Annals of Case Reports & Reviews

Case Report

doi: 10.39127/2574-5747/ACRR:1000121.
Erragh A, et al. Annal Cas Rep Rev: ACRR-121.

Propofol Infusion Syndrome in a COVID19 Patient: A Case Report

Anas Erragh, Mohamed Anass Fehdi, Hasna Darouich, Afak Nsiri, Ouissal Aissaoui, Rachid Alharrar

*COVID-19 Dedicated ICU University Hospital of Casablanca, Morocco
**Hassan II University Ain Chock, Faculty of medicine and pharmacy, Casablanca, Morocco

Correspondent author: Anas Erragh, COVID-19 Dedicated ICU University Hospital of Casablanca, Morocco. E-mail: Erragg44anas@gmail.com

Citation: Erragh A, Fehdi MA, Darouich H, Nsiri A, Aissaoui O (2020) Propofol Infusion Syndrome in a COVID19 Patient: A Case Report. Annal Cas Rep Rev: ACRR-121.

Received Date: 19 May 2020; **Accepted Date:** 21 May 2020; **Published Date:** 28 May 2020

Introduction

SARS-CoV-2 causes a severe respiratory illness, named "COVID-19" by the World Health Organization (WHO), responsible for a pandemic today. The management of these patients requires hospitalization in intensive care unit and the use of mechanical ventilation with prolonged sedation. Propofol is one of the molecules that can be used.

Propofol infusion syndrome (PRIS) is rare, occurring after long-term administration of propofol. It is a real challenge for the critical care practitioner.

We report the case of a patient with COVID-19 with PRIS.

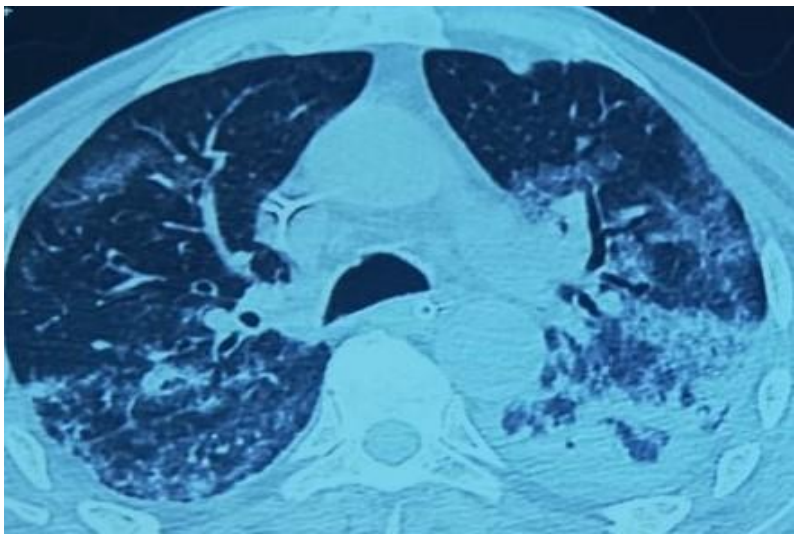
Case

FA, 70 years old, with no particular pathological history, admitted for SARS-COV-2 viral pneumonia at the stage of acute respiratory distress syndrome (ARDS), the interrogation found that the patient had been travelling in an endemic area (the city of Fez) a week before the onset of symptoms. The diagnosis confirmation was made by PCR sampling on nasopharyngeal swab.

The clinical evaluation on admission found a conscious patient, with a GCS at 15/15, with a respiratory rate at 34 cycles / min, an oxygen saturation SpO2 at 85% in ambient air. The patient was hemodynamically stable, he had a capillary glycemia at 1.2 g / L, with a fever at 38.8 ° C. The initial arterial blood gas analysis showed a deep hypoxemia with a PaO2 at 40 mmHg.

A high-resolution computed tomography was performed, showing bilateral ground-glass opacities, with multilobe central and peripheral involvement (Figure 1).

Figure 1:



A biological assessment was carried out objectifying an inflammatory syndrome marked with a hyperferritinemia at 3350 ng/mL, a CRP at 520 mg/L, a lymphopenia at 360 / mm3. In addition, the patient had a normal renal and hepatic function and normal cardiac enzymes.

Oxyegen therapy was initiated using a high concentration mask and a peripheric venous line was inserted. Specific COVID-19 therapeutics associating Hydroxychloroquine

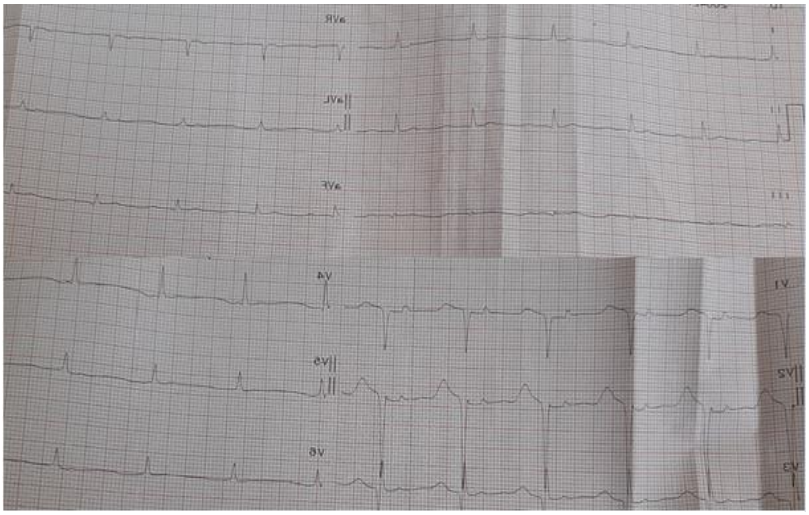
200mg x 3 / day, Azithromicyne 500mg / day and methylprednisolone 60mg / day were administrated.

The patient was intubated after 2 days, protective ventilation was stared, and continuous sedation associating Midazolam 3 mg / h and Fentanyl 50 µg / h was administrated. Hemodynamic instability required the use of norepinephrine.

Due to the appearance of a long QT on the electrocardiogram, Hydroxychloroquine and Azithromycine treatment was discontinued.

Few days later, given the shortage of midazolam, we put the patient on propofol at 4 mg / kg / h for 7 days. The patient presented a disturbance on biological scale, with acute kidney failure (urea = 2g / l, creatine = 22mg / l), creatinine clearance estimated at 31 mL/min, hyperkalemia at 6.5 meq/L, metabolic acidosis and slight hepatic cytolysis. An ECG was performed showing bradycardia with long QT syndrome (figure2).

Figure 2 :



A transthoracic echocardiography was performed and it was normal. The patient management steps were to stop propofol immediately with optimization of the vascular filling and fluids, with symptomatic measures of hyperkaliemia, close monitoring by ECG and daily biological assessments.

The evolution was marked by an improvement in renal function and metabolic acidosis and normalization of kaliemia, and regression of long QT on the ECG.

The diagnosis of PRIS syndrome was retained on the basis of the following criteria : the onset of renal failure and hyperkalaemia, hepatic cytolysis and metabolic acidosis, with the electrical signs in the ECG.

Discussion

PRIS is a rare syndrome, occurring after prolonged administration of propofol in intensive care patients, most often in children with severe diseases [1].

The use of large doses of propofol (> 4 ml/Kg/h), glucocorticoids or catecholamines seem to favor the onset of PRIS [2,3,4], as in the case of our patient.

Arrhythmias and impaired myocardial contractility are the first symptoms of proven PRIS. There is an increase in the concentrations of creatine kinase, lactate dehydrogenase, cardiac troponin I and myoglobinuria [5,6,7,8,9,10,11,12]. These alterations reflect necrosis of the skeletal muscles and the heart muscle. Our patient did not have a disturbance of myocardial enzymes or rhabdomyolysis.

Propofol changes the response of the autonomic nervous system. It decreases the sympathetic tonus more significantly than the parasympathetic tone; therefore, it promotes the appearance of hypotension and bradycardia. Finally, all the factors that will promote the degeneration of myocardial myofibrils also produce a proarrhythmic effect, in particular the concomitant use of catecholamines. Several abnormalities are found on the ECG such as long QT, an idioventricular rhythm, then tachycardia or ventricular fibrillation before cadiac arrest [13]. Our patient had bradycardia with long QT reappearing 10 days after the association Hydroxychloroquine-Azithromycine was discontinued.

The kidney failure observed during PRIS is multifactorial, often worsened by heart failure [14,15]. Our patient had kidney failure that improved after stopping propofol infusion. Hepatomegaly, as well as the fatty changes observed in the liver and impaired liver function, are caused by several mechanisms [16]. In our patient we noted the existence of a moderate hepatic cytolysis without lipid disturbance nor hepatomegaly.

Isolated lactic acidosis is a precursor and indicator of PRIS in children, as well as in adults. Stopping propofol allows a regression of acidosis and a favorable clinical outcome. At a later stage, signs of multivisceral failure develop and the outcome is most often fatal [17,18,19,20]. Our patient had developed metabolic acidosis which improved after stopping propofol infusion.

Management is mainly based on stopping the propofol infusion as well as managing the various multi-organ failures [21,22,23,24,25].

We cannot prove that there is any relation between Covid-19 and the PRIS syndrome. To our knowledge no such case has ever been reported.

Conclusion

The PRIS syndrome is a rare but severe complication of prolonged admistration of propofol. Its management is a real challenge for critical care physiciens.

Ethical approval

Informed consent was obtained from the patient family for publication of this case report.

Declaration of interests

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Authors contributions

Anas Erragh, Mohamed Anass Fehd, Hasna Darouichi: contributed to drafting of manuscript.
Afak Nsiri, Ouissal Aissaoui: contributed to critical revision.
Rachid Alharrar: contributed to conception and final approval.

References

1. Bray RJ. Propofol infusion syndrome in children. Paediatr Anaesth 1998;8:491–9.
2. Rosen DJ, Nicoara A, Koshy N, Wedderburn RV. Too much of a good thing? Tracing the history of the propofol infusion syndrome. J Trauma 2007;63:443– 7.
3. Marinella MA. Lactic acidosis associated with propofol. Chest 1996;109:292.
4. Zaccheo MM, Bucher DH. Propofol infusion syndrome: a rare complication with potentially fatal results. Crit Care Nurse 2008;28:18–26
5. Parke TJ, Stevens JE, Rice AS, Greenaway CL, Bray RJ, Smith PJ, et al. Metabolic acidosis and fatal myocardial failure after propofol infusion in children: five case reports. BMJ 1992;305:613–6
6. Wolf A, Weir P, Segar P, Stone J, Shield J. Impaired fatty acid oxidation in propofol infusion syndrome (lettre). Lancet 2001;357:606–7.
7. Ozlu¨ O, Ozkara HA, Eris S, Ocal T. Propofol anaesthesia and metabolic acidosis in children. Paediatr Anaesth 2003;13:53–7.
8. Withington DE, Decell MK, Al Ayed T. A case of propofol toxicity: further evidence for a causal mechanism. Paediatr Anaesth 2004;14:505–8.
9. Koch M, De Backer D, Vincent JL. Lactic acidosis: an early marker of propofol infusion syndrome? Intensive Care Med 2004;30:522.
10. Kill C, Leonhardt A, Wulf H. Lacticacidosis after short-term infusion of propofol for anaesthesia in a child with osteogenesis imperfecta. Paediatr Anaesth 2003;13:823–6.
11. Sabsovich I, Rehman Z, Yunen J, Coritsidis G. Propofol infusion syndrome: a case of increasing morbidity with traumatic brain injury. Am J Crit Care 2007;16:82–5.
12. Laquay N, Pouard P, Silicani MA, Vaccaroni L, Orliaguet G. Early stages of propofol infusion syndrome in paediatric cardiac surgery: two cases in adolescent girls. Br J Anaesth 2008;101:880–1
13. Vernooy K, Delhaas T, Cremer OL, Di Diego JM, Oliva A, Timmermans C, et al. Electrocardiographic changes predicting sudden death in propofol-related infusion syndrome. Heart Rhythm 2006;3:131–7
14. Stelow EB, Johari VP, Smith SA, Crosson JT, Apple FS. Propofol-associated rhabdomyolysis with cardiac involvement in adults: chemical and anatomic findings. Clin Chem 2000;46:577–81
15. Zarovnaya EL, Jobst BC, Harris BT. Propofol-associated fatal myocardial failure and rhabdomyolysis in an adult with status epilepticus. Epilepsia 2007; 48:1002–6.
16. Ahlen K, Buckley CJ, Goodale DB, Pulsford AH. The propofol infusion syndrome': the facts, their interpretation and implications for patient care. Eur J Anaesthesiol 2006;23:990–8
17. Burow BK, Johnson ME, Packer DL. Metabolic acidosis associated with propofol in the absence of other causative factors. Anesthesiology 2004;101:239–41.
18. Salengros JC, Velghe-Lenelle CE, Bollens R, Engelman E, Barvais L. Lactic acidosis during propofol-remifentanil anesthesia in an adult. Anesthesiology 2004;101:241–3.
19. Liolios A, Gue´ rit JM, Scholtes JL, Raftopoulos C, Hantson P. Propofol infusion syndrome associated with short-term large-dose infusion during surgical anesthesia in an adult. Anesth Analg 2005;100:1804–6
20. Haase R, Sauer H, Eichler G. Lactic acidosis following short-term propofol infusion may be an early warning of propofol infusion syndrome. J Neurosurg Anesthesiol 2005;17:122–3
21. Badr AE, Mychaskiw G, Eichhorn JH. Metabolic acidosis associated with a new formulation of propofol. Anesthesiology 2001;94:536–8
22. Zaccheo MM, Bucher DH. Propofol infusion syndrome: a rare complication with potentially fatal results. Crit Care Nurse 2008;28:18–26
23. Kam PC, Cardone D. Propofol infusion syndrome. Anaesthesia 2007;62:690– 701.
24. Deutschman CS, Harris AP, Fleisher LA. Changes in heart rate variability under propofol anesthesia: a possible explanation for propofol-induced bradycardia. Anesth Analg 1994;79:373–7.
25. Holzki J, Aring C, Gillor A. Death after re-exposure to propofol in a 3-year-old child: a case report. Paediatr Anaesth 2004;14:265–70.