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BRIEF REPORT

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# Abdominal compartment syndrome related to noninvasive ventilation

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**Abstract** *Objective:* To study the effects of noninvasive positive pressure ventilation (NIPPV) on intra-abdominal pressure. *Design and setting:* Single case report from a tertiary teaching hospital. *Patients and methods:* A 65-year-old man who experienced a sudden respiratory and cardiovascular collapse during NIPPV. This was caused by gastric overdistension due to aerophagia followed by raised intra-abdominal pressure leading to intra-abdominal hypertension and abdominal compartment syndrome. *Results:* The respiratory and cardiovascular problems resolved immediately after the introduction of a nasogastric tube. This resulted in normalization of IAP. *Conclusions:* This is the first case reported of an abdominal compartment syndrome related to NIPPV. Clinicians should be aware of this possible complication while using NIPPV.

**Keywords** Cardiovascular collapse · Noninvasive ventilation · Intra-abdominal pressure · Intra-abdominal compartment syndrome · Gastric distention

## Case presentation

A 65-year-old obese man was admitted because of dizziness and associated falls. He had a past medical history of cardiac bypass surgery, bowel cancer, non-insulin-dependent diabetes, and chronic bronchitis. Examination revealed fine crackles over both lungs with prolonged expirium. The abdomen was soft and tender. Initial arterial blood gas analysis on room air was normal. Full blood examination showed raised inflammatory parameters. Cardiac investigation showed normal left ventricular ejection fraction (66%). Atrial fibrillation (AF) was apparent on electrocardiography. Neurological investigation was normal.

Two weeks later he was transferred to the ICU with rapid AF (149 bpm) and shortness of breath. Chest radiography revealed right lower lobe pneumoniae, and antibiotics were started. The AF receded under treatment. Four days later he was stable and transferred back to the ward. However, within 5 days he was readmitted with recurrent AF at 150 bpm and respiratory distress: respiratory rate (RR) of 33 breaths/min, with diffuse crackles and severe wheezing. Abdominal examination remained unremarkable. Nebulized bronchodilators were administered, as were intravenous corticosteroids and oxygen. Dyspnea worsened with deterioration of arterial blood gasses: $PaO_2$ 60 mmHg, $PaCO_2$ 65 mmHg, $HCO_3$ 27.2 mmol/l, and pH 7.25 while on 1.5l/min oxygen via nasospecs.

Due to respiratory acidosis and clinical exhaustion (high RR) he was put on NIPPV with an inspiratory positive airway pressure (IPAP) initially of 14 but later 20 $cmH_2O$ and an expiratory positive airway pressure (EPAP) of 5 $cmH_2O$. Since NIPPV was initially intermittent, a nasogastric tube (NGT) was not placed. He

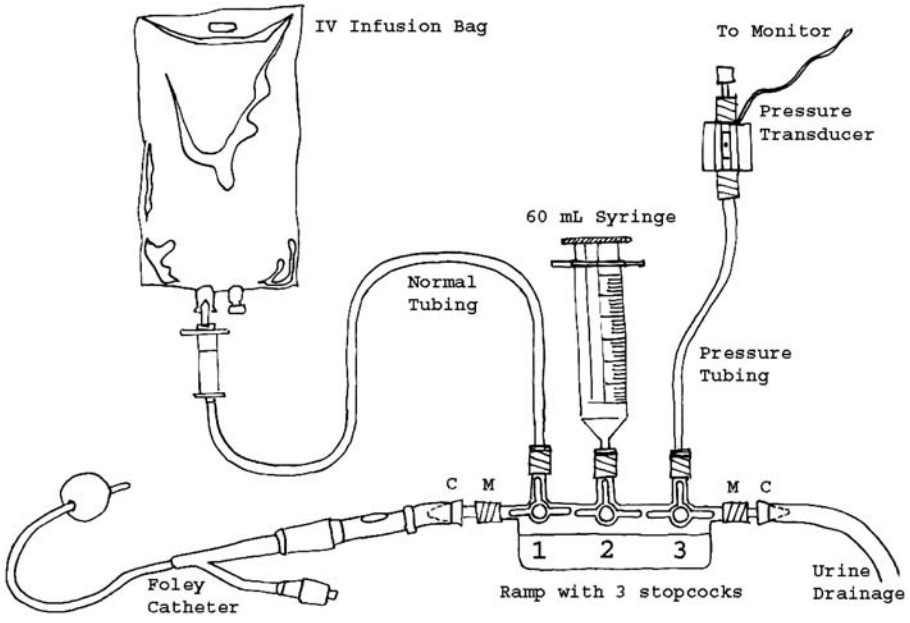


**Fig. 1** A closed needle-free revised method for measurement of intra-abdominal pressure. A Foley catheter is sterile placed and the urinary drainage system connected. Using a sterile field and gloves, the drainage tubing is cut 40 cm after the culture aspiration port. A ramp (Manifold set, Pvb Medizintechnik, Kirchseeon, Germany, or anther manifold set or even three stopcocks connected together will do the job) with three stopcocks is connected to a conical connection piece (*C*, conical connector with female or male lock fitting; Braun, Melsungen, Germany) at each side with a male/male adaptor (*M*, male to male connector piece, Vygon, Ecouen, France) is inserted into the drainage tubing. A standard intravenous infusion set is connected to a bag of 1000 ml normal saline and attached to the first stopcock (*1*). A standard 60-ml syringe is connected to the second stopcock (*2*). The third stopcock (*3*) is connected to a pressure transducer via rigid pressure tubing and a pressurized (at 300 mmHg) bag with 500 ml saline. The system is flushed with normal saline and the pressure transducer is zeroed at the symphysis pubis (or the midaxillary line when the patient is in complete supine position). The pressure transducer is taped at the sypmhysis or the thigh

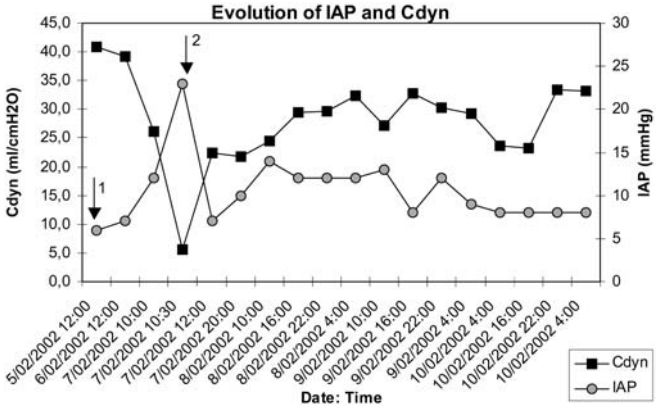


**Fig. 2** Course over time of intra-abdominal pressure (*IAP*, *circles*) and dynamic compliance (*Cdyn*, *squares*). *Arrow 1* Start of NIPPV; *arrow 2* cardiopulmonary resuscitation with intubation and mechanical ventilation. Due to the ACS it was initially impossible to adequately ventilate the patient, and a very low Cdyn was observed

well tolerated the facial mask and remained stable on this treatment for 2 days. Physical examination revealed progressive abdominal distention. IAP measured via the bladder with a modified Kron technique (Fig. 1) showed a rise from 7 to 13 mmHg [1]. Initially tidal volumes remained stable on NIPPV; however, he became exhausted while on NIPPV and oxygen requirements increased, tidal volumes dropped despite an increase in IPAP to 20 $cmH_2O$, and he became NIPPV dependent.

Because of the patient's refusal a NGT was not placed at that time. Gradual deterioration over approx. 24 h resulted in a cardiorespiratory collapse requiring intubation and cardiopulmonary resuscitation (CPR). Just before the collapse he had been put in the upright position by the physiotherapist. Initial rhythm was pulseless electrical activity. He regained pulse and adequate blood pressure after about 5 min CPR; however, he remained poorly saturated despite immediate intubation (esophageal intubation was excluded by esophageal detector device [2]). The difficulty in ventilation was attributed to a mechanical problem as while we were able to effectively hand-ventilate the patient with a 1.5-l adult self-inflating-bag, the ventilator could only deliver approximately 250 ml tidal volume in a volume controlled mode with the maximal airway pressure alarm set at 55 $cmH_2O$. Urgent chest radiography confirmed correct endotracheal tube placement and was comparable to previous radiographs except for an important gastric distention. The IAP at that time was found to be very high at 23 mmHg. This value, together with the underlying organ dysfunction was compatible with the diagnosis of abdominal compartment syndrome (ACS). An NGT was immediately inserted. This resulted in an almost instantaneous resolution of both IAH (normalization of IAP from 23 to 7 mmHg, confirming the ACS) and the respiratory problems, illustrated by a radical improvement in dynamic compliance (Cdyn) from 5.6 to 22.3 ml/cmH2O with a concomitant rise in tidal volume from 280 to 737 ml (Cdyn was calculated as the tidal volume in milliliters divided by plateau pressures minus PEEP). The plateau pressures dropped from 55 to 38 $cmH_2O$ and improvement in oxygenation was noted with a rise in $PO_2$ from 35 to 482 mmHg. One hour later he was stable on $FIO_2$ 0.4. Figure 2 plots the course of IAP and Cdyn over time,

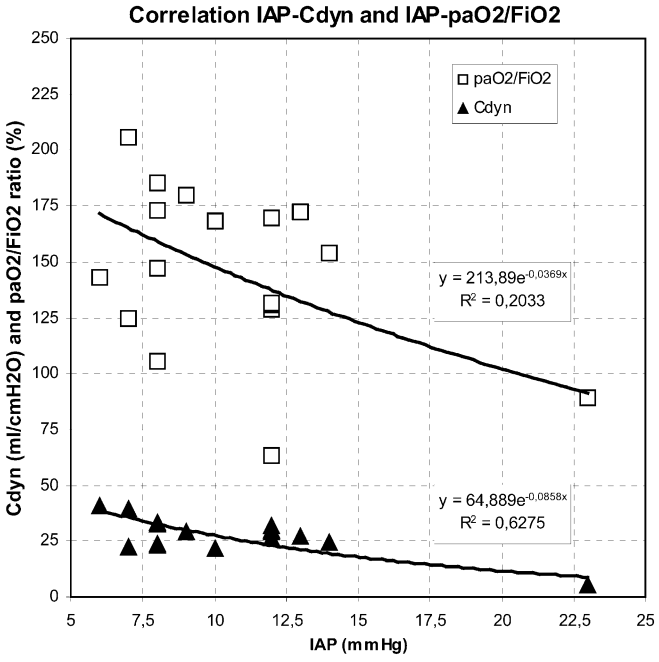


**Fig. 3** Correlation between IAP and Cdyn and between IAP and paO2/FIO2 ratio. *Above* (*squares*) Significant inverse exponential correlation between IAP and paO2/FIO2 ratio: paO2/FIO2= $213.9e^{-0.0369xIAP}$ ($P<0.001$, $R^2=0.2$). *Below* (*triangles*) Significant inverse exponential correlation between IAP and Cdyn: Cdyn= $64.9e^{-0.0858xIAP}$ ($P<0.001$, $R^2=0.63$)

and Fig. 3 shows the significant inverse relationship between IAP and Cdyn and IAP and paO2/FIO2 ratio. Further investigation after stabilization with cardiac transthoracic ultrasound was unremarkable. Bronchoscopy revealed diffuse bronchial edema with friable mucosa. Unfortunately he died 1 month later of complications related to severe septic shock and secondary IAH.

## Discussion

This patient appears to have been at high risk for cardiac arrest given the diagnosis of respiratory failure due to chronic bronchitis, pneumonia, and his underlying cardiac disease. Since other potential differential diagnoses were ruled out, we concluded that the IAH related to aerophagia during NIPPV was the sole cause of this patient's cardiorespiratory collapse. However, it is difficult to explain why NIPPV with an IPAP of 20 $cmH_2O$ caused an increase in IAP of 23 mmHg since NIPPV cannot force air into the stomach against a pressure gradient. Although the high lower esophageal sphincter pressure that usually is well above IPAP could have prevented the air, once swallowed in the stomach from leaving the gastric lumen hence causing further rise in IAP above IPAP. The physiotherapist's having putt the patient in the upright position might have furthermore given rise to IAP causing the collapse [3].

Gastric distention can occur during artificial or mouth-to-mouth ventilation [4]. It is even recommended to logroll the patient and to increase IAP to evacuate the air in the stomach. Abdominal distention caused by aerophagia can lead to increased parasympathetic tone. This can exacerbate bradycardia and asystole [5]. The effects of gastric distention are not univocal and related to baroreceptors. Some patients have a hypersensitivity to gastric distention and its symptoms [6]. Recent publications show that gastric distention leads to bronchoconstriction and bronchial hyperreactivity by inducing airway inflammation [7] and can cause gastric rupture during CPR [8]. Therefore gastric decompression should be routine care.

NIPPV is widely used as treatment for acute respiratory failure and has been shown to decrease the need for intubation [9, 10] the length of ICU-stay [9], mortality [10, 11, 12] and overall cost of care [9, 10, 11, 12]. Complications reported with NIPPV are usually minor but may require withdrawal of the treatment. Commonly seen problems are mask leakage and intolerance, nasal congestion or dryness, nasal bridge redness or ulceration, poor sleep, facial pain and eye irritation, and gastric distention. Adverse hemodynamic effects and barotrauma are uncommon [13]. However, while gastric distention is listed as a minor complication, if unresolved, it may develop to the point that the abdomen compresses the thorax cavity. This condition leads to IAH and possibly to ACS [14, 15]. ACS was initially described as a syndrome resulting in oliguria, hypoxia, hypercarbia, high peak inspiratory pressures, and a tense abdomen [15]. Nowadays ACS is considered to exist whenever IAH progresses to a stage at which systemic organ failure occurs and is defined by the triad of (a) a pathological state caused by an acute increase in IAP above 20–25 mmHg, (b) that adversely affects end-organ function, and (c) in which abdominal decompression has beneficial effects [15]. Our patient hence fulfilled all three criteria of ACS.

Recently Yamada et al. [16] described a case of severe gastric insufflation during NIPPV; unfortunately IAP was not measured (Fig. 4). In our patient gastric distension, and subsequent IAH was responsible for the cardiorespiratory collapse. This is unusual in NIPPV with an IPAP less than 20 $cmH_2O$. Therefore and for technical reasons of mask fitting routine NGT placement is not generally recommended especially in a noncontinuous mode of NIPPV. However, it is widely recognized that raised IAP can increase the intrathoracic pressure through upward deviation of the diaphragm [14, 15]. This elevated intrathoracic pressure can cause an extrinsic compression of the pulmonary parenchyma leading to a restrictive pattern with alveolar atelectasis, decreased oxygen transport across the pulmonary shunt fraction (Qsp/Qt) and reduction in pulmonary capillary blood flow. Resulting in decreased $CO_2$ excretion and increased alveolar dead space characterized by hypoxia and hypercarbia.

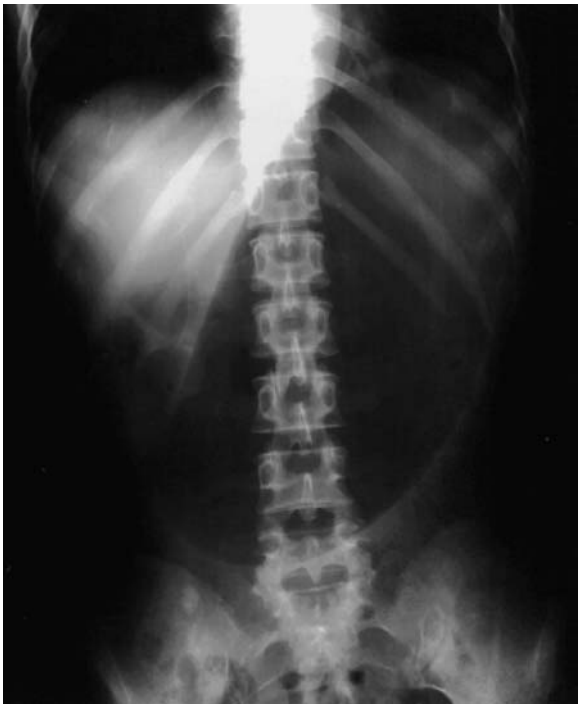

**Fig. 4** Abdominal radiograph in a patient on NIPPV showing extreme gastric dilatation due to aerophagia. (Reprinted with permission from [16])

A similar effect may be noted on the cardiovascular system. An augmented intrathoracic pressure reduces venous return, decreases cardiac output, and increases systemic vascular resistance through compression of both aorta and systemic vasculature [15].

It is recognized that physical examination alone is a very inaccurate method of estimating IAP, and significant IAH may be present despite the absence of gastric distention [17, 18]. Chest and abdominal radiography are also not regarded as reliable tools for identifying IAH. In patients under NIPPV without NGT we recommend a heightened clinical suspicion for gastric distention with serial abdominal examinations. In case of doubt, careful monitoring of IAP is warranted. If IAP is higher than 10 mmHg and the patient is NIPPV dependent, the introduction of a NGT is needed to prevent further complications. This is particularly important in hypovolemic patients who are more susceptible to the effects of IAH. In any patient deteriorating under NIPPV in the absence of a NGT we recommend, in addition to standard procedures, immediate placement of such a tube.

## Conclusion

Although it is difficult to draw conclusions on the basis of one case reported and to be certain that the explanation of our observation is correct, we feel that we can state that the main lesson from this case report is that NIPPV may lead to gastric distention; this may rise IAP leading to IAH and finally ACS. Raised IAP is inversely correlated with Cdyn and pO2/FIO2. The respiratory effects may be exacerbated by bronchoconstriction and bronchial hyperreactivity induced by gastric distention and by putting the patient in the upright position [3]. The effects can be prevented by monitoring IAP and introduction of a NGT when indicated.

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