I read with interest the Chest Film Clinic on pneumomediastinum by Weinstock, Boiselle, and Roberts in the August issue (What caused this woman's pneumomediastinum? J Respir Dis. 2008;29:314-317). In the discussion of the differential diagnosis, the authors did not mention the occurrence of mediastinal emphysema in diabetic ketoacidosis, which was described in 4 patients by Beigelman and associates in 1969.

To the Editor:

I read with interest the Chest Film Clinic on pneumomediastinum by Weinstock, Boiselle, and Roberts in the August issue (What caused this woman's pneumomediastinum? J Respir Dis. 2008;29:314-317). In the discussion of the differential diagnosis, the authors did not mention the occurrence of mediastinal emphysema in diabetic ketoacidosis, which was described in 4 patients by Beigelman and associates in 1969. McNicholl and associates ascribed the pneumomediastinum to the expiratory effort and grunting associated with ketotic hyperventilation. Munsell reviewed 28 cases of spontaneous pneumomediastinum and suggested that an acute transient respiratory obstruction, such as that produced by Valsalva maneuver, cough, emesis, or asthma, was the precipitating factor. Why doesn't mediastinal emphysema develop in a marathon runner during a long-distant run? In this situation, the breathing is normal and brain center-dependent, and it is tapered when the exertion becomes intolerable. (The marathoner is also presumably physically fit and healthy.) In contrast, in diabetic ketoacidosis, the Kussmaul respiration is involuntary and independent of the respiratory center and abnormally expands lung air spaces, causes alveolar rupture, and results in pneumothorax. As the pulmonary and intrathoracic pressures increase, the extra-alveolar air slips through the periadventitial tissue to the mediastinum, subcutaneous tissue, and other anatomic areas. The incidence of pneumomediastinum complicating diabetic ketoacidosis is low. Cases are still anecdotal and are sporadically published as one or a few case reports; they are most likely underestimated because the symptom of breathlessness tends to be overshadowed by hyperventilation in diabetic ketoacidosis. Occasionally, a crackling or crunching sound synchronous with the heartbeat, or Hamman sign, is audible over the left sternal edge. Accompanying retrosternal pain is uncommon. Several factors may contribute to the development of pneumomediastinum. Severe vomiting in diabetic ketoacidosis produces a Valsalva-like effect with large momentary swings in intrathoracic pressure, which can lead to alveolar rupture. This is much more likely to occur when alveoli are already susceptible to over-distention during Kussmaul respiration. Subcutaneous emphysema in the neck may be present in about half the cases. Interestingly, pneumomediastinum associated with severe diabetic ketoacidosis has also been described in the absence of cough and vomiting. In patients with no specific symptoms of pneumomediastinum, the diagnosis may be missed unless a chest radiograph is obtained. Pneumomediastinum complicating diabetic ketoacidosis is more likely to occur in males than in females, and the typical patient is young-usually younger than 20 years; the oldest reported patient was 29. The major differential diagnosis is esophageal rupture (Boerhaave syndrome), which should be excluded by contrast study or endoscopy. The prognosis for patients with pneumomediastinum complicating diabetic ketoacidosis is excellent without special interventions other than management of the ketoacidosis.
References: REFERENCES

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