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Managing the Pregnant Woman with Sickle Cell Disease

—A Practical Guide for the Obstetrician

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Abstract

Sickle cell disease is a multi-systemic, chronic condition characterized by acute episodes (referred to as crisis), persistent anaemia, organ infarction/damage and a shortened lifespan. It is the most common inherited blood disorder in humans and affects millions of people worldwide. Pregnancy in sickle cell patients is fraught with several complications and often results in increased perinatal morbidity and occasionally maternal mortality. Managing a pregnant woman with sickle cell disease can be very challenging. The following is a short update and guide for the busy, practising Obstetrician in a District General Hospital setting who may be faced with such a challenge.

Keywords

SCD-Sickle Cell Disease, BUE & CR-Blood Urea, Electrolytes and Creatinine, LFT-Liver Function Tests, CTPA-Computerized Pulmonary Angiogram, FAS-Foetal Anomaly Scan, ACS-Acute Chest Syndrome RCOG VTE Score-Royal College of Obstetricians/Gynaecologists Thromboembolism Score, MEOWS-Modified Enhanced Warning Score, MDT-Multidisciplinary Team, NSAIDS-Nonsteroidal Anti-Inflammatory Drugs

1. Introduction

Sickle cell disease is one of the most common inherited genetic disorders in the world. It affects more than 6.4 million people worldwide [1]. The disease was first described by JB Herrick in 1910 as a case report in a patient from Grenada. The disease is most prevalent in Sub-Saharan Africa with approximately 75% - 80% of cases occurring in individuals with a background from that region [2]. The "protective effect" and survival advantage of individuals heterozygote for sickle cell

against malaria is thought to contribute to the increased prevalence of the disease in malaria endemic regions. Due to demographic shifts and recent migration trends, the disease now affects people all over the world. In the UK, it is estimated that there are 13,000 people affected and approximately 300 infants born with the condition each year. Advances in the management of patients with haemoglobinopathies have led to a marked improvement in the life expectancy and quality of life of these patients. This has led to a progressive increase in women with SCD who get to childbearing age themselves and express the desire to fall pregnant.

Pregnancy in patients with SCD is often complicated, with several adverse maternal and perinatal outcomes [3]. The chronic and multisystemic nature of the disease creates unique challenges for its management and makes multidisciplinary care essential.

This article is a short update and practical guide for the Obstetrician.

2. Genetic Background/Aetiology

Sickle cell disease (SCD) is a genetic disorder caused by a single point mutation. The disease is caused by a mutation in the haemoglobin subunit beta gene located on the short arm of chromosome 11. A single base mutation (GAT-GTT) in the 6^{th} codon of exon −1 of this chromosome results in the replacement of the hydrophilic glutamic acid with hydrophobic valine at position 6 of the beta globin chain. In a deoxygenated state, the resulting abnormal haemoglobin (HBS), tends to polymerize, attach to each other and form stiff fibres. These fibres cause the erythrocyte to develop the biconcave, sickle shape characteristic of the disease. The disease is inherited in a Mendelian autosomal codominant manner with individuals homozygous for the B(S) allele having the disorder. Individuals who inherit the 2 BS alleles (HBSS) have the most severe from of the disease. Inheritance of a combination of one BS allele and another abnormal allele leads to other SCD genotypes e.g. HBSC, HBS B0thal, HBSB + thal, etc. These other SCD genotypes generally result in milder forms of the disease. HBSC disease is particularly noted for its association with osteonecrosis and retinopathy. Individuals who inherit one normal allele in combination with one BS allele *i.e.* HBAS, are referred to as having Sickle cell trait. They do not have SCD.

3. Pathophysiology

The pathophysiology of SCD is complex and multisystemic. Every major system of a patient with SCD (cardiovascular, respiratory, renal, etc.) can potentially be affected by the disease. The pathological hallmarks of the disease include vasculopathy, chronic haemolysis, a pro-inflammatory state and chronic organ damage.

When deoxygenated, haemoglobin S is converted into a gel-like substance called "tactoids". Erythrocytes containing HBS assume the characteristic biconcave sickled shape. Sickled erythrocytes are highly adhesive. These cells interact with vascular endothelium and, through complex inflammatory mechanisms, activate neutrophils and platelets. Activated platelets form aggregates with erythrocytes, neu-

trophils and monocytes. Endothelial dysfunction occurs as a result of the excessive adhesion of sickled erythrocytes to vascular endothelium. This process (in combination with the chronic haemolysis and reticulocytosis that occurs), results in vaso-oclussion in the microcirculation with consequent chronic ischaemic/hypoxic damage to organs downstream.

SCD causes erythrocytes to have a short half-life when compared to normal erythrocytes. Chronic haemolysis, a central feature of the disease, occurs persistently. Haemolysis and the release of cell-free haemoglobin on a continuous basis depletes hemopexin and haptoglobin stores and results in a reduced bioavailability of Nitric oxide. Free haemoglobin essentially acts as a Nitric oxide scavenger. The ability of sickled erythrocytes to produce nitric oxide to replace this deficit is impaired because of abnormal intracellular signalling pathways. The resulting Nitric oxide deficiency further enhances endothelial dysfunction.

Sickled shape cells, together with chronic haemolysis (and the resulting reticulocytosis) increases blood viscosity, thus increasing the risk of thromboembolism.

4. Clinical Features

SCD is characterized by a background of haemolytic anaemia and chronic low-level pain (mainly in the bones and joints) punctuated with acute exacerbations/complications of the disease referred to as crisis. Below are some of the common clinical manifestations/complications of the disease.

4.1. Vaso-Occlusive Crisis (VOC)/Pain Crisis

This is one of the main complications of the disease and is the principal reason for hospital admission for many patients in the UK [4]. VOC is caused by microvascular occlusion, which (as indicated above) is pathognomonic of the disease. The tissue inflammation, which occurs because of this occlusion, is worsened by the process of reperfusion. Inflammatory mediators which activate nociceptors are then produced resulting in excruciating pain in the part of the body affected. The long bones, back, pelvis and chest are often affected. In children, this process may result in pain and swelling of the hands and feet, a condition referred to as Dactylitis. In an investigation by the Cooperative study of Sickle cell disease (CSSCD), of 3578 patients with SCD, there were 12,290 episodes of pain crises in 18,356 patient years. This reflects the frequency and importance of this complication among SCD patients [5].

4.2. Acute Chest Syndrome (ACS)

This complication is second in frequency only to Vaso-occlusive/pain crises. It is characterized by fever, respiratory symptoms and the presence of pulmonary infiltrates on chest X-ray. Respiratory symptoms include cough, chest pain, dyspnoea, tachypnoea, wheezing, intercostal recession, nasal flaring and haemoptysis. ACS is thought to be caused by occlusion in the pulmonary microvasculature. It occurs as a result of excessive adhesions of sickled erythrocytes to pulmonary vas-

cular endothelium, inflammation and activation of coagulation factors. Nitric oxide deficiency, a persistent feature of Sickle cell crisis, exacerbates the resulting endothelial dysfunction. Physical examination may reveal tachypnoea, reduced air entry and crepitations on auscultation of the chest. Untreated, this complication can lead to hypoxemia and eventually respiratory failure.

4.3. Genitourinary Complications

Hypoxia in the renal medulla of patients with SCD leads to the formation of sickled erythrocytes in the kidney vasculature, microvascular occlusion and consequent infarction. This sequence of events has been described as "Sickle cell nephropathy". The clinical manifestations of sickle cell nephropathy include proteinuria, microalbuminuria, painless haematuria and ultimately the development of chronic renal failure.

4.4. Neurological Complications

SCD increases the risk of cerebrovascular accidents (CVA). It is the commonest cause of strokes in children. CVA may occur as a result of either cerebral infarction or intracerebral haemorrhage. Asymptomatic, silent cerebral infarcts are commoner than overt strokes. Although asymptomatic, frequent repetitive silent cerebral infarcts can eventually lead to neurocognitive impairment. Patients with SCD may also suffer from episodes of acute or chronic headache.

4.5. Infections

SCD compromises the immune status of a patient and leads to an increased incidence of infections. The increased incidence of infection is thought to be a result of the combination of defective splenic function, altered complement activation, impaired leukocyte function and opsonization. Infections with encapsulated organisms are common. Pregnant patients are particularly at risk of recurrent urinary tract infections.

4.6. Splenic/Hepatobiliary Complications

Chronic haemolysis in patients with SCD often leads to an increased turnover of bilirubin and consequently, cholelithiasis. Together with Ischaemia and sequestration, this can lead to the formation of biliary sludge, and in extreme case liver failure.

Blood pooling in the spleen (Splenic sequestration), can lead to an abrupt drop in the haematocrit, a sudden onset of abdominal pain, splenomegaly and hypovolemic shock. Splenic sequestrations is a life-threatening complication of SCD.

5. Sickle Cell Disease and Pregnancy

5.1. Effects of SCD on the Fetus

It is a well-known fact that pregnant women with SCD have a greater risk of developing fetal complications when compared to women with a normal genotype

[5]. This risk is believed to be a result of abnormal placentation. In a case control study by Mohammed Mumuni *et al.* of the histomorphology of the placentae of pregnant women with SCD, the number of aggregates of syncytiotrophoblast nuclei creating syncytial knots was substantially higher in SCD women when compared to controls. It has been hypothesized that the vascular occlusion and congestion which is characteristic of SCD leads to a chronic hypoxic state of the placenta with compensatory increase in the formation of foetal capillaries in the terminal villi through a process of angiogenesis [6]. Increased syncytial knots, increased density of foetal capillaries and syncytial denudation may indicate a chronic hypoxic placental environment and explain the increased adverse consequences for the foetus.

SCD leads to an increased incidence of intrauterine growth restriction (IUGR), Preterm delivery and the delivery of an SGA (Small for gestational age baby). Studies have also shown an increased risk of intrauterine fetal death (IUFD) in patients with Sickle cell (SS) disease [7].

5.2. Effects of SCD on the Pregnant Mother

The physiological expansion of plasma volume in pregnancy often causes a dilutional anaemia. In SCD patients, anaemia may be much more pronounced because of the concomitant occurrence of chronic haemolysis, an essential feature of the disease. Patients may require several antenatal blood transfusions in severe cases.

A study in the UK showed an increased risk of SCD complications (e.g. Vaso-Occlusive crisis and Acute chest syndrome) during pregnancy. These acute episodes/complications may result in multiple hospital admissions. The incidence of non-sickle cell disease pregnancy complications, e.g., hypertension, Venous thromboembolism and infections may also be increased. Infections of the urinary tract are particularly common. Chest infections may occur and can be difficult to differentiate from ACS. Several studies have indicated a higher rate of caesarean sections in SCD (SS) patients. In low-income countries, the maternal mortality rate is also increased.

6. Management

6.1. Preconception Care

Pre-conception care is very important in women of childbearing age who have intercurrent medical disease. For the patient with SCD, pre-conception care (where available) is essential. In the United Kingdom, a few Pre-conception clinics have now been set up for the purpose of caring for such patients. For the potentially affected infant (*i.e.*, where a partner of an SCD pregnant patient is also a carrier or affected by SCD), the principles of non-intervention, Pre-implantation genetic diagnosis (PGID) and antenatal diagnosis should be discussed at the Pre-conception visit. The current state of the disease should be assessed. Baseline renal, cardiac and pulmonary function should be checked. It is generally important to embark on pregnancy when the disease has been optimized. Teratogenic agents used in the

management of the disease should be discontinued before conception. Appropriate contraception should be advised until disease optimization has been obtained.

6.2. Antenatal Management

Initial/Booking Visit

As with all other medical disorders in pregnancy, multidisciplinary care is mandatory. For SCD, the minimum team should consist of an Obstetrician with an interest in medical disorders, a haematologist, specialist screening midwives and anaesthetists. Constant communication between these professionals is key for patient safety and appropriate management. For the Obstetrician, the initial meeting/booking visit is crucial as it would be the first opportunity to assess the state/severity of the patient's disease. Many patients would not have had access to preconceptual care, and it is essential that any pre-existing organ damage is noted at the beginning of the pregnancy. The renal, cardiovascular and respiratory systems, as well as liver function should all be assessed. Blood urea, electrolytes and creatinine should be checked. Urine PCR, to check for microalbuminuria should be obtained. Baseline Liver function test (LFT), a full blood count (FBC) and antibody screen should be performed. Doppler echocardiography is a useful non-invasive tool for identifying the risk of pulmonary hypertension and ventricular diastolic dysfunction. A raised tricuspid regurgitation jet velocity is associated with an increased risk of pulmonary hypertension and mortality. In addition to these specific tests, routine antenatal serological tests should also be done. An early dating ultrasound scan is important. Assessment of placental function with uterine artery doppler studies is useful [8].

For the "at-risk infant" (*i.e.* where the partner of the pregnant woman is either a carrier or an SCD patient himself), antenatal diagnostic testing should be discussed. Chorionic villus sampling (CVS), typically performed between 10 - 13 weeks gestation provides early diagnosis of an affected fetus so that if the couple wishes termination of pregnancy (TOP), a first trimester TOP can be offered. Amniocentesis for SCD testing of the fetus can also be performed at 15 weeks of gestation, where CVS is not available. Both CVS and amniocentesis have a very small risk of fetal loss and appropriate counselling by trained experts is important before either test is carried out. Detection of cell-free fetal DNA in maternal plasma provides a non-invasive test for diagnosing the affected fetus. This test is, however, currently not available routinely for most NHS hospitals in the UK.

For all SCD patients, folic acid supplementation is recommended. 5 mg of folic acid daily should be prescribed even before conception. This treats the folic acid deficiency (common in SCD patients) and prevents neural tube defects in the fetus. Vitamin D supplementation (and monitoring) is recommended. As discussed above, SCD patients are prone to infections (particularly with encapsulated organisms) and daily penicillin prophylaxis throughout the entire pregnancy is advised. Oral Phenoxymethylpenicillin (Penicillin V) is usually used for this purpose when there is no history of penicillin allergy. SCD patients have an increased risk

of Pre-eclampsia and 150 mg of aspirin daily from 12 weeks of gestation is recommended.

Low-dose Aspirin (150 mg daily), commenced before 12 weeks of gestation reduces the incidence of Pre-eclampsia.

Aspirin should be discontinued at 36 weeks in these patients because of the potential risk of bleeding post-partum (PPH). A VTE (Venous thromboembolism) assessment should be undertaken at booking and repeated each time the patient is admitted into hospital. In a recent meta-analysis, the risk of VTE was increased 3.5-fold in SCD patients with complications of VOC and ACS. Most of the thromboembolic episodes occurred in the postnatal period. SCD confers an RCOG VTE score of 3 on a pregnant woman [9]. Consideration should be given to administering LWMH from 28 weeks of gestation until 6 weeks post-partum. When other risk factors are present, LMWH may be commenced earlier.

The patient's vaccination history should be reviewed. Whilst live attenuated vaccines should be deferred until the post-natal period, all other vaccinations should be kept up to date. In the UK, the administration of Flu, RSV and Covid vaccination are routine for all pregnant women. In the SCD patient, pneumococcal vaccination is also recommended.

A complete history should be obtained with emphasis on the frequency of crises, blood transfusions, medication history and the presence of other co-morbidities. Many of the medications used to reduce the frequency of crises and severity of complications (Hydroxycarbamide, ACE inhibitors, ARB's, iron chelators, Voxelotor, crizanlizumab) are contraindicated in pregnancy. Indeed, Hydroxycarbamide, the commonest medication used in the UK to reduce the frequency of SCD crises has been found to be teratogenic in animals and its usage must be discontinued in the pre-conception period. Ferrous sulphate should not be prescribed empirically but only used when serum investigations reveal iron deficiency.

The chronic haemolytic state of Sickle Cell Disease predisposes patients with the condition to iron overload if ferrous sulphate is administered empirically without any evidence of iron deficiency.

Subsequent Antenatal Visits

In a District General Hospital (DGH) setting, care of the patient should be largely shared by an Obstetrician with a special interest in medical disorders/specialist screening midwives at the Antenatal clinic, a haematologist at the Haematology clinic and community midwives. Monthly antenatal clinic appointments are recommended. In a large teaching hospital, where Obstetric/Haematology clinics exist, appointments may be arranged at this clinic to reduce the number of patient hospital visits.

At each appointment, blood pressure and urinalysis should be checked. Patients should be educated on measures which would decrease the frequency of SCD crises e.g., avoiding extremes of temperatures and dehydration. Routine work duties/environment may have to be modified to reduce the physical stress that certain work patterns create. At each visit, it is important to check that the patient is compliant with all her medications (folic acid, vitamin D, antibiotic prophylaxes, etc).

A mid-trimester FAS scan should be performed between 18 and 21-weeks' gestation. Uterine artery doppler studies (UTAD) may be performed concurrently during the FAS scan to detect those foetuses that may require increased surveillance with more frequent Obstetric growth scans. SCD increases the risk of FGR (Fetal growth restriction), SGA (Small for gestational age foetus) and IUFD (Intra-uterine fetal death). Monthly 3rd trimester Obstetric growth surveillance scans starting from 28 weeks of gestation are mandatory. Where UTAD studies show increased resistance in the uterine vessels at the mid-trimester FAS scan, more frequent Obstetric surveillance scans starting from 24 weeks may be necessary.

Antenatal blood transfusions in patients with SCD generally improve the outcomes for both the patient and the foetus by increasing the serum concentration of HBA in the mother (and thus increasing oxygen supply to the foetus), and decreasing the concentration of HBS, thereby reducing the frequency of sickle crisis in the mother.

There are 2 main approaches to antenatal blood transfusion for the SCD patient in pregnancy:

a) The standard approach.

b) Prophylactic transfusions approach.

In the standard approach, blood transfusion is administered when there is a clinical requirement or indication, e.g., when there is acute anaemia or as part of the treatment of a sickle crisis. With the Prophylactic transfusion approach, blood transfusion is administered prophylactically at intervals throughout the pregnancy (*i.e.*, even when there is no specific clinical indication). Prophylactic transfusions over the course of the pregnancy may reduce the frequency of Vaso-occlusive crisis (VOC), acute anaemic episodes, and maternal mortality. It may also improve perinatal outcome by increasing the availability of well-oxygenated blood to the fetus via the placenta. Repetitive transfusions, however, expose the patient to the risk of acute transfusion reactions and alloimmunization. There is currently insufficient evidence to recommend Prophylactic transfusions over standard care [10]. Ultimately, the approach adopted for each patient should be individualized. A final decision on the blood transfusion approach for the pregnancy should be made after a comprehensive discussion of the pros and cons of each intervention (by the haematologist and Obstetrician) with the patient, applying the principle of shared decision making.

At the 36-week antenatal appointment, a plan should be made for the mode and timing of delivery. For patients who have neither maternal nor fetal complications, Induction of labour is advised between 39 and 40-weeks' gestation. Caesarean section should only be reserved for Obstetric indications. SCD is not an indication for a caesarean section.

Managing Acute SCD Crisis during the Antenatal Period VOC (Vaso-occlusive Crisis)

Pregnancy increases the frequency of Acute pain crisis in patients with SCD. Several reasons may account for this increased frequency. Hyperemesis in early

pregnancy may lead to dehydration and precipitate a VOC. The increased physical stress, physiological anaemia, infections (particularly UTI's) and the general prothrombotic state of pregnancy may all be contributary factors. At the beginning of the pregnancy, the MDT should put a plan in place with instructions and advice on how any Sickle Cell crisis should be managed during the pregnancy.

Although mild pain in SCD patients may be manged in the community, there should be a low threshold for referral to Secondary care (hospital-based care) for patients who complain of significant pain unresponsive to simple analgesics. When an SCD patient is admitted into hospital with VOC (or indeed any Sickle cell crisis), all members of the MDT should be informed. The ward onto which the patient is admitted will depend on gestation, severity of illness and hospital facilities. This may be the haematology ward, the Obstetric ward or the ICU. Baseline investigations on admission include FBC, BUE and CR, LFT and Urinalysis. When chest symptoms are prominent, a chest X-ray and a CTPA may also be required. Blood cultures may be needed when there is pyrexia. Regular observations (blood pressure, pulse, respiratory rate, pulse oximetry) should be recorded on a MEOWS chart (Modified Enhanced Obstetric warning score chart). Regular computerized CTG at least twice a day for patients more than 26 weeks pregnant is mandatory. Principles of management of VOC in pregnancy are:

A) Appropriate/Adequate Analgesia
B) Fluid management
C) Thromboprophylaxis

Regular simple analgesics like paracetamol are the first line of treatment for pain. When symptoms persist, dihydrocodeine and stronger opiates like morphine, diamorphine and oromorph may be administered. NSAIDS are contraindicated after 31 weeks of gestation as they can cause premature closure of the Ductus arteriosus. They may be administered with caution in the first trimester.

Careful fluid management is essential. A fluid input/output chart must be commenced and reviewed on a regular basis to ensure dehydration does not occur. In patients who have Pre-eclampsia, it is also important to ensure fluid overload does not occur.

Thromboprophylaxis with LMWH should be commenced immediately for patients who are not already on heparin prophylaxis.

ACS

Acute chest syndrome (characterized by fever, chest symptoms and a pulmonary infiltrate on chest X-ray) is potentially a fatal complication in a pregnant patient with Sickle cell disease and must be treated very seriously. It affects about 10% of pregnant patients with SCD. When accompanied by significant hypoxia (with oxygen saturation persistently below 95%), the patient is best managed in the ICU or an HDU with significant input from the critical care team. In addition to all the basic investigations done for VOC, a CTPA to rule out the differential diagnoses of a Pulmonary embolus is essential. Blood, sputum and nasopharyngeal aspirate cultures should be requested. Prophylactic antibiotics are recommended even when these cultures are negative. Oxygen administration, initially

via face mask, incentive spirometry and in more severe cases, invasive ventilation may be necessary. Blood transfusion (simple top-up transfusion or exchange transfusion as guided by the haematologist) may be required in cases where hypoxia is a persistent feature [11].

Other less frequent Sickle cell disease crisis e.g., aplastic crisis (often caused by Parvovirus and associated with acute anaemia and reticulocytopenia), splenic sequestration and neurological complications (like Acute stroke) may occur during pregnancy and will all need specialist input.

7. Intrapartum Care

In the absence of Obstetric or SCD complications, induction of labour is usually recommended between 38 and 40-weeks' gestation. The risk of unexplained intrauterine fetal death (IUFD) and the unpredictable occurrence of Sickle crisis (which are more frequent in the 3rd trimester) make a post term induction of labour approach unwise. Labour should take place in a hospital capable of dealing with the intrapartum complications of SCD.

When a patient with SCD is admitted to the delivery suite in labour, the senior co-ordinating midwife, consultant Obstetrician, anaesthetist and haematologist should all be alerted.

A Full blood count (FBC) and group/save should be obtained. IV access should be secured. Regular physical observations (blood pressure, pulse, temperature, respiratory rate, oxygen saturation via pulse oximetry) should be plotted on a MEOWS chart. FGR (Fetal growth restriction) and subsequent intrapartum fetal distress are common complications in patients with SCD; therefore, intrapartum continuous electronic fetal heart rate monitoring is mandatory. A long, protracted labour can precipitate a sickle cell crisis and should be avoided. A fluid input/output chart should be commenced on admission. Oral fluids may be encouraged. Dehydration should be avoided as it can precipitate a Sickle crisis. IV (Intravenous) fluids should be administered to maintain fluid balance when oral fluids cannot be tolerated. If oxygen saturation is persistently below 94%, oxygen should be administered via a face mask.

Adequate pain relief is important. Tolerance to the use of opiates for the management of pain crises (VOC) in many SCD patients may lead to the requirement of higher doses of opiates in labour. Epidural analgesia is a safe, suitable alternative for intrapartum analgesia. It provides effective analgesia in labour and can also be "topped up" to provide anaesthesia for a caesarean section if one is needed as an emergency. For all intra-partum operative interventions, Neuraxial anaesthetic techniques are safer than general anaesthesia, which poses additional risks to the SCD patient. The 3rd stage of labour should be actively managed to reduce the incidence of post-partum haemorrhage. Any vaginal tears should be managed by senior Obstetricians for the same reason.

8. Post-Natal Management

Vigilance must be maintained during the initial post-partum period as the physi-

cal stress of labour can precipitate a sickle cell crisis. Patients who have had a caesarean section are more vulnerable. Every effort should be made to prevent a post-caesarean section Sickle crisis. A repeat FBC BUE/CR, and LFT should be requested as a minimum. Prevention of dehydration and attention to fluid management are important. Adequate analgesia must be maintained. NSAIDS can be used during this period. Strong opiates are transferred to breast milk and could potentially lead to sedation in the breastfed infant. They should be avoided in the breastfeeding mother. Dihydrocodeine and tramadol are suitable alternatives. Hydroxycarbamide (Hydroxyurea), which is the commonest agent used in the UK to prevent sickle crisis in SCD patients is also not advised in breast feeding mothers as it is transferred to breast milk. Thromboprophylaxis for the first 6 weeks of the post-natal period with low molecular weight heparin is usually recommended regardless of mode of delivery. When there are additional risk factors for thromboembolism, this period of thromboprophylaxis may be extended.

Unplanned pregnancies pose a significant risk to SCD patients; hence, it is essential that a robust plan for contraception is made before the patient is discharged. The combined oral contraceptive pill can be used by patients with SCD [12]. A retrospective study of SCD patients on the combined pill did not show a significant difference in thromboembolic events between controls and SCD patients. Progesterone-only contraceptives are particularly useful in SCD patients as they seem to reduce the frequency of sickle crises. The injectable progesterone contraceptive (Depo-Provera) often results in amenorrhea, prevents a drop in the HB as a consequence of heavy menstrual bleeding and thus improves laboratory parameters in these patients. LARCS (Long-acting contraceptive agents, e.g., Mirena IUCD, Implanon, Depo-Provera) have the added benefit of providing effective contraception without the need for daily oral medication. Before the patient is discharged, it is important to ensure that all the relevant outpatient appointments with the GP and haematologists have been arranged.

9. Conclusion

Maternal morbidity and mortality rates are increased in pregnant women with SCD. Pregnancy in a woman with SCD must always be considered a high-risk pregnancy. Global travel, recent migration trends, changes in demographics and better healthcare have led to a situation where women of childbearing age with SCD now live and work on all continents of the world [13]. Universally, Obstetricians are therefore occasionally faced with the challenge of managing the pregnancies of these women. There are only a few large randomized controlled trials on pregnancy outcomes in patients with Sickle cell disease. Guidance on management of these patients is therefore often largely based on expert opinion alone. This makes the concept of multidisciplinary care in these patients even more pertinent. It is imperative that Obstetricians, who are essentially the primary carers of pregnant women, engage and familiarize themselves with new developments in the treatment of SCD. This will equip them with the appropriate knowledge and

confidence required for managing these patients. It would also help them make meaningful contributions to the creation of appropriate guidelines for the treatment of pregnant patients with SCD.

Conflicts of Interest

The author declares no conflicts of interest regarding the publication of this paper.

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