

Propanil Poisoning Presenting with Methaemoglobinemia: A Case Report

ABHIJIT NANDA¹, DENNIS MARTIN DAVID², SUNA DENISH KUMAR³, JOEL NANDA⁴, ALISHA JESSY KISPOTTA⁵



ABSTRACT

Propanil is an uncommon cause of poisoning, and the incidence of it in India is unknown. It is a low to medium toxicity agent; however, severe poisoning can lead to death, especially in areas with limited medical facilities. Hereby, the authors present a case of 15-year-old girl who presented to Emergency Department after 72 hours of ingesting an unknown quantity of propanil. She was noted to have peripheral cyanosis, laboured breathing, and a room air saturation of 62%, for which she was intubated and ventilated. Methaemoglobinemia was suspected in the child, which is considered a complication of propanil poisoning, and methylene blue was administered within six hours of admission. Exchange transfusion was performed via a femoral central line catheter when there was no improvement with methylene blue. Sulphaemoglobinemia was also considered as a differential diagnosis. On day 4 of the hospital stay, her saturation improved to 80%. She experienced complications of haemolysis, which were managed conservatively. She improved clinically and was extubated on day 5. Her room air saturation was 93% on day 9, and she was discharged. She had a follow-up appointment in the outpatient clinic after two weeks, where her saturation on room air was recorded as 97%, and her methaemoglobin levels were 8.7% in the postexchange transfusion sample. Propanil has the potential to cause severe, life-threatening clinical symptoms. Exchange transfusions can be lifesaving in situations of severe poisoning. Early transfer of patients to tertiary care Institutions should be considered as peripheral hospitals may lack intensive care facilities and exchange transfusion capabilities.

Keywords: Cyanosis, Haemolysis, Transfusion

CASE REPORT

A 15-year-old girl with no previous co-morbidities presented with an alleged history of consuming Propanil 80% three days prior to presentation. She was initially managed elsewhere with gastric lavage and other supportive care (details of which were not available). She presented with peripheral cyanosis, laboured breathing, and room air saturation of 62%. Arterial blood gas done after intubation followed by bag and tube ventilation showed a high PaO₂ of 426 mmHg, with SpO₂ at 100%. The arterial sample collected was dark red in appearance [Table/Fig-1]. The above features were suggestive of methaemoglobinemia, but co-oximetry was unavailable to confirm the same.



[Table/Fig-1]: Dark coloured blood sample.

A bedside Methemoglobin reference chart [1] was used to compare the colour, which suggested levels >80%. Inj. methylene blue was administered (2 mg/kg) as per recommendations [2], and it was given within six hours of hospitalisation; however, there was no improvement. Relatives were informed about the critical state and advised to seek care at a higher centre, but they chose to

continue treatment at facility. A decision was made to perform an exchange transfusion, which was conducted via the femoral central line on day 2 of the hospital stay. Approximately, 1100 mL of blood was withdrawn, and 1000 mL of whole blood was administered simultaneously based on blood availability. However, there was minimal improvement immediately after the procedure. The Methemoglobin levels were unknown. The bedside Methemoglobin chart indicated gross levels corresponding to approximately 60%, which was an inadequate response. Additionally, there was a lack of sufficient blood for proper replacement.

The case was reassessed, and alternative diagnosis were considered. Tube displacement and pneumothorax were deemed unlikely as there was equal air entry upon examination findings. Sulphaemoglobinemia was also considered as a differential diagnosis since, the poison contained a sulfur compound, for which N-Acetyl cysteine was administered on day 3 of the hospital stay [3]. Given that methaemoglobinemia is a known complication of propanil poisoning and due to the insufficient blood exchange, a second dose of Methylene Blue (2 mg/kg) was administered. It was observed that exchange transfusion is a treatment option for both methaemoglobinemia and sulphaemoglobinemia.

Following the treatments described above, the patient's saturation improved to 80% on day 4. It remained unclear which treatment (N-acetyl cysteine or methylene blue) was effective. Methemoglobin levels were sent to an available laboratory. Her saturation continued to improve, reaching above 90% on day 5. She was gradually weaned off the ventilator to T-piece on day 5 and successfully extubated on day 6. Serial metabolic parameters were monitored and remained within normal limits [Table/Fig-2,3]. Oxygen was tapered and discontinued on day 7. The patient experienced complications of haemolysis, which were managed conservatively. She remained clinically stable, and her room air saturation was 93% on day 9 when she was discharged. A follow-up after two weeks showed a room air SpO₂ of 97%. Methemoglobin levels were reported at 8.7% (levels were measured post-methylene

blue, exchange transfusion, and N-acetylcysteine), confirming the diagnosis of methaemoglobinemia.

ABG	Admission	Midway-transfusion	Post-transfusion	Day 3	Day 5	Day 9
pH	7.51	7.44	7.45	7.57	7.533	7.54
PCO ₂ (mmHg)	27.6	30.5	33.2	29.4	38.3	31.7
PO ₂ (mmHg)	426	9	114	225	262	58
HCO ₃ (mEq/L)	22.5	20.9	23.2	27	32.3	27.5
SPO ₂ (%)	99	99	99	100	100	93

[Table/Fig-2]: Blood gas results during the course of admission.

Parameters	Admission	Pre-exchange transfusion	Midway	Post-exchange transfusion	Day 3	Day 5	Day 7	Day 9
Laboratory parameters								
Haemoglobin (gm%)	11.4	10.5	9.6	9.1	7.1	7.6	7.5	6.8
Platelets per microlitre						94,000	69,000	
Total white blood cells per microlitre of blood						8.27		
Sodium (mEq/L)	149		149	149	139	131	132	136
Potassium (mEq/L)	2.5		3.6	3.6	2.8	3.1	3.2	3.4
Creatinine (mg/dL)						0.6		
Liver functions								
Aspartate aminotransferase (units/litre)					306	411		
Alanine transaminase (units/litre)					223	323		
Alkaline Phosphatase (units/litre)					123	131		
Total Bilirubin (milligram/decilitre)					1.58	1.49		
Direct Bilirubin (milligram/decilitre)					0.47	0.50		
Total Protein (gram/decilitre)					6.0	6.1		
Albumin (gram/decilitre)					2.9	2.8		

[Table/Fig-3]: Metabolic parameters during the course of admission.

DISCUSSION

Propanil, a very potent herbicide belonging to the acetanilide group, is supplied as 36% solutions and is marketed under at least 20 different brand names [4]. In present case author managed to demonstrate that poisoning in a severe form is feasible, especially when taken in huge doses. Around 10 mL of the chemical, undiluted, is the deadly dose. When propanil is diluted, any amount over 200 mL is regarded as severe poisoning [4].

Although symptoms of poisoning appear shortly after ingestion, the period of time to death is typically longer than 24 hours, providing more time for intervention to prevent death. The rate at which methaemoglobinaemia develops is associated with the degree of toxicity [5]. Cyanosis, hypotension, acidosis, progressive end-organ dysfunction are compatible with severe and long-term Methaemoglobinaemia [6]. The biotransformation of propanil into 3,4-dichlorophenylhydroxylamine, which is then co-oxidised with oxyhemoglobin (Fe²⁺) in erythrocytes to the ferric state (Fe³⁺), causes Methaemoglobinaemia [7]. Other less common symptoms include: haemolysis, hepatitis (inflammation of the liver), which may be due to direct oxidant damage of the Red Blood Cell (RBC) [8]. Rittilert P et al., showed in their retrospective cohort study on various clinical manifestations of propanil poisoning that the majority included gastrointestinal symptoms (65.5%). Methaemoglobinemia and haemolysis were observed in 108 patients (39.3%) and 25 patients (9.1%), respectively [6]. The present patient had a rapid onset of methaemoglobinemia, as shown by cyanosis, altered awareness, and low oxygen saturation. This could have been caused by a high dose of propanil intake, which caused a rapid generation of Methemoglobin.

The medication of choice for methaemoglobinemia is intravenous methylene blue at doses of 1-2 mg/kg. It works by accelerating the rate at which Methemoglobin transforms into haemoglobin [2]. In present patient, who received repeated doses of methylene blue with

little effect, this is possible in cases of severe poisoning. Such patients may require cutting-edge therapeutic approaches, such as exchange transfusion, which substitutes Methemoglobin and removes the toxin from the body [4,6,9]. Therefore, we advise prompt patient transfer to tertiary care centers with exchange transfusion capabilities in cases of severe poisoning. But there are instances, like the one involving our patient, where a transfer is not urgently possible.

Due to minimal response to methylene blue in our patient, an exchange transfusion was performed. Postexchange transfusion, there was minimal improvement, and an alternative diagnosis of Sulfhaemoglobinemia was considered, and the patient was

also given N-acetylcysteine [3]. The role of N-acetylcysteine in methaemoglobinemia is unclear and controversial, which has been discussed by Woo SH et al., [10]. Following, the above management, there was rapid improvement. Although the alternative measure proved effective, authors anticipated the need for more studies to identify treatment modalities that are practical in situations with limited resources.

CONCLUSION(S)

Propanil causes serious clinical effects when ingested in large quantities and has been classified as a mildly toxic compound, but it can be fatal if, not treated appropriately. Treatments such as intravenous methylene blue or exchange transfusion are not available in local hospitals or are not easily accessible in local hospitals that have a high number of cases of acute propanil poisoning. Therefore, early transfer to a tertiary care hospital should be taken into consideration. When transfer to a tertiary care hospital is not possible due to a lack of facilities, alternative treatment options are required. This is not uncommon in remote places in India. In present case, methylene blue, exchange transfusion, and N-acetyl cysteine were used as life-saving treatments. However, further studies in a large patient population are needed to determine the timing and effectiveness of these treatment options. Nationwide bans on high-risk pesticides could reduce the number of suicides in India, as well as occupational poisonings, with minimal impact on farm productivity.

REFERENCES

- Patton TG, Blamer SL, Horak KE. Detecting Methemoglobinemia in animals with a drop of blood. *PLoS One*. 2016;11(12):e0167942.
- Wright RO, Lewander WJ, Woolf AD. Methemoglobinemia: Etiology, pharmacology, and clinical management. *Ann Emerg Med*. 1999;34(5):646-56. Doi: 10.1016/s0196-0644(99)70167-8. PMID: 10533013.
- Lu HC, Shih RD, Marcus S, Ruck B, Jennis T. Pseudomethemoglobinemia: A case report and review of sulfhemoglobinemia. *Arch Pediatr Adolesc Med*. 1998;152(8):803-05.

- [4] Ranasinghe P, Dilrukshi SA, Atukorala I, Katulanda P, Gnanathanan A. Exchange transfusion can be life-saving in severe propanil poisoning: A case report. BMC Res Notes. 2014;7(1):700.
- [5] Roberts DM, Heilmair R, Buckley NA, Dawson AH, Fahim M, Eddleston M, et al. Clinical outcomes and kinetics of propanil following acute self-poisoning: A prospective case series. BMC Clin Pharmacol. 2009;9:3.
- [6] Rittlert P, Sriapha C, Tongpoo A, Pradoo AO, Wananukul W, Trakulsrichai S. Clinical characteristics, treatment and outcomes of acute propanil poisoning in a 7-year retrospective cohort study. Toxicol Rep. 2022;9:1180-88.
- [7] McMillan DC, Leakey JE, Arlotto MP, McMillan JM, Hinson JA. Metabolism of the arylamide herbicide propanil: II. Effects of propanil and its derivatives on hepatic microsomal drug-metabolizing enzymes in the rat. Toxicol Appl Pharmacol. 1990;103(1):102-12.
- [8] Kurukulasuriya AP, Asokan P, Dissanayake HW. Direct oxidant damage to red cells associated with propanil ingestion. Ceylon Med J. 2003;48(3):88-89.
- [9] De Silva WA, Bodinayake CK. Propanil poisoning. Ceylon Med J. 1997;42(2):81-84.
- [10] Woo SH, So BH, Choi KH, Park KN, Lee WJ. Fatal Propanil pesticide poisoning presenting with Methemoglobinemia. J Korean Soc Emerg Med. 2006;17(3):268-72.

PARTICULARS OF CONTRIBUTORS:

1. Senior Medical Officer, Department of Paediatrics, Evangelical Mission Hospital, Tilda, Chattisgarh, India.
2. Director, Department of Orthopaedics, Evangelical Mission Hospital, Tilda, Chattisgarh, India.
3. Junior Medical Officer, Post MBBS, Evangelical Mission Hospital, Tilda, Chattisgarh, India.
4. Junior Medical Officer, Post MBBS, Evangelical Mission Hospital, Tilda, Chattisgarh, India.
5. Junior Medical Officer, Post MBBS, Evangelical Mission Hospital, Tilda, Chattisgarh, India.

NAME, ADDRESS, E-MAIL ID OF THE CORRESPONDING AUTHOR:

Dr. Abhijit Nanda,
Senior Medical Officer, Department of Paediatrics,
Evangelical Mission Hospital, Tilda-493114, Chattisgarh, India.
E-mail: anabhijit7@gmail.com

PLAGIARISM CHECKING METHODS: [\[Lain H et al.\]](#)

- Plagiarism X-checker: Mar 06, 2024
- Manual Googling: Apr 23, 2024
- iThenticate Software: Jun 11, 2024 (11%)

ETYMOLOGY: Author Origin**EMENDATIONS:** 7**AUTHOR DECLARATION:**

- Financial or Other Competing Interests: None
- Was informed consent obtained from the subjects involved in the study? Yes
- For any images presented appropriate consent has been obtained from the subjects. NA

Date of Submission: **Mar 05, 2024**Date of Peer Review: **Apr 18, 2024**Date of Acceptance: **Jun 12, 2024**Date of Publishing: **Aug 01, 2024**