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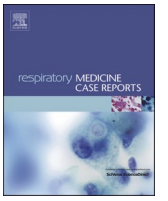

Case Report

A case of severe acute respiratory failure after elective abdominoplasty

Anna V. Rylova *, Rahul Kapil, Jay Parekh, Lauren Mays, Jeffrey Kwon

Yale New Haven Health System, Bridgeport Hospital, Department of Internal Medicine, 267 Grant st, Bridgeport, CT, 06610, USA

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ABSTRACT

Surgical correction of abdominal muscle diastasis may decrease intra-abdominal volume and increase intra-abdominal pressure. The induced changes may ultimately lead to respiratory compromise. In abdominoplasty, one of the most frequently performed esthetic procedures, those changes are believed to be transient and clinically insignificant. We describe a case where acute change in respiratory physiology after abdominoplasty led to severe respiratory failure with significantly decreased pulmonary compliance in a young and otherwise healthy patient. In this case mechanical ventilation failed to improve compliance, and reversal of abdominoplasty was required to restitute pulmonary function.

1. Introduction

Surgical correction of abdominal muscle diastasis with muscle tightening may decrease intra-abdominal volume and increase intra-abdominal pressure. This may impair normal respiratory physiology by reducing respiratory system compliance. The incidence of acute respiratory failure after abdominal wall reconstruction is common, and has been reported to occur in 6–20% of cases [1]. Older age, presence of chronic lung disease, congestive heart failure, and obesity are reported to be independent risk factors [1]. Conversely, abdominoplasty, one of the most frequently performed esthetic procedures, has not been previously linked with a risk for post-surgical respiratory failure. Here we report a case of severe respiratory failure following abdominoplasty in a patient due to alterations in anatomy and respiratory physiology from surgery. Written informed consent for publication was obtained from the patient.

2. Case presentation

A 48-year-old woman (weight 90.5kg, BMI 34.25 kg/m2) with past medical history of well controlled hypertension and hypothyroidism underwent abdominoplasty with repair of rectus diastasis and bilateral breast implantation in October 2021. The procedure was uneventful, and patient was discharged home. Two days later, she presented to the emergency department in severe respiratory distress and hypoxemia, with an oxygen saturation of 80% on room air. Patient reported that she had felt short of breath since her surgery, and her symptoms had become progressively intolerable. She also complained of poorly controlled pain from her surgical wounds. Arterial blood gas showed acute hypercapnia with pH 7.25 and PaCO2 56. Computed Tomography (CT) angiography was performed and was negative for pulmonary embolism. Lung windows were significant for bilateral lower lobe lung atelectasis with prominent reduction of lung volumes (Fig. 1). Chest x-ray also demonstrated a significant reduction in lung volumes when compared to pre-operative films (Fig. 2a and b).

* Corresponding author.
E-mail address: anna.rylova@bpthosp.org (A.V. Rylova).

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Patient was managed with non-invasive positive pressure support and admitted to the Medical Intensive Care Unit. Due to persistent respiratory acidosis and breathing fatigue, patient was eventually intubated and supported on volume-cycled ventilation. Initial ventilator settings were tidal volume (TV) 380 ml, respiratory rate (RR) 20 and positive end expiratory pressure (PEEP) of 5 cm H2O. Arterial blood confirmed correction of acute hypercapnia. Peak inspiratory pressure (PIP) was 40 cm H2O, and plateau pressure (Pplat) was 32 cm H2O. Static compliance was calculated to be 13 ml/cm H2O. Bladder pressure measurements were performed which showed borderline to grade 1 intraabdominal hypertension (11–14 mm Hg).

Over the next 48 hours, the patient was unable to tolerate pressure support breathing consistent with ongoing ventilatory failure and repeat chest x-rays showed persistently low lung volumes. On day 4 of her ICU stay, patient was taken to the operating room for reversal of abdominoplasty. The midline muscle repair was released and removed with an expansion and loosening of the fascia, with an almost immediate discernible improvement in inspiratory pressures. By the following day, static compliance had improved to 24 ml/cm H2O and gradually improved to the mid-30s by the time she was successfully extubated. Postoperative chest x-ray (Fig. 2c) showed restoration of lung volumes.

3. Discussion

According to the American Society of Plastic Surgeons, 129,753 abdominoplasties were performed in 2017. Abdominoplasty is generally considered a low-risk surgery, and the most common complications reported after abdominoplasty are hematoma (1.4–5.8%), wound infection (1.1–9.9%), and venous thromboembolism (0.06–1.0%) [2]. Although abdominoplasty has been reported to increase intraabdominal pressure and reduce pulmonary function, it has been considered a transient, self-resolving and subclinical phenomenon [3–5]. To our knowledge, ours is the first report of severe acute ventilatory failure due to reductions in lung volume and respiratory system compliance after abdominoplasty.

Two components, in our opinion, contributed to our patient's presentation: 1) insufficient postoperative pain control leading to splinting with development of atelectasis and, more importantly, 2) significant decrease in respiratory system compliance from reduced intraabdominal volume that created intraabdominal hypertension and pushed both hemi-diaphragms upward, thus preventing diaphragmatic excursion.

After intubation, our patient's static respiratory system compliance was measured at 13 ml/H20, which is extremely low. Such a "stiff" system would require considerable respiratory muscle strength to overcome. Spontaneous breathing would not be expected to be sustainable under such conditions. For comparison, patients on mechanical ventilation with normal respiratory physiology should have a respiratory system compliance somewhere between 50 and 100 ml/H20 [6].

The low compliance of patient's respiratory system is likely due to the increase in intraabdominal pressure from a drop in intraperitoneal volume and the decrease in abdominal wall compliance after surgery [7]. Preoperative muscle diastasis leads to increased muscle tension and loss of elasticity that ultimately affects abdominal wall compliance after repair [7]. Transmission of intra-abdominal contents into the thoracic cavity likely led to an increase in intrathoracic pressure presenting as decreased lung volumes, atelectasis, increased dead space, and possibly pulmonary hypertension, pulmonary inflammation and edema [8].

Change in thoracoabdominal mechanics ultimately affects diaphragm mobility [5] for the reasons mentioned above, resulting in significant muscle dysfunction. Lack of improvement with non-invasive or controlled mechanical ventilation in our patient pointed towards intraabdominal changes as the primary cause of decreased mobility rather than postsurgical diaphragmatic paralysis.

Only after our patient underwent reversal of her abdominoplasty did her static respiratory compliance and intra-abdominal pressure improve to the point where spontaneous breathing was achievable, and she could successfully liberate from mechanical ventilatory support. The dramatic changes in lung volumes noted on her chest imaging before and after abdominoplasty and after reverse abdominoplasty support these physiologic alterations.

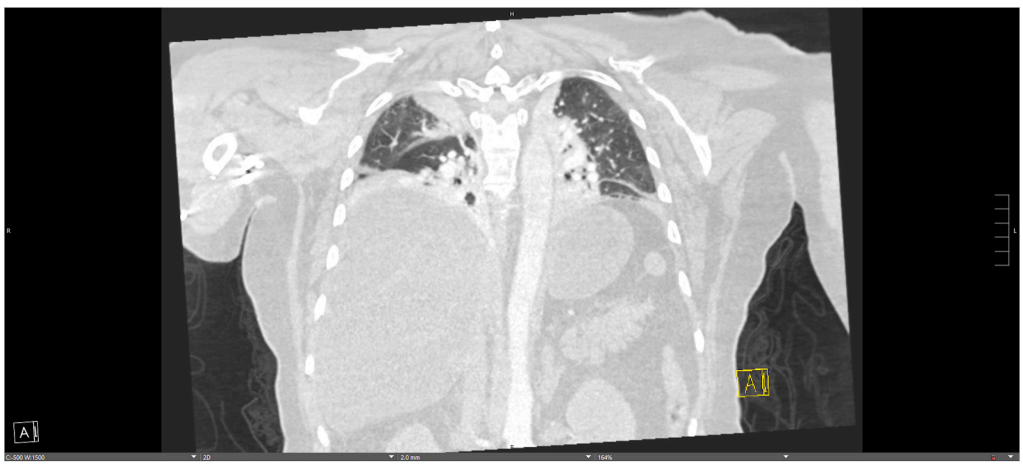

Fig. 1. Computed tomography angiography on admission.

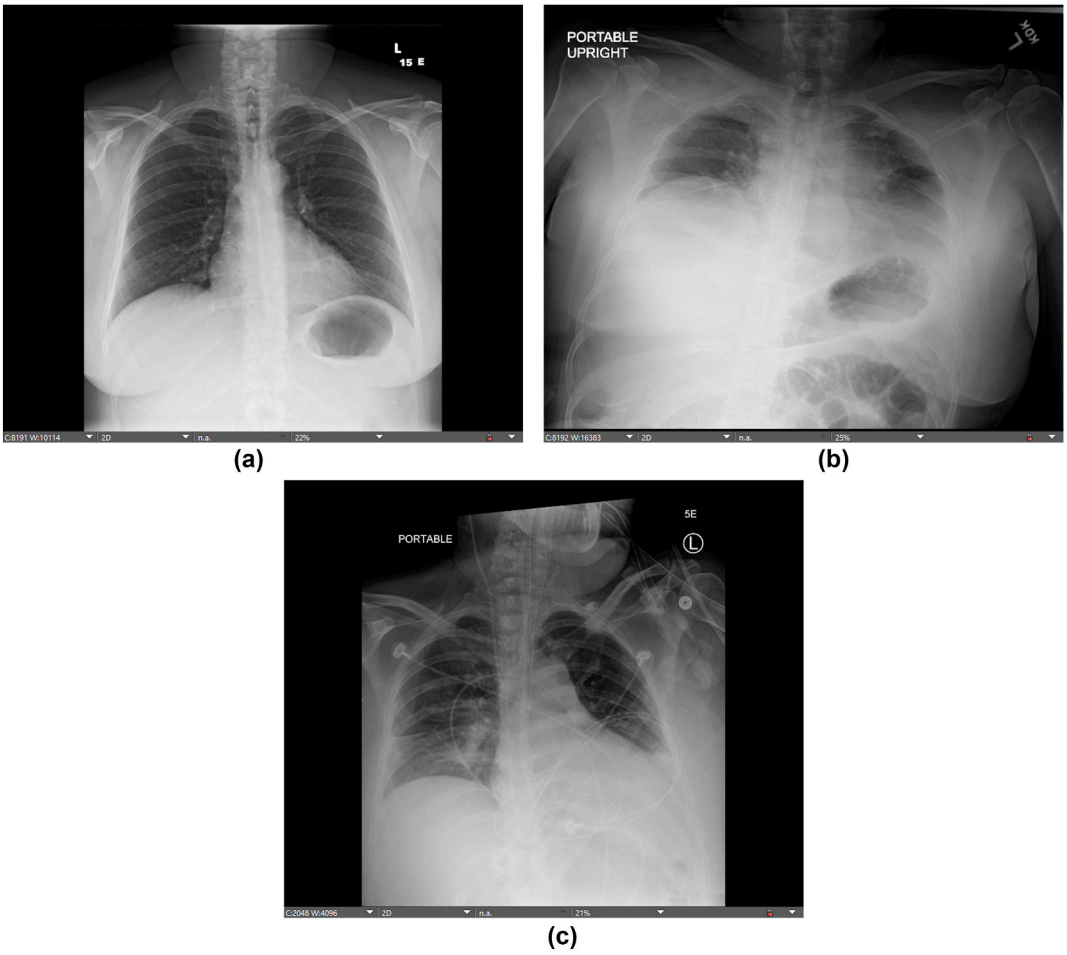

Fig. 2. Chest x-ray
a. preoperatively, b. on admission, c. after revision of abdominoplasty.

4. Conclusion

Abdominoplasty remains popular due to its great cosmetic effect and favorable complication rates. Our case demonstrates that changes to abdominal wall anatomy may induce a dramatic drop in respiratory compliance that can precipitate acute respiratory failure. Although presumably rare, there should be increased awareness of this potential complication, even in a carefully selected, healthy patient population.

Author statement

Anna V Rylova: conceptualization; writing – original draft.
Rahul Kapil: resources; visualization.
Jay Parekh: resources; writing – review and editing.
Lauren Mays: resources.
Jeffrey Kwon: conceptualization; methodology; writing – review and editing.
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Declaration of competing interest

None.

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