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VASO OCCLUSIVE DISORDER -A CASE REPORT

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ABSTRACT

This narrative review aims to highlight the current paradigm on pain management in sickle cell vaso-occlusive crisis. It specifically examines the pathophysiologic mechanisms of sickle cell pain as well as the pharmacologic and nonpharmacologic methods of pain management. Recurrent painful episodes constitute the major morbidity in sickle cell disease (SCD). Management of disease-related pain should be based on its pathophysiologic mechanisms instead of using recommendations from other non-SCD pain syndromes. Pain management in vaso-occlusive crisis is complex and requires multiple interventions such as pharmacologic, nonpharmacologic, and preventive therapeutic interventions. This clinical case report the management of vaso-occlusive disorder cases that were successfully treated using Drugs.

Keywords: Painful crisis, Vaso-occlusive crisis, Sickle cell

INTRODUCTION:

Vaso-occlusive crisis, often known as an acute painful crisis, is the primary morbidity in SCD. Even in what seems to be a simple painful crisis, fever frequently occurs, indicating that the symptom may be typical of the crisis itself and not always be an indication of infection. The primary cause of this crisis is medication, and with each cycle, pain treatment has increasingly turned its attention away from the solely physical aspect of pain and toward combining psychological, social, and behavioural

elements **[1]**. Thus for patients to receive comprehensive care, a multifaceted approach to pain management is still essential. Understanding the underlying mechanisms is crucial in order to correctly diagnose, forecast, or avoid the start of VOC, as well as its accompanying pain and other consequences. Vasoocclusion and VOC are brought on by chronic vascular inflammation, which activates endothelium and blood cells and triggers the release of macrophages, mast cells, and platelets as well as multicellular adhesion and nociceptors. Vaso-occlusion involves numerous factors.

involves the blockage of tiny blood arteries by sickled red blood cells (RBCs) and adherent blood cells, as well as large-vessel intimal hyperplasia, thrombosis, and bone marrow fat embolization, all of which cause hypoxia, ischemia, tissue damage, and inflammation. SCD pain is distinct because to the interaction of inflammation, ischemia tissue damage, and hypoxia/reperfusion injury **[2]**.

CASE REPORT:

A 12-year-old male patient reported to the IP with the chief complaint of fever (high grade) for the one day and chest pain with body ache for one. Joint pain for one day. Patient is known complain of sickle cell disease admitted earlier SSG Hospital with vaso-occlusive crisis. Patient was admitted to the same hospital for Same disease on date 18 August 2015 and advised with plenty of fluid orally,

- Tab. MVBC BD,
- Tab. Folic acid(5mg) OD,
- Tab. Calcium(250mg) BD,
- Cap. Hydroxy urea (500 mg) OD.

On 15 January 2016 patient was advised with

- Tab. MVBC BD,
- Tab. Folic acid(5mg) OD,
- Tab. Calcium(250mg) BD,
- Cap. Hydroxy urea (500 mg) OD.

On 14 July 2017 patient was advised with

- Tab. MVBC BD,
- Tab. Folic acid (5mg) OD,
- Tab. Ibuprofen (200mg) SOS.

On 20 March 2019 patient was advised with

- Tab. Folic acid (5mg) OD,
- Tab. Ibuprofen (200mg) SOS,
- Hydroxy urea(500mg) OD.

On 27 January 2022, 05 June 2022 and 29 September 2022 patient was advised with

- Tab. Folic acid (5mg) OD,
- Tab. Ibuprofen (200mg) SOS,
- Hydroxyurea(500mg) OD.

On General examination Pallor positive found with temperature 104^0F and blood pressure 115 mmHg systolic with 70 mmHg diastolic observed. SpO2 found 97% at room air In CVS S1 S2 positive with no murmur. RS AEB/L is clear with no added sound. P/A 4 cm palpable. CNS Alert and conscious. Lab parameters were observed

Hb 8.30, Neutrophils 79%, Lymphocytes 17%, MCV 68.2, PCV 23.2%, RBC 3.4 million cell/cm^2, RDWcv 21.9%. Medications were prescribed to the patient Inj. Ceftriaxone 800mg BD for 3 days, Inj. Paracetamol 300mg TDS for first day, Inj. Ibuprofen 300mg TDS for first day, Inj. Ondansetron 4mg TDS for first day, Inj. DNS 85 cc/hr for 3days, Tab. Domperidom 10mg BD for second day, Cap. Hydroxyurea 500mg OD for second and third day. Tab Ibuprofen 300mg TDS for second and third day, Tab Folic acid 5mg OD for third day.

DISCUSSION:

A that comprises giving morphine intravenously (IV) at doses of 5-7.5 mg every four hours for the first 24 hours was used in an observational research. before moving to oral analgesia using paracetamol or NSAIDs, combined with 5 mg of morphine every six hours as needed (NSAIDs). 80% of the SCD cases were successfully handled within 72 hours after admission, according to the findings. When the number of patients discharged was compared to the old protocol employed in the same hospital, 71% of the patients were discharged vs 83% of the patients discharged (following the new protocol), showing a considerably higher percentage of patients discharged after 72 hours of admission (P 0.00) **[3]**. The use of NSAIDs should be tailored to the patient's needs based on risk factors and probable adverse effects, and the lowest effective dose should be provided while ensuring thorough monitoring in SCD patients. NSAIDs have the potential to play a significant role in pain management regimens **[4]**. Hydroxyurea usage is another paradigm for treating acute painful crises **[1]**. Red blood cells with deformed sickle or crescent shapes are a symptom of the group of diseases known as sickle cell disease, which damages haemoglobin. Anaemia, heightened susceptibility to infections, and pain episodes are its hallmarks. The condition is brought on by receiving faulty genes from both parents, and this combination results in the disease's various forms. It is believed that individuals with sickle cell disease are more likely to experience a folate shortage because of their enhanced erythropoiesis. This is why sickle cell disease patients, especially those with sickle cell anaemia, frequently take 1 mg of folic acid orally every day in the hopes that it may replenish folate that has been lost **[5]**. Clinical problems from the imbalance of the calcium/magnesium ratio in sickle cell disease could occur (SCD). Sickling, increased polymerization, and vaso-occlusion (VOC) in sickle cells because to cell dehydration have all been linked to low magnesium levels. Significantly rising amounts of magnesium impede the K-Cl cotransport, which is crucial in sickle cell dehydration. In SCD patients and "healthy"

controls, the study calculated the calcium/magnesium ratio and measured total serum magnesium levels **[6]**.

CONCLUSION:

In sickle cell vaso-occlusive crisis, pharmacologic pain treatment strategies are helpful for both juvenile and adult patients. The mainstay of analgesic therapy for acute and chronic disease-related pain is still opioids and non-opioids. It appears that structured, tailored analgesic regimens and multimodal analgesia are more effective in improving treatment outcomes.

CONFLICT OF INTEREST:

The author declares no conflict of interest.

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