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Bradycardia Secondary to Negative Suction Pressure Applied to Chest Drain

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Abstract

Positive pressure generated in peritoneal cavity by gas insufflation during laparoscopic procedures can cause hemodynamic instability. There are a few case reports suggesting similar occurrences during thoracoscopic procedures as well. The mechanism behind the conditions above is explained to be due to stretch force applied to peritoneum and pleura which causes vagal stimulation. We wish to present a case where a high negative pressure applied to pleural cavity lead to treatment-resistant bradycardia. The possible mechanism behind this occurrence was traction pressure on pleura which triggered vagal activity. The bradycardia subsided on reducing or discontinuing negative suction pressure. To best of our knowledge this the first case report on bradycardia associated with high negative suction pressure applied to inter costal drain.

Keywords

Chest Drain, High Negative Pressure, Bradycardia, Pleural Traction, Vagal Stimulation

1. Introduction

Very often patients who present with Pneumothorax receive a surgical chest drain and many at times the chest drain doesn't cause complete resolution of pneumothorax. Not infrequently the cardio-thoracic team advises to connect the chest drain to a negative suction pressure. In one of our patient, application of negative suction to chest drain resulted in bradycardia. This bradycardia disappeared each time the negative suction pressure was reduced or discontinued. We wish to share our experience with readers with a case that happened in our Intensive Care Unit which we think occurred secondary to vagal stimulation.

Peritoneal gas insufflation during laproscopic procedures can cause hemody-

namic instability [1] [2]. There are a few reports suggesting similar occurrences during thoracoscopic procedures as well [3] [4]. This instability, which is often associated with bradycardia, is due to stretch force applied to peritoneum and pleura causing vagal stimulation [5].

In this case reported here, a high negative pressure applied to pleural cavity probably caused a traction on pleura which in turn triggered increased vagal tone mediated bradycardia.

2. Case

A 60-year-old female patient presented with Type 1 respiratory failure secondary to chest sepsis. Patient was a recently diagnosed with multiple myeloma and underwent chemotherapy for about 4 months (4 × 28 days cycle of Bortezomib, cyclophosphamide and dexamethasone) before admission to hospital. Initial X-ray chest (**Figure 1**) showed widespread consolidations.

Next day patient was intubated and ventilated due to worsening hypoxia and exhaustion. Arterial blood showed overall improvement in gas exchange. On Day 3, high airway pressures were noticed on ventilator and x-ray chest (**Figure 2**) was done which showed a right-sided pneumothorax. A surgical chest drain placed at that time. Patient remained cardiovascular stable with a reasonable gas exchange on arterial blood gas.

Serial arterial blood gases showed good gas exchange along with other parameters. Over next couple of days, her lung compliance got worse and the chest drain kept on bubbling air which pointed towards continuing air leak. A repeat X-rays (**Figure 3**) and a CT-thorax (**Figure 4**) thereafter showed persisting residual pneumothorax on the same side.

The case was discussed with cardiothoracic surgeons at nearby tertiary referral center and the explanation behind persisting pneumothorax was relatively stiff lungs patient had due to consolidations secondary to chest infection. They also advised to put chest drain under negative suction pressure of −1 KPa (kilo pascals).

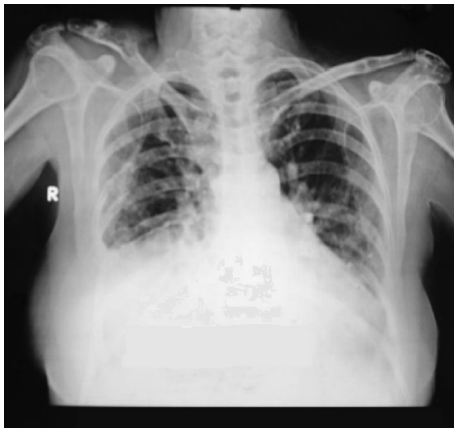

Figure 1. X-ray of chest on admission showing widespread consolidations.

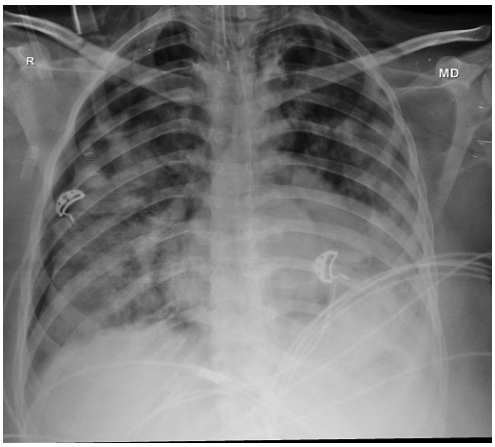

Figure 2. X-ray chest done on Day 3 showing a right-sided pneumothorax.

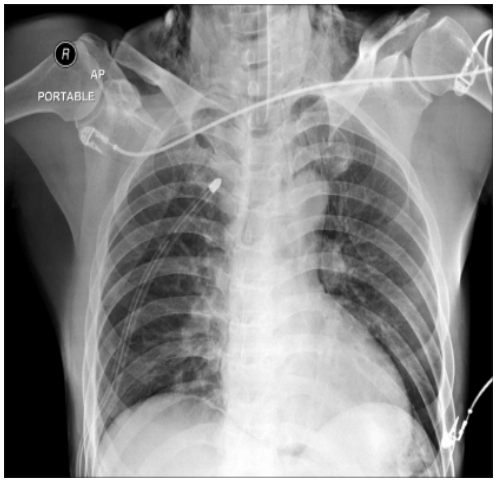

Figure 3. X-ray done after chest drain insertion showed residual pneumothorax on the right side.

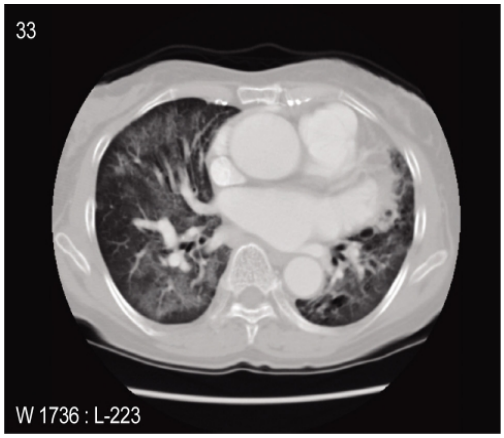

Figure 4. CT thorax done after chest drain insertion confirmed residual pneumothorax on right side and widespread lung consolidations.

An hour after applying negative pressure to chest drain patient started getting bradycardiac. A 12 lead ECG was done which showed sinus bradycardia and no other ST or T wave changes. Patient was reviewed by cardiologist and had emergency ECHO (reported as normal), the cardiologist didn't think of any coronary event (Troponin-i was negative) or any cardiac involvement secondary to multiple myeloma. All the relevant investigations were reviewed including radiological scans, arterial blood gasses, blood reports and they did not point towards any cause for resistant bradycardia.

As bradycardia required frequent boluses of atropine patient was rushed to cardiac.

Intervention lab: during transfer the chest drain was connected to a portable suction apparatus. On way to cardiac lab, the bradycardia resolved. But a temporary pacemaker was inserted as a precautionary measure and on reaching back to intensive care unit and attaching the original suction tubing to chest drain patient again started showing paced rhythms and remained fully pacing dependent for next 24 hours.

In the following days, the chest drain was manipulated on 3 occasions as it would stop swinging or bubbling. A CT scan of chest was considered but cancelled as patient was unstable for transfer to the scanner.

The next night patient continued to have tidal volume loss thru chest drain and on checking the suction pressure settings it was realized that the needle on suction pressure dial was stuck on −1 Kpa although the knob for pressure adjustment was turned to maximum. That means patient was on negative suction pressure much higher than advised. The suction apparatus was changed for a new one and the bradycardia as evident by receding pacing dependency resolved in few minutes.

The temporary pacemaker was removed the very next day and patient was extubated. The air leak resolved with significantly improved lungs and resolved pneumothorax as evident on x-ray chest.

As the case was of equipment malfunction, matter was referred the concerned department. This event was discussed with the unit consultant and was again brought up in morning handover, and again, in department's "mortality and morbidity" meeting.

When assessed to have mental capacity, the patient was informed about the sequence of events happened during the period, and the patient was sedated and ventilated. While patient was under sedation the family was kept updated, events and communications were documented promptly.

As an outcome of this incidence it was agreed to use the other wall-mounted suction apparatus as mentioned above for applying negative pressures to chest drain and to make it sure that suction settings as seen on dial are clearly visible as there is no alarm system and be recorded hourly against the target negative pressure as there is no provision for its automatic entry in "Electronic Patient System". The chest drain insertion is not an infrequent occurrence in intensive care unit and gets audited regularly and would include documenting negative

pressure level as well.

The overall impression is that the high negative suction pressure caused traction on pleura which lead to episodes of bradycardia which is quite similar to bradycardia seen during traction on peritoneum or viscera often encountered during laparoscopy or laparotomies [1] [2].

A standard surgical drain has 5 openings—one apical and other 4 on side walls. Due to high suction pressure probably all these openings got blocked by approximation of visceral and parietal pleura which formed a tight sleeve around them and also caused intermittent stopping of air bubbling through chest drain as happened on Day 4 which was perceived as chest drain being positional and not capturing the pneumothorax.

The formation of tight sleeve of pleura around the tube's opening is more pronounced when the drains tip is towards apex of pleura due to smaller intra-pleural volume as compared to middle and lower zones.

3. Review of Literature

Positive pressure generated in peritoneal cavity by gas insufflation during laproscopic procedures can cause hemodynamic instability [1] [2]. There are a few reports suggesting similar occurrences during thoracoscopic procedures as well [3] [4].

The mechanism behind the above conditions is explained to be due to stretch force applied to peritoneum and pleura causing vagal stimulation [5].

It is well known that stretching of hollow viscera and the body cavity linings trigger vagal response causing bradycardia and hemodynamic instability [6] [7] [8]. Both pleura and peritoneum can trigger vagal activity upon stretching [9]. In this case, the high negative pressure applied to pleural cavity probably caused a traction on pleura causing vagal mediated bradycardia. Other reasons could be due to effect of negative intrapleural pressure on the right and left ventricles [9]. Role of Bezold-Jarish reflex could be another underlying mechanism [10].

Learning point: A new onset bradycardia which has developed after applying excessive negative suction pressure to intercostal drain should raise the suspicion of vagal stimulation secondary to pleural traction when other causes have been ruled out; this case report intends to do the same.

It is advised to keep negative pressure applied to the chest drain at a lower value (−1 KPa was advised in our case), but variations are allowed as per the discretion of treating physicians and cardiothoracic surgeons on individual basis. The suction apparatus needs frequent testing and calibration by biomedical department.

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Conflicts of Interest

"Consent to publish" was obtained and was added to patient's clinical notes. The author declares that there are no competing interests.

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