MULTIDISCIPLINARY APPROACH TO ACUTE PAIN MANAGEMENT IN THE ADULT SICKLE CELL DISEASE PATIENT

* Eyelade Olayinka R, **Amanor-Boadu Simbo D Department of Anaesthesia College of Medicine, University of Ibadan / University College Hospital,Ibadan, NIGERIA.

ABSTRACT

Pain is the hallmark of sickle cell disease and is a major concern of patients and their families. The anaesthetists as pain specialists are sometimes involved in the management of intractable acutely painful vaso-occlusive crises in these patients.

We present our experiences in these 3 case reports of known haemoglobin S patients in acute painful sickle cell crises. Two of the patients were males aged 32years and 29years and the third patient was a female aged 25years. The male patients required admission into the intensive care unit for close monitoring while on continuous intravenous opioids analgesia. The female patient was successfully managed on the medical ward with parenteral and oral analgesics.

Keywords: Acute pain, sickle cell disease, multidisciplinary management

Introduction

Pain is the hallmark of sickle cell disease and the major concern of patients and their families¹. Pain is also the most common indication for hospital admission in the patient with sickle cell anaemia and therefore the main requirement is adequate pain relief².

Anaesthetists as pain specialists are sometimes involved in the management of painful vaso-occlusive crises in the sickle cell anaemia patients and also play major roles when intensive care unit management is required. Acute pain management in the sickle cell disease (SCD) individuals is often initiated by attending health care professionals including haematologists. However, when critical clinical problems such as intractable pain, acute chest syndrome and overwhelming sepsis supervenes, a multidisciplinary team of healthcare providers is required for expert and effective management. We present three cases managed by a team of physicians including the anaesthetists in the University College Hospital (UCH), Ibadan to illustrate our experiences.

Case 1

LJ, a businessman, is 32 year old known sickle cell anaemia (SCA) patient who was being managed by haematologists at Haematology Day care unit (HDCU), in UCH, Ibadan, and the working diagnosis being acute chest syndrome. The initial treatment consisted of intramuscular (im) pentazocine 30mg 6hourly and (Dihydrocodene 118) tablets 60mg 8hourly. However, substernal chest and lumbar pain remained bad at ¹⁰/₁₀ on the Visual Analogue Scale (VAS), in addition to increasing respiratory distress. Associated symptoms included fever (resolving), poor sleep and loss of appetite. He had earlier been treated for a mild cough at a private hospital a week prior to presentation at HDCU. The substernal pain was described as throbbing and sharp, associated with breathing difficulties. On this account, he was referred to the anaesthetists for ICU care.

Physical Examination by the anaesthetists revealed an acutely ill man, who was moderately dehydrated, pale and in severe pain charted on the Body chart as located in substernal chest and lumbar regions of the body. Heart rate was 95beats/min, blood pressure- 135/90mmHg, respiratory rate 35breaths/minute and arterial oxygen saturation (SaO₂) in room air was 92%. The abdomen was tender with non-tender hepatomegaly. Chest x-ray was unremarkable and the ECG shows sinus tachycardia. He was admitted into ICU.

Treatment in ICU included oxygen therapy (facemask oxygen), rehydration with intravenous fluid and bolus intravenous doses of pethidine 50mg 4hourly titrated to effect. Parenteral antibiotics (Crystalline penicillin and Gentamicin) were also administered. He however developed radiological signs of infection on the chest X-ray while in the ICU and antibiotics were changed to third generation cephalosporin.

The pain and respiratory symptoms improved remarkably over a period of four days in the ICU and he was gradually weaned off oxygen therapy and parenteral analgesic. In the course of admission, Packed Cell Volume (PCV) ranged from 15 to 21%, he received 2units of whole blood transfusion when the PCV dropped from 21 15%. He was in ICU for 5days and then discharged to lying-in ward and home subsequently.

CASE 2: AA, is a 25 year old female undergraduate known SCA patient admitted in bone pain crises precipitated by an attack of malaria. She presented with pain in arms, lower limbs and lumbar area. Initial treatment consisted of intramuscular (im) pethidine 50mg 6hourly, DF118 60mg 8hourly, oral feldene (piroxicam) 200mg 12hourly. All analgesics were administered at the same time (starting at 6am). Pain relief was achieved for between 2- 3hours after analgesic administration. She was referred to anaesthetist on account of intractable pain. Examination by the anaesthetist revealed a young lady in excruciating pain, VAS was 7/10, sharp ache, throbbing and constant. She was crying and was very irritable. Associated symptoms include anorexia and insomnia. Other body system was essentially normal. Review of the nurses' drug chart revealed that analgesics were not given as prescribed for fear of overdose as well as opioid addiction. In the course of admission the PCV range from 22 24%. There was no need for blood transfusion.

Our approach to management of the patient included: Education of the ward nurses' on the need for opioid analgesic in acute severe pain, the prescribed analgesics were to be administer in a staggered manner such that the patient received an analgesic every 2hours unless she was pain free or asleep. Pain scoring was incorporated to the vital signs monitoring chart. Pain was to be assessed before and 1hour after analgesic administration. Pain relief in this

patient improved considerably within 24hours of the above regimen and by the 36th hour, pethidine was discontinued. She had piroxicam and DF118 for another 36hours. She was discharged home on Paracetamol tablets patient improved considerably within 24hours of the above regimen and by the 36th hour, pethidine was discontinued. She had piroxicam and DF118 for another 36hours. She was discharged home on Paracetamol tablets.

CASE 3:AO, is a 29 year old medical officer, known SCA patient admitted into the ward on account of pain crises precipitated by stress of work. He was being managed jointly by the orthopaedic surgeon and haematologists. He was referred to the anaesthetist on the 7th day of admission on account of intractable pain that was not responding to treatment. The initial treatment comprised im pethidine 50mg 6hourly, DF118 60mg 8hourly, and Novalgin tablets 2 8hourly.

On review by the anaesthetist, he was in excruciating pain VAS 7/10 despite analgesics, pain was mainly in the lower limbs, lumbar area and the temporomandibular joint (TMJ). Involvement of the TMJ precludes mouth opening, associated with anorexia, poor sleep, reduced appetite and physical activity, in addition to fear of im injections. Pain relief with initial treatment was 30-40%.

He was admitted into ICU and commenced on oxygen therapy, continuous opioid infusion (iv morphine 2mg/hour). Monitoring included continuous ECG, SaO2, and hourly pain assessment using VAS. At 6hours post admission into ICU, pain score was down to 5/10 from 7/10 and was 3/10 by the 12th hour of commencing iv morphine infusion. The infusion was continued for another 12hours, at which time he was able to open his mouth and was taking oral fluids. Analgesics were then changed to oral piroxicam at 40mg 12hourly.

In the course of admission, PCV range was 18 21% and no blood transfusion was required. He was in ICU for 48hours and subsequently discharged into the lying-in ward and home very happy and quite appreciative.

DISCUSSION

Individuals undergoing sickle cell crises can experience extreme pain that is not always effectively managed. When patients with sickle cell anaemia (SCA) present in the hospital in acute pain, the differential diagnosis would include bone pain crises for obvious reasons, sequestration with anaemia, infection and joint capsular distention among other². The precipitating factor could be malaria infection, stress of day to day living or chest infection⁴ as was seen in the cases presented above. Families and relations would have tried few treatments at home before the patient presents in the hospital, presuming that this could be one of such pain that the persons usually have. On presentation in the hospital the attending health care worker usually would investigates the cause of the pain, treats and this is often followed by resolution of the symptoms.

In the patients presented above, investigations were carried out to determine the cause of the pain and appropriate management administered yet the pain remained a major clinical problem.

Treatment of vaso-occlusive crises pain is aimed at treating the underlying problem and ensuring hydration, blood transfusion may be required as was the case in the first patient presented here⁵.

first patient presented here⁵.

Adequate hydration of the patient is essential due to the fact that the pain is often associated with poor appetite and poor fluid intake, and in Case 3, the involvement of the temporomandibular joint (TMJ) actually precludes mouth opening; this was ensured in all the three patients. More so, that dehydration will precipitate more sickling of the red cell resulting in a vicious cycle which worsens the pain.

Analgesics is the mainstay of therapy, and pain relief is achieved using opioid or non-opioid analgesics⁶. Opioid analgesics, non-steroidal anti-inflammatory drugs (NSAID)s and simple analgesics such as Paracetamol can be used singly or combined as illustrated in Case 2 above. These analgesics would obtund the pain transmission and counteract the inflammatory response (NSAID) that may be associated with the vaso-occlusive crises. These analgesics can be administered through oral or parenteral route as deem appropriate. The use of Patient Controlled Analgesia or continuous infusion of opioids had been noted to provide effective and better analgesia, less delay in providing analgesia and effective use of nursing time⁷. Anxiolytic such as diazepam would help to relax the patient and enhance the sleep pattern of the patient, especially when there is associated insomnia as seen in case 1 and 3.

SCA pain is often poorly managed because of ignorance, inexperience, tradition and overwork on the part of health care professionals⁷, in addition fear of addiction if opioids were to be used is also a contributing factor as illustrated in case 2. Therefore, there is need for regular education of health care professionals on pain management.

The acute chest syndrome (ACS) as seen in case 1 is one of the most frequent causes of hospitalization in SCD⁸. ACS is a complication of SCD characterized by chest pain, fever, rales on lung auscultation and pulmonary infiltrates on chest X-ray⁹. The initial chest sign were absent in case 1 but later became obvious in the course of treatment. It could be caused bacteria or could be as a result of thromboembolic process. Fever is also a common presenting symptoms¹⁰, though this was not noted in the patients presented above, this may be due to the fact that antimalaria treatment and antibiotics had earlier been used before presentation in the hospital.

It is of particular importance to appreciate that stress of daily living could precipitate painful crises and so adequate rest and relaxation is recommended for the SCA patient. Early management of impending complications is essential as well as adequate nutrition, psychotherapy, physiotherapy and genetic counseling.

In conclusion, the quality of analgesia given to the SCA patient in acute painful crisis can be improved with the provision of acute pain teams, multidisciplinary care and regular education of health care professional on pain management.

REFERENCES:

- 1. Ballas SK: Sickle Cell Pain in Progress in Pain Research and Management vol 11 chapter 3-4: IASP Press 1998, Seattle, USA.
- Kotila TR. Management of acute painful crises in sickle cell disease. Clin Lab Haem 2005; 27: 221 223.
- Vijay V, Cavenagh JD and Yate P. The anaesthetist's role in acute sickle cell crisis. BJA 1998; 80: 820 828.
- 4. Vichinsky EP, Neumayr LD, Earles AN, Williams R, Lennette ET,et al. Causes and outcomes of the acute chest syndrome in sickle cell disease. NEJM 2000; 342: 1855-1865.
- 5. Adedeji MO: Complications of sickle cell disease in Benin City, Nigeria. East Afr Med J 1988; 65: 3 7.

- 6. Okpala I and Tawil A: Management of pain in sickle cell disease. Journal of the Royal Society of Medicine 2002; 95: 456 458.
- 7. Justins D, Richardson PJ: Clinical management of acute pain. Br Med Bull 1991; 47: 561 563.
- 8. Castro O, Brambilla DJ, Thorington B, Reindorf CA, Scott RB, Gillette P et al: The acute chest syndrome in sickle cell disease: incidence and risk factors. Blood 1994; 84: 643–649.
- 9. Taylor C, Carter F, Poulose J, Rolle S, Babu S, Crichlow S. Clinical presentation of acute chest syndrome in sickle cell disease. Postgrad med J 2004; 80 (944): 346 349.
- 10. Ibidapo MO and Akinyanju OO. Acute Sickle Cell Sydromes in Nigerian adults. Clin Lab Haem 2000; 22 (3): 151 155.