CASE REPORTS

Does noninvasive ventilation work in ARDS? A case report and review of the current literature

P Malhotra, S K Jindal

The role of noninvasive positive pressure ventilation (NIPPV) in adult respiratory distress syndrome (ARDS) is controversial, in contrast to its well established benefits in other types of respiratory failure, especially acute exacerbations of chronic obstructive pulmonary disease and cardiogenic pulmonary oedema. We report a case of ARDS caused by Mycoplasma pneumoniae in a 70 year old man, treated with NIPPV in addition to standard medical therapy and analyse current evidence regarding the role of NIPPV in patients with ARDS.

A 70 year old retired police inspector presented to the emergency department with a 10 day history of fever followed by rapidly progressive dyspnoea for 3 days prior to admission. He was a non-smoker and teetotaller and there was no prior history of any medical illness. Physical examination revealed marked tachypnoea (respiratory rate 40 breaths/min), central cyanosis, and extensive fine inspiratory crackles on auscultation of the chest. Haemogram revealed haemoglobin 124 g/l and total leukocyte count 30 000/mm³ with a differential of 84% neutrophils, 9% lymphocytes, 5% monocytes, and 2% eosinophils. Erythrocyte sedimentation rate was 35 mm/hour and platelet count 263 000/mm³. Renal and liver function tests were within normal limits. There was severe type 1 respiratory failure on arterial blood gas analysis (table 1). Chest x ray revealed bilateral diffuse alveolar infiltrates with normal cardiac size (fig 1A). The patient therefore fulfilled three of the four criteria for the diagnosis of ARDS. Extensive ground glassing was observed on high resolution computed tomography (CT) scan of the chest (fig 1B). Serum cold agglutinins were positive, with a titre of 1:64. Blood culture and review of the current literature revealed bilateral diffuse alveolar infiltrates with normal cardiac size consistent with ARDS; right panel, a series of high resolution chest CT scans demonstrating bilateral extensive ground glass opacities.

DISCUSSION

It is now recognised that Mycoplasma pneumoniae, traditionally believed to cause mild disease, is the aetiological agent in about 2–7% cases of severe community acquired pneumonia (CAP). However, ARDS due to M. pneumoniae is distinctly unusual, with only sporadic case reports in the English literature.

NIPPV has now assumed a central role in the management of hypercapnic respiratory failure due to chronic obstructive pulmonary disease (COPD) and in cardiogenic pulmonary oedema; however, its role in ARDS secondary to severe pneumonia is highly controversial. Early studies comprising a heterogenous group of patients with acute respiratory failure treated with NIPPV suggested that pneumonia was a poor prognostic factor. As an illustration, in Antonelli’s study, all patients with pneumonia randomised to NIPPV failed therapy, whereas there was a 60% success rate in those patients in whom respiratory failure was not attributable to pneumonia. The authors speculated that the higher failure rate in patients with pneumonia was due to difficulty clearing secretions, reduced pulmonary compliance, and nonhomogeneous gas exchange. Analysis of the first randomised trial of NIPPV in 56 patients with pneumonia revealed that only the subgroup of patients who had concomitant COPD appeared to benefit from NIPPV.

In a study by Rocker et al, there was a 66% success rate when NIPPV was used as the initial mode of assisted ventilation in 10 patients with ALI/ARDS. More recent data

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The authors found that patients whose respiratory rates decreased at the end of the first hour of NIPPV were less likely to require intubation than those in whom the respiratory rate either remained stable or increased. Our patient had shown a significant decrease in respiratory rate as well as dyspnoea at the end of the first hour of NIPPV. However, we feel that this dictum of clinical improvement at 1 hour predicting a successful outcome of NIPPV is applicable more to cases of COPD than severe pneumonia, as the latter is characterised by alveolar filling, ventilation perfusion mismatch, and decreased pulmonary compliance, all of which are less rapidly reversible than the hypercapnic respiratory failure of COPD, in which respiratory muscle fatigue plays a predominant role. The most recent and largest study (105 patients) to address the issue of NIV in severe pneumonia found that the use of non-invasive ventilation compared with high concentration oxygen therapy decreased the need for intubation, the incidence of septic shock, and intensive care unit mortality, even though the seven patients with ARDS had a poor outcome (five died). Current evidence therefore suggests, as was demonstrated in our case, that NIPPV is warranted in most patients of ARDS due to severe community acquired pneumonia not responding to standard medical therapy. However, as intubation rates are high, NIPPV should ideally be used in such patients with a conventional mechanical ventilator on standby. It is the subgroup of patients with relatively mild, early ARDS who are likely to benefit from NIPPV and avoid intubation.

### Table 1

<table>
<thead>
<tr>
<th>At admission (FiO₂ 0.5)</th>
<th>Day 3 of antibiotics (FiO₂ 0.5)</th>
<th>On NIPPV (IPAP 8, EPAP 4)</th>
<th>At discharge (room air)</th>
</tr>
</thead>
<tbody>
<tr>
<td>PaO₂</td>
<td>86</td>
<td>86</td>
<td>67</td>
</tr>
<tr>
<td>PaCO₂</td>
<td>23</td>
<td>21</td>
<td>31</td>
</tr>
<tr>
<td>SaO₂</td>
<td>86</td>
<td>88</td>
<td>97</td>
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<tr>
<td>%</td>
<td>20</td>
<td>19</td>
<td>22</td>
</tr>
<tr>
<td>HCO₃</td>
<td>20</td>
<td>19</td>
<td>22</td>
</tr>
<tr>
<td>pH</td>
<td>7.48</td>
<td>7.49</td>
<td>7.44</td>
</tr>
</tbody>
</table>

### LEARNING POINTS

- *Mycoplasma pneumoniae* can cause ARDS in adults
- NIPPV is warranted as a ‘bridge’ in patients with ARDS due to severe pneumonia until antibiotics take effect.
- NIPPV is especially likely to benefit patients with a rapidly reversible cause of ARDS.

### Authors’ affiliations

P Malhotra, S K Jindal, Department of Pulmonary Medicine, Postgraduate Institute of Medical Education and Research, Chandigarh, India

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Correspondence to: Dr S K Jindal, Department of Pulmonary Medicine, Postgraduate Institute of Medical Education and Research, Chandigarh 160012, India; skjindal@indiachest.org

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Ring down artefacts on abdominal sonography to predict pulmonary abnormalities in the emergency department
C-L Tsai, H-P Wang, W-C Lien, C-C Chen, T-I Lai, W-J Chen

Ring down artefacts are sometimes found when emergency physicians perform abdominal ultrasound to differentiate between various abdominal problems. We describe a patient who presented with right upper quadrant abdominal pain and whose ultrasound examination showed ring down artefacts posterior to the right hemidiaphragm, which led to the eventual diagnosis of pneumonia. Ring down artefacts on ultrasound may be used to predict pulmonary abnormalities. Awareness of this sonographic finding may assist in accurate diagnosis and administration of appropriate treatment without delay.

Emergency physicians (EPs) frequently encounter abdominal complaints. Abdominal ultrasound (US) is of great assistance in differential diagnosis of these problems. However, during the abdominal US examination, meaningful artefacts are sometimes seen. In particular, posterior to the liver, the examiner may encounter multiple, vertical, long and narrow bands or lines trailing down from the posterior surface of the right hemidiaphragm, the so-called “ring down” artefacts. We describe a patient whose US examination showed these ring down artefacts, which contributed to his final diagnosis of pneumonia. We discuss the implications of these findings for EPs.

CASE REPORT
A 58 year old man presented to the emergency department (ED) with sudden onset of right upper quadrant abdominal pain. No fever, diarrhoea, or nausea was noted. He was a heavy smoker who had suffered from chronic productive cough for years. He denied other significant past medical or surgical history. On arrival, blood pressure was 113/59 mmHg and body temperature was 38°C. On physical examination, the breath sounds were relatively clear. The abdomen was soft, and there was a positive Murphy’s sign. Laboratory examinations showed leucocytosis with white blood cell count of 13,870/ml and 80% neutrophils. Aspartate amino-transferase and bilirubin values were within normal limits. Emergency US was performed by an EP, working on suspicion of acute cholecystitis. However, the US did not show gallstones or wall thickening of gall bladder, which would have supported the diagnosis of acute cholecystitis. Instead, numerous ring down artefacts posterior to the right hemidiaphragm were disclosed with the US probe placed transversely, right subcostally, and in the cephalic direction (fig 1), which led to suspicion of abnormalities in the right lung base.7 They found that various pulmonary diseases can show ring down artefacts on US scan. In their study, when the pulmonary abnormalities are localised, ring down artefacts are seen focally at the area of abnormalities. In contrast, nearly all cases of idiopathic interstitial pneumonia elicit numerous ring down artefacts.3 In our case, although the patient’s Murphy’s sign was positive, it did not carry sufficient weight to establish the diagnosis of cholecystitis.6 Furthermore, clinical presentation with right upper quadrant abdominal pain has been reported to be associated with pulmonary pathologies, such as pulmonary embolism or tension pneumothorax.7,8 Therefore, abdominal US serves a crucial role to confirm diagnosis in patients presenting with right upper quadrant abdominal pain.

Ring down artefacts were found in our patient and redirected the physicians to the possibility of pneumonia. The case underlines the importance of ring down artefacts—that is, to remind clinicians that the possible diagnosis may be located above the diaphragm.

Figure 1 Abdominal sonogram shows numerous ring down artefacts (arrow) posterior to the aspect of the right hemidiaphragm.

DISCUSSION
Traditionally, ring down artefacts have been thought to be similar to the comet tail artefacts that are associated with foreign bodies, particularly metallic objects and cholesterol crystals.3,4 These two artefacts are both reverberation artefacts, producing a series of parallel bands radiating from their sources. They appear when a large mismatch in acoustic impedance occurs between two types of tissue. This interface has two effects: firstly, as it reflects 99% of the sound beam and produces strong reverberation artefacts parallel to the transducer, the interface totally obscures the underlying tissue, and secondly, it generates showers of vertical echo that will be projected into the underlying tissue.5 Avruch and Cooperberg postulated that ring down artefacts appear because of multiple reflections of the US pulse occurring between air bubbles of the lung parenchyma.4 Lim et al further speculated that the distribution and extent of ring down artefacts posterior to the right hemidiaphragm may depend on the distribution and the severity of abnormalities in the right lung base. They found that various pulmonary diseases can show ring down artefacts on US scan. In their study, when the pulmonary abnormalities are localised, ring down artefacts are seen focally at the area of abnormalities. In contrast, nearly all cases of idiopathic interstitial pneumonia elicit numerous ring down artefacts.3 In our case, although the patient’s Murphy’s sign was positive, it did not carry sufficient weight to establish the diagnosis of cholecystitis.6 Furthermore, clinical presentation with right upper quadrant abdominal pain has been reported to be associated with pulmonary pathologies, such as pulmonary embolism or tension pneumothorax.7,8 Therefore, abdominal US serves a crucial role to confirm diagnosis in patients presenting with right upper quadrant abdominal pain.

Abbreviations: ED, emergency department; EP, emergency physician; US, ultrasound
Brugada syndrome, manifested by propafenone induced ST segment elevation
E Aksay, T Okan, S Yanturali

We report a case of a 43 year old man who was diagnosed with Brugada syndrome after propafenone administration for chemical cardioversion of new onset atrial fibrillation. Brugada syndrome has been described in the medical literature and is thought to be responsible for the majority of sudden cardiac deaths in patients without ischaemic heart disease. This syndrome has not yet been extensively discussed in the emergency medicine literature despite its importance. Emergency physicians should consider Brugada syndrome in patients who present to the emergency department with right bundle branch block and ST segment elevation in the right precordial leads, which is the classic electrocardiographic pattern of this syndrome.

Ventricular fibrillation (VF) is the main cause of sudden cardiac death (SCD). Most SCDs are associated with acute coronary ischaemia near the time of death. However, 10–20% of SCD patients have no evidence of structural or ischaemic heart disease, and these patients have been referred to as having “idiopathic VF”.1,2 Brugada syndrome (BS) is now suspected to be responsible for 40–60% of cases of idiopathic VF.3

References


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Correspondence to: Dr C-L Tsai, Department of Emergency Medicine, National Taiwan University Hospital, No. 7, Chung-Shan South Road, Taipei, 100, Taiwan; chulintsai@ntu.edu.tw

REFERENCES

Because atrial fibrillation was considered as new onset, 600 mg of propafenone was administered orally to achieve chemical cardioversion. After 6 hours, a repeat ECG showed 1–3 mm ST segment elevation in leads V1–3, and T wave inversion in leads V1 and V2 (fig 1B). The patient denied any chest pain or angina equivalent during the observation period. Cardiology consultation was obtained because of suspected silent acute myocardial infarction, and repeat cardiac markers were ordered. Subsequently, ST segment elevation was persistent for 6 hours and then progressively declined to baseline. Additionally, atrial fibrillation converted to sinus rhythm, and the RBBB resolved (fig 1C). At 12 hours, cardiac markers were not elevated. The patient was admitted to the coronary care unit with a diagnosis of suspected BS.

DISCUSSION

BS was first described by Pedro and Josep Brugada in 1992. The syndrome is characterised by SCD or episodes of syncope resulting from polymorphic VT or VF in previously healthy people, with the classic ECG finding of RBBB and ST segment elevation in the right precordial leads. Patients with BS may be completely asymptomatic, and are often recognised by chance because of ECG screening for insurance, sport licensing, atypical complaint, or investigation of other family members with known BS. Therefore, BS should be considered and investigated in patients with the characteristic ECG pattern, even if they are asymptomatic.

BS is a primary electrical disorder resulting in abnormal electrophysiological activity, and is typically seen in the fourth or fifth decade of life in men. Although the syndrome has autosomal dominant transmission, sporadic cases have been reported. Mutation of the SCN5A gene, which encodes for cardiac sodium channels, causes loss of cardiac sodium channel function, resulting in a shortening of the action potential duration in the right ventricular

Table 1 Diagnostic criteria for Brugada syndrome

1. Appearance of a coved type ST segment elevation (gradually descending terminal portion) in more than one right precordial lead, in the presence or absence of a sodium channel blocker and one of the following clinical criteria:
   - Documented ventricular fibrillation
   - Self terminating polymorphic VT
   - Family history of SCD (<45 years)
   - Coved type ECGs in family members
   - Electrophysiological inducibility
   - Syncope or nocturnal agonal respiration

   The appearance of the ECG features, without these clinical symptoms, is referred to as an idiopathic Brugada ECG pattern (not BS).

2. Appearance of saddle back type ST segment elevation (terminal portion >1 mm) in more than one right precordial lead under baseline conditions with conversion to coved type following challenge with a sodium channel blocker. Drug induced ST segment elevation >2 mm should raise the possibility of BS when one or more of the aforementioned clinical criteria are present.

3. Appearance of saddle back type ST segment elevation (terminal portion <1 mm) in more than one lead under baseline conditions with conversion to coved type following challenge with a sodium channel blocker.

Table 2 Treatment criteria for Brugada syndrome

All symptomatic patients and inducible asymptomatic patients should be treated.

Non-inducible asymptomatic patients should be observed with follow up, and treatment is not recommended for these patients.

Figure 1 (A) Initial ECG: atrial fibrillation with rapid ventricular response and incomplete RBBB, with no evidence of Brugada syndrome. (B) Repeat ECG after propafenone administration: 1–3 mm ST segment elevation in leads V1–3, and T wave inversion in V1 and V2. (C) ST segment elevation and RBBB resolved and atrial fibrillation converted to sinus rhythm.
epicardium, which causes a transmural voltage gradient, seen as ST elevation and re-excitation on the ECG. This voltage gradient creates a vulnerable window for extrasystoles or premature impulses to initiate phase 2 re-entry, triggering VF. Class IA (for example, ajmaline, procaniamide) and class IC (for example, propafenone, flecainide) antiarrhythmic agents and heightened parasympathetic tone increase ST segment elevation and may precipitate VF. Sympathetic activation, stress testing, isoproterenol, and dobutamine may decrease ST segment elevation and result in transient normalisation of the ECG. The VF frequently seen during sleep in patients with BS is probably due to a decrease in sympathetic tone.

The typical presentation of BS is syncope or SCD, depending on the duration of VT. If VT is persistent, it eventually degenerates into VF and results in SCD. If VT is self terminating in a short period, it results in syncope or near-syncope. In addition, it has been suggested that there is a higher than normal incidence of supraventricular tachyarrhythmias in Brugada patients. Nearly 10% of patients with BS have concomitant atrial fibrillation. Therefore, BS should not be only considered in SCD victims or syncope patients, but also in patients who present to the ED with new onset atrial fibrillation with ST segment elevation in the right precordial leads.

Half of patients with BS have non-diagnostic testing ECG, and in 42% of cases, the ECG can normalise transiently. Because of the intermittent and concealed nature of the ECG signs, diagnosis of BS may be difficult. A consensus report of the diagnostic criteria for BS is outlined in table 1.

Patients with suspected BS should undergo a pharmacological challenge test with class IA antiarrhythmic drugs in the electrophysiology laboratory. It has been reported that propafenone has an unmasking effect and can reveal a concealed BS, as occurred in our patient. False positive pharmacological challenge tests have not been reported so far.

Electrophysiological testing is useful for both further confirmation of the diagnosis and determination of treatment strategies. In the largest series yet reported in the literature, 30% of patients with suspected BS patients developed a recurrent arrhythmic event (VF, SCD, or syncope due to ventricular arrhythmias) at a mean (SD) of 31 (41) months' follow up. In the same study, while 12% of asymptomatic patients who were induced by programmed ventricular stimulation developed their first arrhythmic event, only 1.1% of non-inducible asymptomatic patients developed arrhythmias. Treatment criteria for BS are shown in table 2. Because of the autosomal dominant inheritance, other family members should be referred to a cardiologist for diagnostic testing.

The mortality of BS is approximately 30% at 2 years following the diagnosis. An implantable cardioverter defibrillator (ICD) implant is the only effective treatment option for prevention of SCD in patients with BS. In one study, with use of an ICD, the mortality at 10 year follow up was 0%. Antiarrhythmic drugs, including amiodarone and beta blockers have not been shown to reduce mortality or recurrence of ventricular arrhythmias.

CONCLUSION

BS is a preventable cause of SCD. Failure to diagnose the syndrome results in a high mortality rate. Early recognition of this syndrome may contribute to a decrease in the frequency of idiopathic VF and may improve prognosis of patients with BS. Therefore, emergency physicians should be familiar with the ECG findings of BS, and patients with suspected BS should be referred to a cardiologist. For all patients with new onset atrial fibrillation with RBBB, if chemical cardioversion is planned with propafenone in the ED, they should be also observed for ST segment elevation, which would be indicative of BS.

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Trans-sternal cardiac injury caused by a hooked needle

H Yanar, M Aksoy, K Taviloglu, E S Unal, M Kurtoglu, K Nisli

Cardiac injuries remain the most challenging of all injuries seen in the field of trauma surgery. Penetrating injury to the heart generally occurs less frequently than blunt injury and most commonly injures the large anterior right ventricle. We present an unusual and to our knowledge a previously unreported, cause of cardiac penetrating trauma in a child, involving a hooked needle (a 15 cm long, metallic device usually used for crocheting or lacemaking). A ventricular septal defect was managed conservatively shortly after the primary cardiorrhaphy. Evaluation methods for this rare presentation and its possible surgical treatments are discussed.

CASE REPORT

A 5 year old girl was admitted to our emergency department with a hooked needle penetrated in her chest wall from the left edge of the sternal corpus, in a mediolateral and craniocaudal trajectory (fig 1). The accident had happened when she fell over while running at home with a hooked needle in her hand, just 40 minutes before admission. Her blood pressure was 105/60 mm Hg, and the heart rate was 120 beats/min. The exposed part of the hooked needle in her hand, just 40 minutes before admission. Gentle removal of the foreign body in the operating theatre was planned under mild anaesthesia, with conditions for general anaesthesia set in place in case of emergency. Four minutes after removing the hooked needle, a sudden bradycardia developed and central venous pressure rose to 15 cm H2O. The patient was intubated endotracheally and a left anterolateral thoracotomy via Sparango incision was performed. During the thoracotomy, pericardial tamponade was eased by pericardiotomy. There was a perforation measuring 3×4 mm on the anterior wall of the right ventricle, which was repaired with horizontal Halsted mattress sutures, (Prolene no. 2-0), without need for a pericardial patch. The patient was taken to the intensive care unit (ICU) postoperatively.

After two uneventful postoperative days in the ICU, the patient developed tachypnoea and dyspnoea with auscultable rough crackles from either hemithorax. Consultation with the paediatric cardiology unit (PCU) revealed congestive heart failure, thus intravenous dopamine and dobutamine treatment was started. Echocardiography was performed the same day and showed a traumatic VSD of 3.5 mm in length in the muscular portion of the interventricular septum with rough margins and an evident left to right shunt with a pressure gradient of 77 mmHg. The right ventricle was mildly dilated and its anterior wall hypokinetic. The ejection fraction was found to be 67% (fig 3).

The treatment in the ICU relieved the symptoms and the child was discharged to the PCU with no surgical problem on the sixth postoperative day, continuing on digoxin, captopril, and prophylaxis for endocarditis.

After 30 days of follow up in the PCU, the anti-congestive therapy was stopped. The patient was discharged with an asymptomatic patent VSD, and close six monthly follow up with echocardiography for the first year was planned.

DISCUSSION

Penetrating cardiac trauma in children is seldom reported in the literature and the incidence of penetrating cardiac trauma in children is lacking. Data on the effects of acute traumatic injury to specific cardiac components come mostly from the adult literature and consists of atioventricular valve insufficiency, aortic insufficiency, VSD, atrial septal defect, coronary artery injury, haemopericardium, cardiac rupture and cardiac contusion.

Physiological condition, cardiovascular respiratory score and mechanism of injury plus initial rhythm are reported to...
be significant predictors of outcome in penetrating cardiac injuries in adults. The case presented here, and our experience in cardiovascular trauma surgery supports that these predictive factors are strongly reliable. We also agree that the critical time period of pericardial tamponade acts on the patients’ outcomes as a positive or negative predictive factor.

There are several methods of evaluating cardiac injuries. Subxiphoid pericardial window remains the gold standard of all procedures for the diagnosis and treatment but with the availability of ultrasound (US) in trauma centres, this technique has been relegated to a second line of evaluation. Surgeon performed US decreases the time between arrival and definitive treatment in patients with penetrating cardiac traumas.

Three categories of electrocardiographic (ECG) interpretation exist in penetrating cardiac trauma pre and post operatively: acute myocardial infarction, pericarditis, repolarisation unspecific changes. But ECG abnormalities are said to occur less commonly in children than in adults, as we experienced.

Two dimensional echocardiography has been shown to have a 90% accuracy, a 97% specificity, and a 90% sensitivity in detecting penetrating cardiac injuries by Jimenez et al. Unfortunately we could not perform an echocardiography preoperatively because of the course of our cases, but echocardiography gave us the chance of detecting the VSD postoperatively, and we believe that it must be the first choice as a diagnostic tool in the follow up period of patients.

Chest CT has been shown to have a high sensitivity, specificity and accuracy rate in the setting of penetrating thoracic injuries. CT findings of our patient were the same as we detected in the operation. We think that CT scans can be used for diagnosis of a haemodynamically stable patient who can be closely monitored during the transfer and scanning period.

Unlike our case, emergency department thoracotomy can be used as a diagnostic and therapeutic tool in unstable patients and it continues to be widely used in children.

Haemodynamically stable selected patients with isolated penetrating cardiac injuries can be followed up conservatively in ICU as in penetrating abdominal injuries. Series aiming to emphasise the point that there is a place for conservative management of selected cases with penetrating cardiac injuries has been reported, and we will report the results of our conservative management in penetrating cardiac trauma in the near future. Therefore, we tried to give the chance of non-operative management to our patient, but due to the reasons mentioned above, thoracotomy was performed.

Another point of debate is the reparation of injuries with life threatening free wall wounds of the heart. It is usually quite hard to detect accompanying intracardiac lesions in emergency surgery situations because of the negative effects of increased operation time to the traumatic patient. Intraoperative cardiac sampling following penetrating wound as a technique for early detection of traumatic intracardiac shunts in a case has been reported. In the literature numerous studies have reported residual intracardiac lesions which are not identified in the first operation from 4% to 56% of cases. Functional sequelae in such patients have rarely been described.

Tesinski reports that almost 25% of the patients are later diagnosed to have suffered injury also to one of the intracardiac structures, a VSD caused by penetrating injury to the heart is found to in 2–10% of the survivors and interval repair should be performed according to the clinical status of the patient and mostly depends on the size of the left to right shunt. Delayed repair of cardiac trauma allows tissue healing to a varying degree which may or may not be beneficial. VSDs can shrink or even close spontaneously with time, but this is unlikely to occur with tricuspid valve injuries.
We agree the authors who are in favor of delayed repair as it is more rationalist to repair residual intracardiac lesions in an elective operation. However the patients should be followed closely for delayed sequel of these lesions in a multidisciplinary fashion.

**Authors’ affiliations**
H Yanar, M Aksoy, K Taviloğlu, E S Unal, M Kurtoğlu, Department of General Surgery, Emergency Surgery Unit, Istanbul Medical Faculty, Istanbul University, Istanbul, Turkey
K Nişli, Pediatric Cardiology Unit, Department of Pediatrics, Istanbul Medical Faculty, Istanbul University, Istanbul, Turkey

Competing interests: none declared

Correspondence to: Dr H Yanar, Trauma and Emergency Surgery Unit, Department of General Surgery, Istanbul Medical Faculty, University of Istanbul, Capa, Istanbul, 34390 Turkey; hyanar@yahoo.com

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**REFERENCES**


**More than just an ocular solution**

M Pekdemir, S Yanturali, G Karakus

Apraclonidine eye drop is an alpha adrenergic agonist derived topical clonidine, used for the treatment of intraocular pressure elevation. We report what is to our knowledge the first case of systemic toxicity of apraclonidine resulted from repeated local administration. Clinical manifestation of toxicity was similar to oral clonidine overdose. Toxicities of ocular drugs should always be considered when a patient presents with new systemic problems.

A praclonidine is a widely used topical ophthalmic alpha agonist used to reduce intraocular high pressure. We present a case of systemic toxicity of this drug. Our literature search revealed no reported case of systemic toxicity from local apraclonidine administration, thus we believe this is the first such case.

**CASE REPORT**

A 56 year old woman was taken to the emergency department (ED) of Kocaeli University because of altered mental status, difficulty in speaking, and drowsiness. She had undergone laser capsulotomy for cataract removal 1 day previously, after which apraclonidine hydrochloride ophthalmic solution (lopidine 0.5%; Alcon) was prescribed. The patient misunderstood the instruction of the medication, and administered the drug every 5 minutes. After 4 hours, she developed generalised weakness and headache.

On admission to the ED, her blood pressure (BP) was 170/110 mmHg and heart rate (HR) 60 beats/min. The patient’s complaint was considered to be associated with high blood pressure. She was given 25 mg captopril and discharged.

After discharge, she had continued to apply her eye medication at the same dosage. Eight hours later, she developed lowered consciousness, difficulty in speaking, and drowsiness. She had a history of hypertension for which she had been taking losartan. On her second admission to the ED, her vital signs revealed hypotension (BP 80/60 mmHg; HR 65 beats/min). She was drowsy (Glasgow Coma Score 13). No focal neurological deficit or other abnormality was found on physical examination. She was placed on a cardiac monitor and a saline infusion bolus was initiated. Initial serum electrolytes and complete blood count were within normal limits except for mild increased blood urea nitrogen and mild anaemia. ECG showed sinus bradycardia without any significant ischemic findings, and computed tomography of the brain was normal. During observation in the ED, she had developed bradycardia (HR 40 beats/min; BP could not be measured at this stage). After normal saline bolus and intravenous atropine 1 mg, blood pressure and heart rate returned to normal (BP 140/100 mmHg, HR 100 beats/min).

Detailed history discovered that she had used the entire 5 ml apraclonidine solution within 12 hours after the operation.

**Abbreviations:** BP, blood pressure; ED, emergency department; HR, heart rate; IOP, intraocular pressure

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There was no other reason to explain her central nervous system depression and cardiovascular compromise. She gradually improved, with all symptoms and findings disappearing within 48 hours. After full recovery, she was discharged.

**DISCUSSION**

It was estimated by nowadays, 200,000 cataract operations performed annually within the UK National Health Service. Laser capsulotomy is a widely used technique for cataract removal. A rise in intraocular pressure (IOP) is a common side effect of this technique. Apraclonidine 1% is a potent and relatively selective alpha-2 agonist used for the control or prevention of postoperative elevations in IOP, and was derived from the systemic antihypertensive drug clonidine. It was the first agent to be approved for reducing incidence of postoperative IOP spiking following laser capsulotomy.

Aralonidine is more polar and less lipophilic than clonidine, which probably allows less penetration into systemic circulation. It also does not cross the blood brain barrier and therefore does not cause systemic hypotension.

Oral clonidine toxicity manifests as central nervous system depression, cardiovascular compromise, and respiratory depression. Signs and symptoms of a clonidine overdose include altered mental status, bradycardia, hypotension, respiratory depression, hypothermia, and miosis. Intoxication from patch forms of clonidine have been reported. In these cases, depression of mental status, bradycardia, and hypotension predominated, similar to oral clonidine overdose.

The clinical presentation of our patient was similar to oral clonidine intoxication. The patient developed lowered consciousness, hypotension, and bradycardia. She was managed with saline infusion and atropine for symptomatic bradycardia and hypotension. Her cardiovascular and central nervous system findings improved completely during hospitalisation. Her increased blood pressure during the initial episode can be attributed to paradoxical hypertension, similar to oral clonidine overdose. Additionally, administration of antihypertensive treatment in this stage and use of her routine hypertensive treatment in this stage and use of her routine

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**Authors’ affiliations**

M Pekdemir, Department of Emergency Medicine, Kocaeli University, Kocaeli, Turkey
S Yanturali, Department of Emergency Medicine, Dokuz Eylul University, Izmir, Turkey
G Karakus, Department of Internal Medicine, Kocaeli University, Kocaeli, Turkey

Competing interests: none declared

Correspondence to: Dr S Yanturali, Dokuz Eylul University Medical School, Department of Emergency Medicine, 35340, Inciralti, Izmir, Turkey; sedat.yanturali@deu.edu.tr

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**Early carbon monoxide intoxication: happy to be poisoned?**

**S F J Clarke, A Crosby, D Kumar**

Carbon monoxide poisoning is the commonest cause of death by poisoning in the UK, and chronic exposure is thought to be a frequently missed diagnosis. Although much has been written about the signs and symptoms of both acute and chronic intoxication, the features of early, mild, acute exposure have received much less attention. Early recognition of carbon monoxide poisoning is vital to institute prompt treatment and to prevent exposure to others.

We describe an episode of mass exposure to carbon monoxide that resulted in previously unreported changes in mood, which may provide a useful warning symptom.
Early carbon monoxide intoxication: happy to be poisoned?

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Acute aortic dissection provoked by sneeze: a case report

A Baydin, M S Nural, H Güven, T Deniz, F Bildik, A Karaduman

The response of the abdominal viscera and the contraction of the intercostal muscles during the respiratory phase of sneezing increases intrathoracic pressure, which may lead to several complications. However, there are no reports in the literature concerning aortic dissection after sneezing. We report a patient in whom the development of dissection was secondary to sneezing, although hypertension was present as a risk factor, and we discuss the relationship between sneezing and aortic dissection. To our knowledge, this is the first report of aortic dissection provoked by sneezing in the literature.

Aortic dissection occurs from a rupture of the intima, which allows blood to enter the media and dissect between the intimal and the adventitial layers. The estimated incidence of aortic dissection is 5 to 30 cases per million population per year. Chronic systemic hypertension is the most common factor predisposing to aortic dissection and is present in 62–78% of patients with aortic dissection.

The increase in intrathoracic pressure during sneezing may lead to serious complications. We present a case in which the development of aortic dissection was considered secondary to sneezing, although hypertension was present as a risk factor, and we discuss the relationship between sneezing and aortic dissection. To our knowledge, this is the first reported case of aortic dissection provoked by sneezing.

CASE REPORT

A 51 year old man was admitted to the emergency department (ED) with severe, sudden onset chest pain and dyspnoea following sneezing. The patient reported that the pain was very severe and by the time he arrived at the ED (a period of about 20 minutes), had spread towards his back. He had a history of hypertension diagnosed 5 years previously, which was regulated with nifedipine (Adalat) and lisinopril 20 mg plus hydrochlorothiazide 12.5 mg (Sinoretik).

On physical examination, blood pressure was 190/110 mmHg, pulse rate 90 beats/min, respiration rate 24 breaths/min and temperature 36°C. He was conscious, cooperative, and in moderate health. All other physical findings were normal. Laboratory findings showed glucose of 1.14 g/l (normal 0.7–1.1 g/l) and albumin 33 g/l, while other findings were within normal limits. There was no abnormal finding on 12 lead electrocardiography.

Chest x ray revealed a widening in the aortic knob and the lateral margin of the descending aorta (fig 1A). Spiral computed tomography (CT) was performed to rule out aortic dissection, but showed an intimal flap that seperated the aortic lumen into two parts, extending from the proximal descending aorta to the left common iliac artery. The ascending aorta and supraaortic branches were normal (fig 1B). Using transoesophageal echocardiography, a dissection was detected starting just after the subclavian artery orifice and extending downwards distally.

After clinical and radiological evaluation, a Stanford type B aortic dissection was diagnosed and medical treatment commenced in the ED, after which the patient was admitted to a hospital ward. During follow up, the chest pain had not subsided and the patient's blood pressure could not be controlled despite appropriate treatment. A control CT scan taken 1 week later showed some abnormalities in the descending aortic contour at the level of right pulmonary artery orifice and a small effusion in the left pleural space (fig 1C). The patient underwent surgery for possible aortic aneurysm rupture.

Figure 1 (A) Chest radiograph shows widening of the aortic knob and the lateral margin of the descending aorta (white arrow). (B) Type B aortic dissection. Axial contrast enhanced spiral CT scan shows an intimal flap in the descending aorta that separated the lumen into two parts; true (black arrow) and false (white arrow) lumens. The ascending aorta is normal. (C) CT scan obtained at the level of the right pulmonary artery showing mild irregularities in the lateral contour of the descending aorta (black arrow) and associated left pleural effusion.
DISCUSSION
During the sneeze reflex, the intrathoracic pressure is raised, which may lead to several complications. Brock et al reported a case of chest pain following sneezing, which occurred from fractures of ribs 8, 9, 10, and 11. There are some reports of lung herniation triggered by sneezing, as demonstrated on chest x-ray.  

The increase in intrathoracic and intra-abdominal pressure raises the venous pressure in these spaces, leading to a further increase in the epidural veins and a corresponding increase in intracranial pressure. A case has been reported with a temporary hemiparesis caused by a sneezing induced intracranial aneurysm. Sharir et al reported a patient who had an attack of acute angle closure glaucoma precipitated by sneezing, which may have been the result of an increase in the ocular venous return precipitated by the increase in the intrathoracic pressure. Whitehead et al and Azem and Calderelli have reported patients who developed hearing loss in association with stapes fracture due to increased intracranial pressure following sneezing. 

Coughing, sneezing, and the other Valsalva manoeuvres not only increase intrathoracic pressure but also alter the haemodynamics. Initially, the arterial pressure increases with a rise in respiratory tract pressure, but then decreases following a decline in venous return. It has also been reported in another study that systolic and diastolic arterial pressure increase during the Valsalva manoeuvre. Systemic hypertension, one of the most common predisposing factors of acute aortic dissection, increases the tension on the aortic wall. We believe that in our hypertensive patient, the increase in arterial pressure caused by the rise in intrathoracic pressure during sneezing, enhanced the aortic wall tension, and that this sudden increase of pressure on the aortic wall triggered the intimal tear.

Aortic dissection is an important disease because, although rare, it may be life threatening. In over 38% of patients the diagnosis is missed at the first evaluation. In untreated patients, the mortality rate is 25% within 24 hours, 70% by 2 weeks and 90% after 2 weeks.

Chest pain is the most common symptom in aortic dissection cases presenting to the ED. Typically, the pain starts suddenly and is unbearable. Pain is the most intense when the symptoms begin. The localisation of the pain is related to the region in which the intimal rupture occurs; if the tear is above the aortic valve the pain is felt anteriorly, but if it is distal to the left subclavian vein it is felt in the back. Spreading of the pain may be a sign of progressive dissection. It has been reported that >90% of the patients with acute aortic dissection undergo intense chest pain. In our patient, the chest pain spread to the back, and the dissection was detected in the descending aorta.

It has been reported that >90% of the patients with aortic dissection have widening of the mediastinum and aortic knob detected by chest radiography. In a study by Kodolitsch et al, it was found that that mediastinal and/or aortic widening was related to dissection in 39% of cases. In addition, it has been found that the existence of chest pain and changes in pulsation and blood pressure are related to the dissection in 83–100% of cases. In our case, there was a typical spontaneous and spreading type of chest pain. A widening of the aortic knob and the descending aortic lateral wall was revealed by chest radiography. However, the blood pressure was high but the pulse was normal.

CONCLUSION
Aortic dissection should be considered a possible diagnosis in patients who present to the ED with complaints of atypical chest pain after sneezing. If the patient’s history implies intrathoracic pressure increase and other suggestive findings are present, as in our case, noninvasive diagnostic methods such as CT, transoesophageal echocardiography and magnetic resonance imaging should be performed promptly to rule out aortic dissection, which is a very severe life threatening condition. Aortic dissection may be added to the complications seen after sneezing.