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Pneumomediastinum in Diabetic Ketoacidosis: Comments on Mechanism, Incidence, and Management*

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We describe three cases of pneumomediastinum associated with diabetic ketoacidosis and give reasons why these disorders may coexist more frequently than heretofore realized. The pathogenesis of pneumomediastinum in such patients remains obscure. Previous authors have considered the pneumomediastinum to be a consequence of vomiting or hyperpnea accompanying diabetic ketoacidosis, but we point out that it sometimes precedes the onset of the metabolic disturbance. We also acknowledge that the appearance of these entities in the same patient could be coincidental. In each of the 11 documented cases so far, primary therapeutic attention to the diabetic ketoacidosis has resulted in an uneventful course and rapid recovery. This experience calls for conservative management of the pneumomediastinum.

More than 30 years ago Hamman¹ described a patient in whom pneumomediastinum and diabetic ketoacidosis coexisted, but only recently has anyone directed attention to such an association.²⁻⁵ We comment here on the pathogenesis, incidence, and treatment of pneumomediastinum accompanying diabetic ketoacidosis and present three illustrative cases.

CASE REPORTS

CASE 1

A previously healthy 16-year-old boy entered the hospital in August 1970 complaining of diffuse abdominal discomfort, occasional vomiting, and pleuritic, precordial chest pain, all of one day's duration. He had experienced polydipsia and polyuria during the preceding two weeks.

On examination he was hyperpneic, lethargic, and dehydrated. His blood pressure was 120/60 mm Hg; pulse, 120 beats per minute; respirations, 40 per minute; and oral temperature, 100.2°F. Subcutaneous crepitus was present in the right supraclavicular area. A popping, extracardiac sound (Hamman's sign) was audible along the left sternal border in the fourth interspace. No other physical abnormalities were evident.

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Initial laboratory studies gave the following results: hematocrit reading, 48 percent; total leukocyte count, 17,500/mm³; urine sugar and acetone concentrations, "4+"; blood sugar level, 406 mg percent; blood urea nitrogen concentration, 15 mg percent; serum CO₂-combining power, less than 5 mEq/liter; and plasma ketone value, "4+", at a dilution of 1:8. The electrocardiogram demonstrated sinus tachycardia. Roentgenograms of the chest disclosed air in the mediastinum and in the right supraclavicular and infraclavicular spaces (Fig 1).

Therapy consisted of insulin and fluids. The ketoacidosis, chest pain, abdominal discomfort, and Hamman's sign disappeared within 24 hours. Four days after admission the subcutaneous crepitus had resolved and chest roentgenogram was normal.

CASE 2

A 13-year-old girl, previously in good health, entered the hospital in December 1970 complaining of retrosternal pleuritic pain of five days' duration and nausea and vomiting of two days' duration. The chest pain had begun gradually, could not be related to a precipitating event, and was not associated with fever, cough, or sputum production. Polydipsia and polyuria had been present for the two weeks preceding admission.

Physical examination revealed a stuporous, dehydrated, and hyperpneic girl whose blood pressure was 110/70 mm Hg; pulse, 130 beats per minute; respirations, 40 per minute; and rectal temperature, 98.2°F. The only other abnormality was a strikingly loud, crunching noise (Hamman's sign) audible over the midsternum and synchronous with the heart beat. Subcutaneous crepitus was absent.

Results of initial laboratory studies were as follows: he-

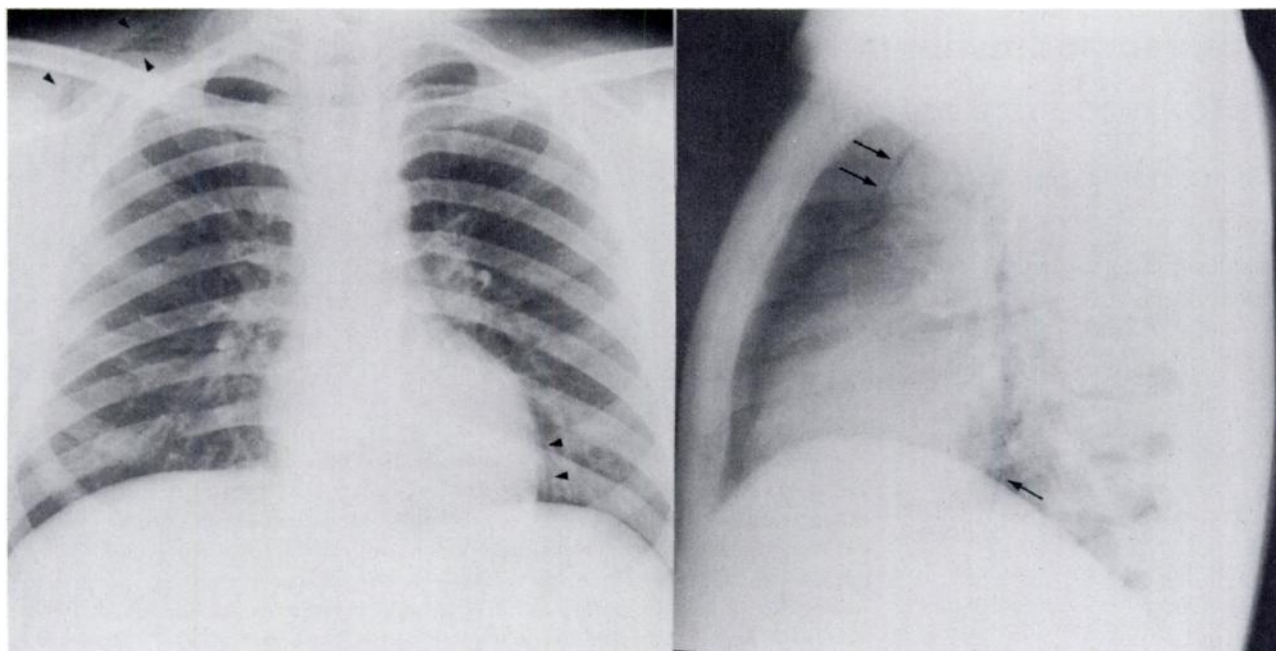


FIGURE 1, Case 1. Chest roentgenograms demonstrating air in the mediastinum and right supraclavicular and infraclavicular spaces.

matocrit reading, 43 percent; total leukocyte count, 39,000/mm³; urine sugar and acetone concentrations, "4+"; blood sugar level, 708 mg percent; blood urea nitrogen concentration, 24 mg percent; serum creatinine value, 4 mg percent; serum CO₂-combining power, 6 mEq/liter; and plasma ketone value, "4+", at a dilution of 1:8. Arterial blood gas analysis during oxygen therapy revealed a pH of 6.91, a PO₂ of 158 mm Hg, and a PCO₂ of 8.2 mm Hg. Electrocardiogram showed sinus tachycardia.

The admission chest roentgenograms (Fig 2) were reviewed independently by six staff radiologists, each aware that pneumomediastinum was suspected clinically. Five diagnosed mediastinal emphysema, and with subsequent films for

comparison, the sixth agreed that pneumomediastinum had been present initially.

Twenty-four hours after institution of fluid and insulin therapy, chest pain, Hamman's sign, and ketoacidosis disappeared, but small, bilaterally symmetrical, pleural effusions became evident on portable chest x-ray film. These roentgenographic changes were not associated with detectable clinical evidence of cardiopulmonary disease or fluid overload and cleared rapidly without specific treatment. On the third hospital day, the patient appeared clinically well and all of her previously mentioned laboratory tests gave normal results. Five days after admission, chest films (Fig 3) showed no abnormality.

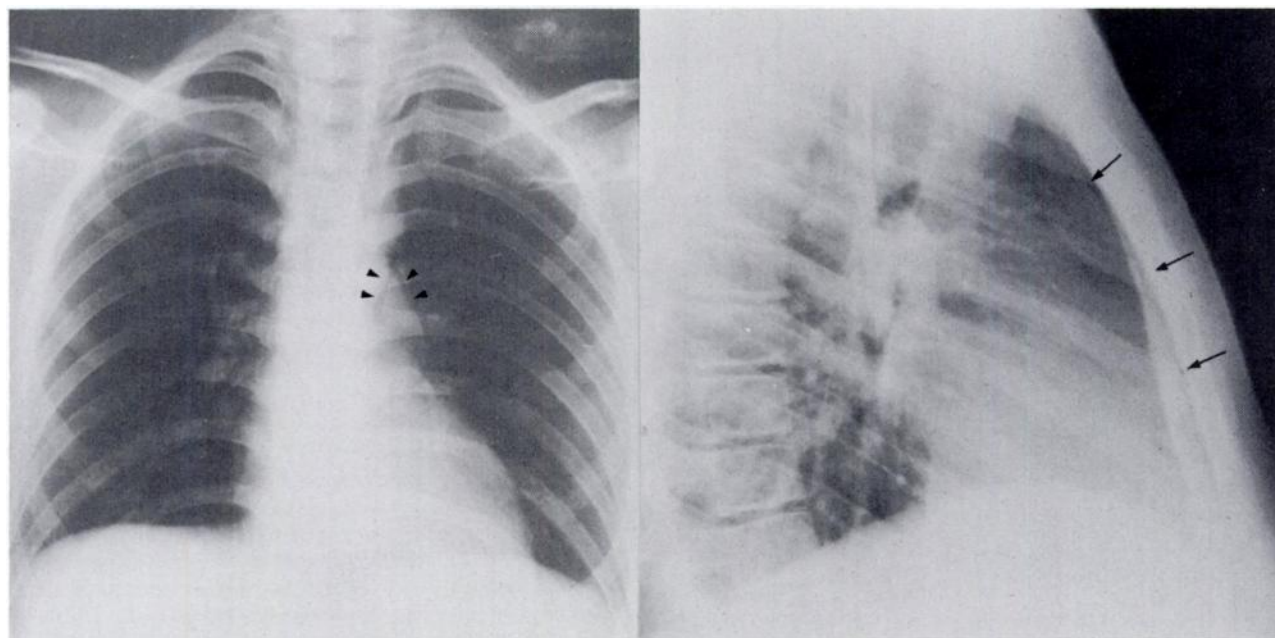


FIGURE 2, Case 2. Chest roentgenograms on admission revealing mediastinal emphysema.

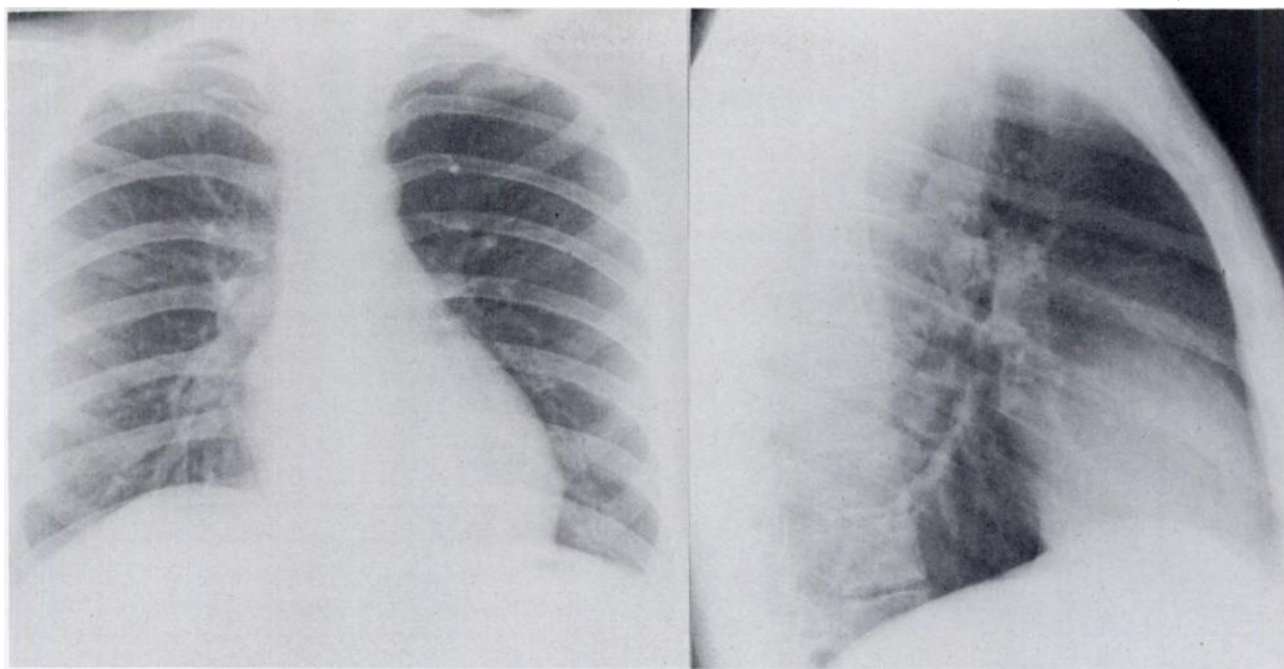


FIGURE 3, Case 2. Chest roentgenograms five days after admission showing no abnormality.

CASE 3

In January 1971 a previously healthy 19-year-old diabetic boy entered Bayshore Hospital, Pasadena, Texas, because of nausea, vomiting, and diffuse abdominal pain of one day's duration and progressively severe retrosternal pleuritic pain of three hours' duration. His diabetes mellitus, present for eight years, has been managed easily with insulin therapy.

On examination he was hyperpneic and dehydrated. His blood pressure was 135/60 mm Hg; pulse, 136 beats per

minute; respirations, 22 per minute; and oral temperature, 97°F. A scratchy to-and-fro sound thought to represent a pericardial friction rub was audible along the left sternal border. No other abnormalities were noted.

Initial laboratory studies gave the following results: hematocrit reading, 54 percent; total leukocyte count, 24,400/mm³; urine sugar and acetone concentrations, "4+"; blood sugar level, 710 mg percent; blood urea nitrogen value, 37 mg percent; and serum CO₂-combining power, 12 mEq/liter. Electrocardiogram demonstrated sinus tachycardia.

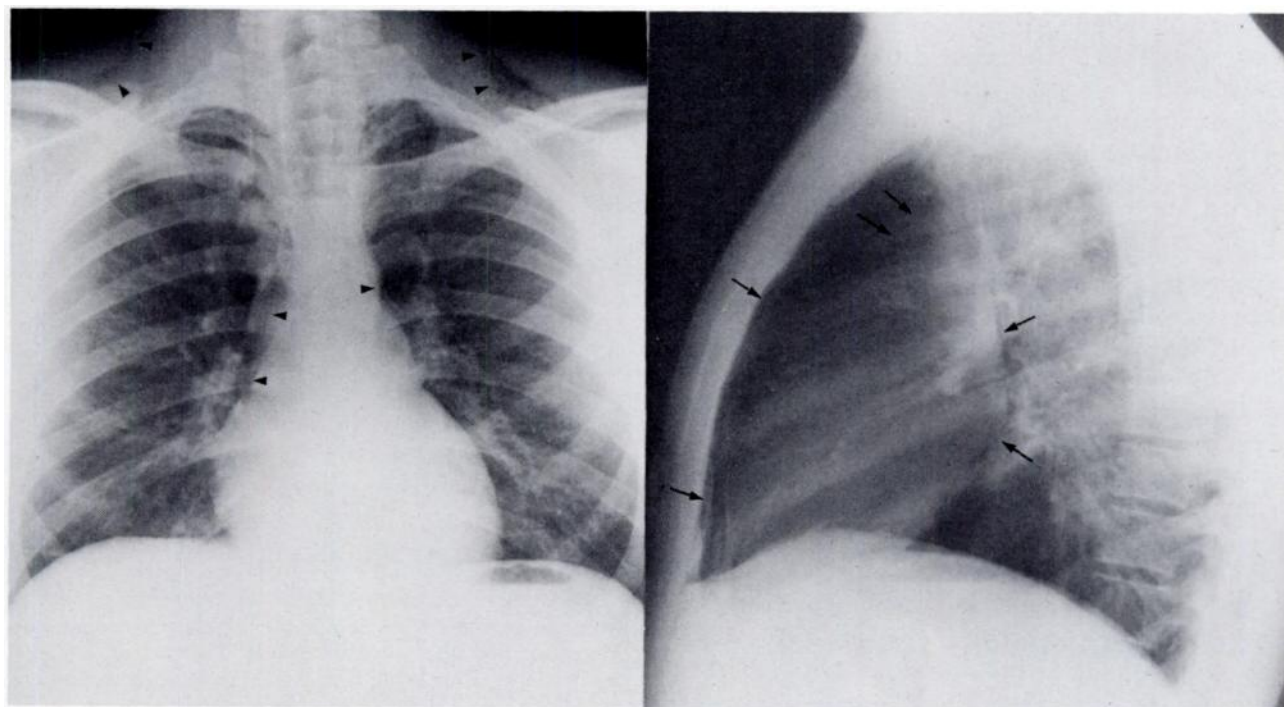


FIGURE 4, Case 3. Chest roentgenograms depicting air in the mediastinum and both supra-clavicular spaces.

Roentgenograms of the chest disclosed air in the mediastinum and in the right and left supraclavicular spaces (Fig 4). It then became apparent that the "pericardial friction rub" was, in fact, Hamman's sign. Moreover, reexamination of the patient revealed subcutaneous crepitus above both clavicles.

Therapy with insulin and fluids corrected the ketoacidosis within 12 hours. By the next morning the patient was asymptomatic and Hamman's sign no longer was detectable. On the fifth hospital day subcutaneous crepitus disappeared. One week after admission chest roentgenogram showed no abnormality.

DISCUSSION

Pneumomediastinum develops experimentally when a sudden unfavorable pressure gradient between alveolar spaces and lung interstices causes rupture of alveoli with consequent dissection of air along perivascular sheaths toward the hilum.⁶ This usually occurs clinically as a result of trauma,⁷ following Valsalva's maneuver during parturition,⁸ or with mechanical obstruction of the airways, as in bronchial asthma.⁹

The pathogenesis of pneumomediastinum in the 11 reported patients with associated diabetic ketoacidosis remains obscure. Previous authors have suggested that severe vomiting¹ or "the expiratory effort and grunting associated with the ketotic hyperventilation"² play a causative role in its production. We question these hypotheses, because in Hamman's patient,¹ symptoms of pneumomediastinum definitely appeared before hyperpnea and were not associated with hyperemesis. Moreover, in our second patient, chest pain also occurred long in advance of vomiting or hyperpnea. Such observations imply that pneumomediastinum sometimes precedes the onset of diabetic ketoacidosis and perhaps initiates or hastens progression of the metabolic abnormality.

Review of data in all of the cases under discussion provides no clue to the mechanism(s) responsible for the pneumomediastinum. Seven of the patients were males and four were females. The youngest was seven years of age² and the oldest, 29 years.⁴ Duration of diabetes mellitus varied from two weeks^{2,*,**} to 12 years.⁴ Findings typical of pneumomediastinum *per se* consisted of chest pain in five patients,^{1,3,*,**,*} Hamman's sign in eight,^{1,2,4,5,*,**,*} and subcutaneous crepitus in eight.^{1,2,4,5,*} Chest roentgenograms uniformly demonstrated mediastinal emphysema and, with one exception,^{**} also showed subcutaneous emphysema. The emphysema resolved within 4* to 25⁴ days. Pulmonary parenchymal changes appeared in two patients^{3,4} and questionably in a third.^{**}

* = Case 1, this report.

** = Case 2, this report.

† = Case 3, this report.

Esophagography gave normal results in the five patients who underwent such examination.^{3,4} Observations consistent with those of diabetic ketoacidosis alone were hyperpnea in all patients, tachycardia and tachypnea in ten^{1,2,4,5,*,**,*} and vomiting in nine.^{2,4,5,*,**,*}

The true incidence of pneumomediastinum in patients with diabetic ketoacidosis may be greater than the number of articles on the subject would indicate. Not only did we encounter our three cases within five months, but Beigelman and colleagues⁴ also observed their four patients during a two-year span. In fact, reports on all but one of the 11 cases have appeared since 1968. The growing number of documented cases may reflect more than broadening interest in the subject and suggests to us that the two disorders are causally related. We acknowledge, however, that because unexplained pneumomediastinum is being recognized with increasing frequency in apparently healthy young adults,^{10,11} its appearance in a patient with diabetic ketoacidosis could be coincidental.

Several factors could account for failure to recognize pneumomediastinum in patients with diabetic ketoacidosis. Unless both clinician and radiologist are alert to the possibility of free mediastinal air, the metabolic disturbance will preoccupy their attention. To complicate matters, symptoms and signs of pneumomediastinum such as chest pain, subcutaneous crepitus, and the mediastinal crunch of Hamman, can be misinterpreted, evanescent, or absent. Finally, if only posteroanterior or anteroposterior chest x-ray films are obtained—an approach usually deemed adequate in a severely ill person—pneumomediastinum will escape notice in about one-half the cases.¹² Addition of lateral chest films improves diagnostic accuracy considerably. Yet, as our second case illustrates, evidence of pneumomediastinum, even on the lateral view, may not be convincing to all experienced radiologists, including those looking specifically for it.

Prognosis in patients with pneumomediastinum and diabetic ketoacidosis has been excellent. In each case primary therapeutic attention to the diabetic ketoacidosis resulted in an uneventful course and rapid recovery. This experience to date calls for conservative management of the pneumomediastinum.

ADDENDUM

In April, 1971, after this paper was accepted for publication, we encountered a fourth case of pneumomediastinum coexisting with diabetic ketoacidosis. The patient, a previously healthy 15-year-old boy, entered the hospital because of polydipsia and polyuria of two weeks' duration, progressively disabling epigastric discomfort of two days' duration, nausea and vomiting of 12 hours' duration, and severe retrosternal

pleuritic pain of four hours' duration. On examination he was stuporous and afebrile and had hyperpnea, tachypnea, tachycardia, and Hamman's sign. Subcutaneous crepitus was absent. Chest roentgenogram demonstrated free air in the mediastinum and in the right supraclavicular and infraclavicular spaces. Blood chemical studies revealed ketoacidosis and hyperglycemia. Thirty-six hours after institution of fluid and insulin therapy, ketoacidosis resolved, the patient appeared clinically well, and Hamman's sign no longer was detectable.

ACKNOWLEDGMENTS: Doctors Harold Selzman and Bernard J. Blumenthal kindly permitted us to include cases 2 and 3, respectively.

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The Birth of Psychoanalysis

The third period of psychiatric historiography was initiated with Freud's *Interpretation of Dreams*, published in 1900, although the volume was actually ready in 1899. These same years coincided with the flourishing of naturalism of Dostoevski, Zola, Taine, of the individualism of Kierkegaard, of Nietzsche. No matter how strong an influence these men and others exercised on young Freud, it cannot be doubted that the *Interpretation of Dreams* opened a new era in the history of psychiatry. It was fortunate that Freud deliberately avoided studying the historical antecedents of his concepts because he was

more free to develop his ideas in an original way. Furthermore, in considering that up to that time psychiatric histories dealt almost exclusively with psychotic institutionalized patients and that Freud himself, was not interested in this type of patient as not amenable to psychoanalysis, it is not surprising that he disregarded the tradition of care and treatment of mental patients.

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