

Chronic Widespread Pain in a Patient with Sickle Cell Disease

Blankson-Darku, Anna Doris¹ Djagbleley Robert^{1,2} deGraft-Johnson Papa Kobina¹ Darso Owusu-Sekyere¹ Tahiru, Zan-nairah¹

¹Korle- Bu Teaching Hospital, Department of Anaesthesia and Critical Care, Accra, Ghana,

²University of Ghana Medical School, Department of Anaesthesia and Critical Care, Accra, Ghana



Introduction

Chronic widespread pain(CWP) is a complex condition presenting significant management challenges, particularly in patients living with chronic diseases in low-middle income countries(LMICs).

Case Description

We report a 27-year-old West African male with sickle cell disease(SCD; HbSS) who presented with severe pain described as persistent, sharp, and "bone-breaking," with neuropathic features. Initially treated for recurrent vaso-occlusive crises, his pain evolved over two years into constant daily pain, with a numeric rating scale(NRS) of 10/10.

Despite previous management with NSAIDs and opioids, symptoms persisted, developing opioid tolerance. Examination revealed a chronically ill, underweight man(51.5kg, 174cm) with diffuse tenderness, no focal neurological deficits, and unremarkable investigations. Based on the 2016 American College of Rheumatology criteria, a diagnosis of CWP secondary to SCD was made, with concern for opioid-induced hyperalgesia.

Management consisted of a novel outpatient multimodal infusion; KLMN(ketamine-0.4mg/kg, lidocaine-1.5mg/kg, magnesium sulphate-50mg/kg, and nefopam 20mg) infused over two hours. Infusions were administered every two weeks combined with oral amitriptyline 25 mg, pregabalin 75 mg twice daily and nefopam 30mg twice daily. After six sessions, his NRS improved to 4/10 with marked functional gains, allowing transition to an oral-only regimen. No adverse events occurred. He also received pain science education and psychotherapy.

Discussion

The KLM infusion alone, has been shown to lower pain scores and reduce opioid-related adverse effects^{1,2}. Ketamine and magnesium sulphate exert synergistic analgesic effects through NMDA receptor modulation^{5,6}, while lidocaine decreases neuronal hyperexcitability across sodium, potassium, calcium, and glutamate pathways^{4,6}. Addition of nefopam provides monoamine neurotransmitter-reuptake inhibition and further NMDA-modulating activity³,strengthening descending inhibition and reducing central sensitisation. This offers a mechanism-based approach to difficult-to-treat chronic pain patients, which in our setting has shown meaningful improvements in pain intensity and function.

Learning Points

The complementary mechanisms of KLMN, together create a practical, opioid-sparing combination particularly suited to LMIC settings where access, cost, and resource limitations shape clinical decision-making.

References

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