Acute cardiac failure during influenza A (H1N1) treated with noninvasive ventilation

Primena neinvazivne ventilacije kod akutne srčane insuficijencije uzrokovane influencom A (H1N1)

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ABSTRACT

The female patient, 64 year old, was hospitalized in the intensive care unit of Clinic for Pulmonology, Clinical Center Kragujevac, due to fever, dry cough, dyspnea, fatigue and swelling of lower limbs. The symptoms started abruptly, 4 days before the admittance to the hospital. Upon admission, somnolence and signs of central cyanosis were noted. Auscultatory finding on the lungs was characterized by bilateral inspiratory crackles, while cardiac sound was silent, HR=120/min, TA=110/60mmHg. Analysis of arterial blood gases registered acute respiratory failure: pO₂=5.1kPa, pCO₂= 9.2kPa, pH=7.45, SAT=62%. Chest X-ray showed bilateral condensations in the lung parenchyma, predominantly in parahilar area. Cardiac shadow was enlarged. Laboratory tests showed increased value of pro-BNP (4086 pg/mL) (reference values: < 125 pg/mL – negative; 125–400 pg/mL – intermediate; > 450 pg/mL – heart failure). Echocardiographic exam upon admittance showed dilated cardiac cavities, global hypokinesis and ejection fraction of 35%. Noninvasive ventilation (NIV) treatment was started (BiLEVEL, IPAP 26cmH₂O, EPAP 6cmH₂O) at first continuously and then only during the night, with intense cardio-diuretic therapy. On the fifth day of hospitalization, the results of virology analyses confirmed influenza A (H1N1) infection. On the 6th day, gas exchange was normalized, complete radiological regression occurred, as well as normalization of echocardiographic exam finding. Our case shows that influenza A (H1N1) virus infection may be complicated by acute cardiac failure. If started on time, NIV treatment with simultaneous cardio-diuretic therapy, could have a positive impact on the resolution of acute heart failure during H1N1 virus infection.

Key words: respiratory insufficiency; noninvasive ventilation; influenza A virus, H1N1 subtype; heart failure; echocardiography.

APSTRAKT

Bolesnica stara 64 godine hospitalizovana je u našoj klinici u jedinici intenzivne nege zbog visoke febrilnosti, suvog kašlja, gušenja, lakog zamaranja i otoka potkolenica. Navedeni simptomi počeli su naglo 4 dana pre prijema. Na prijemu pacijentkinja somnolentna sa znacima centralne cijanoze. Auskultatornim nalazom na plućima registruju se pukoti u inspirijumu obostrano, dok su srčani tonovi mukli HR=120/min, TA=110/60mmHg. Uzete gasne analize arterijske krvi pokazale su akutnu globalnu respiratornu insuficijenciju: pO₂=5.1kPa, pCO₂= 9,2 kPa, pH=7.45, SAT=62%. Radiološkim pregledom pluća registrovane su kondenzacije plućnog parenhima obostrano, levo i desno dominantno parahilarno, srčana senka uvećana. U laboratorijskim analizama registrovane su povišene vrednosti proBNP (4086) (referentne vrednosti negativan < 125; siva zona 125-400; sigurna insuficijencija > 450 pg/ml). Ehokardiografski nalaz na prijemu pokazao je dilatirane srčane šupljine, globalnu hipokineziju i EF od 35%. Započeli smo tretman NIV-om (BiLEVEL, IPAP 26, EPAP 6cmH₂O) kontinuirano a potom samo u toku noći uz intenzivnu kardio-diuretsku terapiju. Petog dana hospitalizacije, pristigao je virusološki rezultat, koji je potvrdio infekciju virusom gripa A (H1N1). Šestog dana dolazi do normalizacije gasne razmene, kompletne regresije na kontrolnoj radiografiji pluća i normalizacije ehokardiografskog nalaza. Naš prikaz pokazuje da infekcija virusom gripa A (H1N1) može biti komplikovana akutnom srčanom insuficijencijom. Ako se započne na vreme primena NIV-a, uz kardio-diuretsku terapiju, može imati pozitivan uticaj na rešavanje akutne srčane insuficijencije u toku H1N1 virusne infekcije.

Ključne reči: respiratorna insuficijencija; neinvazivna ventilacija; influenza A virus, H1N1 podtip; srčana insuficijencija; ehokardiografija.

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INTRODUCTION

In the last decade viral pandemics had a significant impact on the health of populations worldwide. Pandemic influenza A (H1N1) virus, during 2009, caused more than 18 000 lethal outcomes within the first season.¹ The association of infection with influenza A (H1N1) virus and acute, reversible cardiac dysfunction was noticed during pandemic.^{2,3}

CASE REPORT

64 year old female patient was hospitalized in the intensive care unit of Clinic for Pulmonology, Clinical Center Kragujevac, in a serious general condition. The symptoms started abruptly, 4 days before the admittance to the hospital due to high fever (39°C), dry cough, dyspnea, weakness, fatigue and swelling of lower limbs. Previously, she was treated for hypertension. She is non-smoker. Upon admission, somnolence, high fever (38.4°C), tachycardia (100 bpm), tachypnea (rf: 30/min), with signs of central cyanosis were noted. Auscultatory finding on the lungs was characterized by impaired breathing sound, prolonged expirium, and the mass of bilateral late-inspiratory crackles, while cardiac sound was silent. Pretibial edemas were noted at lower extremities. Analysis of arterial blood gases on admittance registered acute respiratory failure: $pO_2=5.1\text{ kPa}$, $pCO_2=9.2\text{ kPa}$, $pH=7.45$, $SAT=62\%$. Electrocardiogram on admission showed sinus rhythm, with heart rate of 82/min, ST depression in D1, D2, incomplete right bundle branch block. Chest X-ray showed bilateral condensations in the lung parenchyma, diffuse in the left lung and predominantly in parahilar area in the right lung. Cardiac shadow was enlarged. Costophrenic angles were without fluid. (Figure 1).

Figure 1. Chest radiography at hospital admission

Laboratory tests on admission showed: WBC within reference value ranges, slightly increased CRP value (25.6 mg/L) (reference value < 5.0 mg/L), cardiospecific markers of myocardial damage within reference value ranges, increased value of pro-BNP (4086 pg/mL). Echocardi-

graphic exam upon admittance showed dilated cardiac cavities, global hypokinesis and ejection fraction of 35%. (EDD 64mm, ESD 23mm, VS 10mm, PW 10mm, EF 35% Simpson, Ao 34mm, LA 42mm, RV 31mm, PA 25mm, MR mild to moderate, TR moderate, SRVP 49mmHg) (Figure 2).

Figure 2. Echocardiography on admission

Suspecting the infection with influenza A (H1N1) virus, throat swab was taken, which was sent to the tertiary institution for definitive confirmation of the diagnosis.

Two hours after the admission, as there was no improvement in oxygenation with strictly controlled oxygen therapy, NIV treatment was started (BiLEVEL, bilevel positive pressure support ventilation. IPAP 26cm H₂O, EPAP 6cm H₂O, back-up rate 24) continuously and then only during the night, with intense cardio-diuretic therapy. During hospitalization, because of the occurrence of moderate hemoptysis and high value of D-dimer (1.77 µg/ml) (reference values: negative < 0,50 5 µg/ml FEU), due to suspicion of pulmonary thromboembolism, MSCT pulmonary angiography was performed, thereby excluding the existence of significant pulmonary embolism, but confirming the hyperintense fluid condensation of the type of ground glass, on both sides in the upper lobes predominantly on left side, which could be attributed to lung congestion (Figure 3)

Figure 3. Pulmonary angiography

On the fifth day of hospitalization, the results of virology analyses confirmed influenza A (H1N1) infection. (Figure 4)

Figure 4. H1N1 confirmation

On the 6th day of hospitalization, gas exchange was normalized (PaO_2 9,4Kpa, PaCO_2 5,7Kpa), as well as echocardiographic exam finding. (EDD 55mm, ESD 35mm, VS 10mm, PW 10mm, EF 50% Simpson, Ao 34mm, LA 39mm, RV 26mm, PA 21mm, MR mild, Tr mild, SRVP 31mmHg). (Figure 5)

Figure 5. Echocardiography on the 6-th day of hospitalization

Control chest X-ray showed complete regression of previously noted changes (Figure 6).

Figure 6. Chest radiography on hospital discharge

DISCUSSION

Our case report describes the patient with Influenza A (H1N1) virus infection complicated by acute myocardial dysfunction, even though the patient had no prior history of heart failure. Systolic dysfunction that occurs in the acute viral syndrome and passes away after resolution of the viral illness, supports the concept that influenza A (H1N1) is the reason of acute heart failure caused by virus toxic effects on the myocardium².

Reviewing the literature, acute heart failure, in most of the cases, is developing during the first week of illness (3). In the patient that we presented, clinical, laboratory, radiographic and echocardiographic signs of heart failure were registered on admission. Acute heart failure in patients with influenza A (H1N1) infection is usually manifested by viral myocarditis, previous coronary disease exacerbation or worsening of congestive heart failure³.

Although influenza A (H1N1) virus infection is not displayed with signs of heart failure, literature review suggests that the infection with this virus may be associated with cardiac failure and the cardiac arrest as the primary clinical manifestation. In cases, when presented with respiratory tract infections, alongside which there are signs of heart failure, it is necessary to test patients for influenza A (H1N1) virus infection⁵.

According to literature data, independent factors associated with the success of the application of NIV in acute heart insufficiency and influenza A (H1N1) virus infection are hemodynamic stability and presentation of changes (opacification) on radiological examinations of the lungs in less than two quadrants of the lung parenchyma⁶. In our patient, the significant changes were seen in at least three quadrants. Patients treated with NIV had a lower incidence of new infectious complications and an increase in in-hospital survival when compared to those not treated with NIV^{4,7}. Early use of NIV is associated with rapid improvement of gas exchange in the first few hours from the beginning of the application, as it is in our patient⁶. Studies have shown that NIV is efficient in preventing endotracheal intubation in 48% of patients with acute cardio- respiratory insufficiency during the H1N1 infection⁴. In acute heart failure NIV improves cardiac performance by reducing the "preload" and "afterload", improves respiratory function by reducing the work of breathing musculature which, in overall, leads to an improvement in oxygenation⁸.

CONCLUSION

It could be concluded that our case shows that influenza A (H1N1) virus infection may be complicated by acute cardiac failure. If started on time, NIV treatment with simultaneous cardio-diuretic therapy, could have a positive impact on the resolution of acute heart failure during H1N1 virus infection.

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