs are typically referred to as 'bullae', and patients most often receive a clinical diagnosis of 'bullous emphysema'. The term 'bulla' refers to an air-filled space that may be subpleural or intrapulmonary in location, with a diameter >1cm and a wall <1mm thick [1–3]. Bullous emphysema results when pulmonary bullae form as a consequence of alveolar breakdown in a normal lung, or as a sequela of chronic obstructive pulmonary disease (COPD) with resultant emphysema. Bullous emphysema may also be considered as a progressive complication of pulmonary emphysema. Here we report an incidental finding of a discrete pulmonary bulla during routine cadaver dissection.

Case Report

Upon removal of the right lung from the thoracic cavity of an 87 year old male donor, the dissection team noted the presence of a distinctive structure on the diaphragmatic surface of the lung. This anatomical variation resembled a balloon-like dilation protruding out from otherwise normal lung tissue, and was not adherent to the underlying diaphragm. The site of attachment on the lung displayed a defined concavity (Figure 1a, 1b).

* Part of this manuscript was presented at the 2013 Southern Illinois University School of Dental Medicine Research Day at Alton, IL, USA on April 9, 2013. The dilation which measured approximately 3cm in diameter, appeared inflated with a thin, translucent wall. A vascular network resembling trabeculae was clearly evident within the wall of the dilation (Figure 1c). Palpation revealed that the unusual structure was an air-filled space. Application of finger pressure caused an indentation of the surface which resolved when pressure was eliminated, indicating a direct connection with the lung proper.

The team proposed a differential diagnosis of emphysema, bullous emphysema, and metastatic neoplasm, based on the gross observations. The specimen was photographed and adjacent lung tissue collected for routine histological examination. The adjacent lung tissue exhibited normal histological features as seen on routine Hematoxylin and Eosin staining (Figure 2a and 2b). Based on the documented morphological features, literature review and expert opinions of clinicians/pathologists, the dissection team confirmed the observed variation as a case of 'bullous emphysema'.

Discussion

Histologically, pulmonary bullae are characterized by an outer surface composed of visceral pleura and an inner surface consisting of destroyed, fibrous lung tissue [3]. The internal structure contains trabeculae of fibrotic interlobular and alveolar septae [4]. Pulmonary bullae can be histologically



Figure 1. Photographs showing the morphological features observed. **a)** The right lung shows the presence of a balloon-like dilation located on the diaphragmatic surface. **b, c)** Close-up view of the dilation/bulla reveals the thin translucent wall, indentation of the surface caused by application of finger pressure and the vasculature within the wall.



Figure 2. Histological images of tissue adjacent to the bulla showing epithelial characteristics of normal lung tissue. **a)** Low magnification image. **b)** Higher magnification of the area outlined in blue in Figure 2a, shows thin-walled alveoli formed by a single layer of squamous epithelial cells. Thin layers of connective tissue and blood vessels can also be seen in the field of view.

differentiated from pulmonary cysts and pulmonary blebs. Pulmonary cysts are developmental in origin and are lined by respiratory epithelium, while pulmonary blebs are small subpleural air collections, usually caused by ruptured alveoli [3, 4]. Pathogenesis is attributed to a combination of a protease-anti protease imbalance, and an oxidantantioxidant imbalance, both of which are augmented by leukocytes [5]. These mechanisms share common etiologies, with the most common form of bullous emphysema being associated with chronic smoking. Other mechanisms include advanced pulmonary emphysema, pulmonary sarcoidosis, pneumoconiosis, α 1-anti trypsin deficiency, Marfan's syndrome and Ehlers-Danlos syndrome [6].

One classification of bullae takes into account the anatomy of the bulla and the histology of the underlying lung tissue. Based on this classification: Group I – single large bulla with normal underlying lung; Group II – multiple bullae with normal underlying lung; Group II – multiple bullae with diffuse emphysema of underlying lung; Group IV – multiple bullae with underlying lung affected by other diseases, the anatomical variant that we documented was a Group I bulla. This classification is typically used to qualify patients for surgical intervention. Patients with Group I and II bullae are typically the best candidates for surgery [3, 7].

The clinical significance of bullous emphysema includes compression of adjacent lung tissue and subsequent respiratory distress, fluid collection, potential infection and rupture leading to pneumothorax [5]. Degenerative alterations of the terminal bronchioles which constrict their lumens, lead to alveolar distention and resultant emphysema. These changes are irreversible, and reduce the total surface area available for alveolar gas exchange. Radiographically, an associated pneumothorax is seen as a "double-wall sign" on a chest CT. This indicates the presence of air on both sides of the bulla wall [8].

If selected for surgical intervention, one of two surgical approaches is currently used to resect giant lung bullae. The most common technique is a stapling resection of the entire bulla, using Video Assisted Thoracoscopic Surgery (VATS) [2, 7, 9, 10]. Another surgical approach is the modified Monaldi technique, which involves opening the bulla, placing a pursestring suture at the neck of the bulla and closing the overlying bullous sac with a running back-and-forth plicating stitch [11]. Smoking cessation and aggressive pulmonary rehabilitation are critical for successful treatment of patients with bullous lung disease.

In the dental setting, it is vital for the clinician to be able to identify a patient with the cardinal signs of COPD, including, dyspnea on exertion, chronic cough and sputum production. Sputum production is often minimal when cough is present. These patients are generally thin and barrel-chested. Auscultation reveals poorly discernible breath sounds and hyperresonance to percussion of the thorax. As is typical in emphysema, clinical signs have less diagnostic value than radiographs that show evidence of overinflation of the lungs and constricted peripheral vasculature. In patients diagnosed with emphysema or COPD, appropriate management is important not only for successful dental treatment, but also for a safe and comfortable dental appointment for the patient. Some key precautions with these patients in a dental setting include, avoiding immediate treatment if an upper respiratory infection is present, keeping the dental chair upright to maintain vital capacity, monitoring with pulse oximetry. supplementing with low-flow O2 (below 3L/min) and avoiding nitrous/02 inhalation sedation in severe cases [12-15].

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