Treating Sickle Cell Disease in Africa

Benta Achieng[1] Elton Akpabio[2] Joseph Ndebo Balikwisha[3] Monica Atieno Omondi[1] Yuanita Hongo[1] Peter Chappell[4] Harry van der Zee[5]

[1] Kenya
[2] Nigeria
[3] Democratic Republic of Congo
[4] United Kingdom
[5] The Netherlands

Address for correspondence Harry van der Zee, MD, P.O. Box 68, 9750 AB Haren, The Netherlands (e-mail: harry@homeolinks.nl).

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Summary

Sickle cell disease (SCD) is a condition causing lots of complications and suffering. In the past decades, treatment modalities have been developed that increase life expectancy and the quality of life of SCD patients. In Africa, these are not available, resulting in SCD patients to die young after many hospitalisations for serious and often painful crises. A new homeopathy-based remedy for SCD has been used with good results since 2013.

Keywords

- Sickle cell disease
- Sickle cell anaemia
- Source resonances
- SR1436n/PC1436n

Evans by Harry van der Zee

In April 2013, I received an email message from a concerned mother in Kenya. It was about her 3-year-old son Evans. He was born with sickle cell disease (SCD), which caused him a lot of suffering. Besides his lack of stamina due to low levels of red blood cells, he suffered from pains and swelling of his legs, which made him cry a lot. Your child suffering from horrible pains while there is nothing you can do is a nightmare for every parent. It renders you powerless and desperate. With SCD, crises of acute pain can occur on a weekly basis. So, little Evens and his mum were having a very hard time. One of the Kenyan volunteers I was working with had referred Evans' mum to me.

But, I was not in Africa at the time, so what was to be done?

Disease-Specific Remedies for Africa

Let me give a short history of why I could have been in Africa? Since 2004 I have been witnessing the amazing results in AIDS patients with a remedy called PC1,[1] I have been travelling to Africa regularly to treat epidemic diseases and relieve trauma. Based on the work that Peter Chappell started in Ethiopia in 2001, several protocols have been developed based on the disease-specific homeopathic resonances that were received by Peter.[2] In 2007, this resulted in the founding of the Amma Resonance Healing Foundation (ARHF; see www.arhf.nl). The treatment of HIV/AIDS, the treatment and prevention of malaria,[3] and the relief of (collective) trauma have been the main targets of ARHF. The scope of the training and treatment ARHF makes available in Africa is much wider though. Dozens of these so-called PC Remedies are being used by hundreds of Africans that have been trained by ARHF volunteers throughout the years. Conditions like AIDS, malaria, and also TB, hepatitis, gonorrhoea, syphilis et cetera are treated successfully and are showing reliable results. Source Medicine makes Source Resonances available to ARHF at cost price and recently changed their names from PC Remedies into Source Resonances (see www.sourcemedicine.zone). In Africa they are still being called PCs. Besides the PCs for epidemic and infectious diseases, and those for trauma (e.g. war and genocide, rape, natural catastrophe), PC Remedies for chronic diseases have also been developed and used with success (e.g. diabetes and hypertension).

And as it goes, there are always patients presenting new conditions for which treatment is asked. Little Evans was one of them.

Back to Evans

As sickle cell anaemia is related to a history of malaria in the ancestors, his treatment started with PC240m, the general malaria resonance. It was available near where Evans lived, so he could be started with that immediately. In the meantime, a new PC resonance for SCD was made by Peter

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Fig. 1 Evans and his mum on his first day going to school (July 2013).

Chappell, further prepared by Hahnemann Pharmacy in the Netherlands and mailed to Evans' mum with urgency.

In July 2013, his mother writes: 'Baby Evans has been so good; good appetite and at least there is an improvement because from his eyes I can see he has not too low blood like he used to have' (►**Fig. 1**).

On the first day of 2014, his mother sends a picture of Evans (►**Fig. 2**) and says, 'Evans is doing very fine'. His pains have subsided and his haemoglobin that could be as low as 3.2 g/dL has gone up to a completely normal 9.2 g/dL.

From very regular acute crises involving a lot of suffering, to hardly any pains and normal haemoglobin is a dramatic improvement and a relief for the mother and the child.

What is sickle cell disease?

Fig. 2 Evans at Christmas 2013.

Sickle Cell Disease and Sickle Cell Anaemia

The term *sickle cell disease* describes a group of inherited red blood cell disorders. People with SCD have abnormal haemoglobin, called *haemoglobin S or* sickle haemoglobin, in their red blood cells.

People who have SCD inherit two abnormal haemoglobin genes, one from each parent. In all forms of SCD, at least one of the two abnormal genes causes a person's body to make haemoglobin S. When a person has two haemoglobin S genes, haemoglobin SS, the disease is called *sickle cell anaemia*. This is the most common and often most severe kind of SCD.

Haemoglobin SC disease and haemoglobin Sβ thalassemia are two other common forms of SCD.

Sickle haemoglobin can form stiff rods within the red cell, changing the normal disc shape into a crescent, or *sickle* shape.

Sickle-shaped cells are not flexible and can stick to the walls of small blood vessels, causing a blockage that slows or stops the flow of blood. When this happens, oxygen cannot reach nearby tissues (►**Fig. 3**).

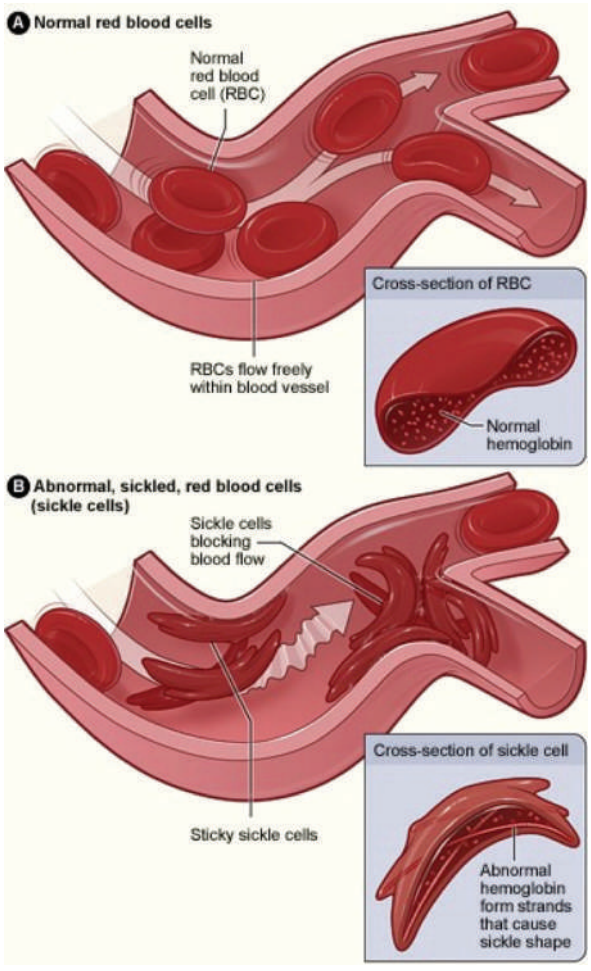

Fig. 3 Form and blood flow of normal red blood cells (**A**) and sickle cells (**B**) (www.nhlbi.nih.gov).

The lack of tissue oxygen can cause attacks of sudden, severe pain. These pain crises can occur without warning, and a person often needs to go to the hospital for effective treatment.

Most children with SCD are pain free between painful crises, but adolescents and adults may also suffer with chronic pain.

The red cell sickling and poor oxygen delivery can cause organ damage to the spleen, brain, eyes, lungs, liver, heart, kidneys, penis, joints, bones or skin.

As sickle cells cannot change shape easily, they tend to burst apart (haemolysis). Normal red blood cells live approximately 90 to 120 days, but sickle cells last only 10 to 20 days. As a consequence, the bone marrow may have trouble keeping up with how fast the cells are being destroyed. Anaemia and low energy are the result.[4]

Outlook

The severity of SCD varies widely from person to person.

In high-income countries like the United States, the life expectancy of a person with SCD, most of which are of African ancestry, is approximately 40 to 60 years. (In 1973, this was only 14 years.) Advances in the diagnosis and care of SCD have made this improvement possible.

In sub-Saharan Africa, percentage of people born with sickle cell trait (carriers) can be as high as 25%, while up to 3% of all babies inherit the trait from both parents and develop SCD. Most African babies born with SCD die during childhood.[5]

The problem in haemoglobin S is caused by a small defect in the gene that directs the production of the *β globin* part of haemoglobin. This defect changes the way that haemoglobin works.

People with sickle cell trait (carriers) are generally healthy.

If a child's parents both carry sickle cell trait or another abnormal haemoglobin gene (like thalassemia, haemoglobin C, haemoglobin D, haemoglobin E), that child has a 25% chance of having SCD and 50% chance of becoming carrier.[4]

Signs, Symptoms and Complications

If a person has SCD, it is present at birth. But most infants do not have any problems from the disease until they are around 5 or 6 months of age.

Early symptoms of SCD may include:

- Painful swelling of the hands and feet (dactylitis).
- Fatigue or fussiness from anaemia.
- Jaundice of the skin or icterus of the eyes.

The signs and symptoms of SCD will vary from person to person and can change over time. Most of the signs and symptoms of SCD are related to complications of the disease.[4]

Acute Pain Crisis

Sickle cell or vasoocclusive crises can occur without warning when sickle cells block blood flow and decrease oxygen delivery. People describe this pain as sharp, intense, stabbing, or throbbing. Severe crises can be even more uncomfortable than postsurgical pain or childbirth. Pain can strike almost anywhere in the body and in more than one spot at a time. But the pain often occurs in the lower back, legs, arms, abdomen and chest.

A crisis can be brought on by illness (e.g., malaria), temperature changes, stress, dehydration or being at high altitudes. Often a person does not know what triggers, or causes, the crisis.[4]

Chronic Pain

Many adolescents and adults with SCD suffer from chronic pain. This kind of pain has been hard for people to describe, but it is usually different from crisis pain or the pain that results from organ damage. Chronic pain can be severe and can make life difficult. Its cause is not well understood.[4]

Anaemia

People with SCD usually have mild to moderate anaemia. At times, however, they can have severe anaemia. Severe anaemia can be life threatening. Severe anaemia in an infant or child with SCD may be caused by the following:

- **Splenic sequestration crisis.** A splenic sequestration crisis occurs when red blood cells get stuck in the spleen, making it enlarge quickly and causing severe anaemia.
- **Aplastic crisis.** This crisis is usually caused by a parvovirus B19 infection, also called *fifth disease* or *slapped cheek syndrome*. Parvovirus B19 is a very common infection, but in SCD it can cause the bone marrow to stop producing new red cells for a while, leading to severe anaemia.

Adults with SCD may also experience episodes of severe anaemia, but these usually have other causes. Severe anaemia may lead to symptoms that include:

- Shortness of breath.
- Being very tired.
- Feeling dizzy.
- Having pale skin.

Infections

People with SCD who have damaged spleens are at risk for serious bacterial infections that can be life threatening. Some of these bacteria include:

- Pneumococcus.
- Hemophilus influenza type B.
- Meningococcus.
- Salmonella.
- Staphylococcus.
- Chlamydia.
- *Mycoplasma pneumoniae*.

Acute Chest Syndrome

Sickling in blood vessels of the lungs can deprive a person's lungs of oxygen and cause damage to areas of lung tissue. Acute chest syndrome often starts a few days

after a painful crisis begins and is a life-threatening situation.[4]

Brain Complications

A clinical stroke occurs when sickling in blood vessels blocks the blood flow to a part of the brain. Also silent brain injury without outward signs of stroke occurs, resulting in learning problems or trouble making decisions.[4]

Other Problems

- Eye problems with damage to the retina.
- Heart disease: enlarged heart and pulmonary hypertension.
- Kidney problems.
- Priapism.
- Gallstones.
- Liver complications.
- Leg ulcers.
- Joint complications.
- Delayed growth and puberty.

Pregnancy

Pregnancies in women with SCD can be risky for both the mother and the baby.

Diagnosis

In high-income countries, it is easy to diagnose whether a person carries a gene for abnormal haemoglobin that they could pass on to a child. In Africa, these tests are not generally available. Also newborn screening is not the norm, let be prenatal screening. The diagnosis of SCD in a child is only made once it has been hospitalised with symptoms for the first time.

Treatment Options in Regular Medicine

Currently, hematopoietic stem cell transplantation (HSCT) is the only cure for SCD. It may be clear that this option is fiction for Africans born with SCD.

In the West, early diagnosis and effective treatments that can reduce symptoms and prolong life have raised life-expectancy of children born with SCD considerably.

Prospects for African children born with SCD are still very poor. Antibiotics and painkillers are available if needed. Intravenous fluids are given in acute crises and blood transfusions in cases of severe anaemia. Other remedies like hydroxyurea to prevent or decrease complications are simply not provided by most African clinics.

Fever is a medical emergency in SCD. As the incidence of SDC is highest in areas where malaria is endemic, it is often malaria that creates a crisis in SCD patients. It is often malaria that causes their death.

More Case Reports on PC1436n/SR1436n

As the case with Evans indicated, it is possible to treat SCD with a disease-specific remedy for it. As indications are not allowed on a label, the remedy is called PC1436n or SR1436n. To be able to advice and prescribe this remedy with confidence, more feedback from treated cases was needed. The reports below are from people who have been trained by the Amma Resonance Healing Foundation and apply PC resonances to the benefit of the people in their region.

Report from Kenya in 2014 by Benta Achieng

[Transcript video recording by Nico Beentjes on PC1436n for sickle cell anaemia]:

> I am Celestine A.O. I am mother to two kids with sickle cell. They are doing well after PC Sickle cell. Before PC Sickle cell they were sick. They had joint pain, yellow eyes, yellow urine; they had problems with their stool, they had no strength. They had a lot of pains, both of them. Now they are doing well. They are now doing fine; no more symptoms.
>
> Erokamano mama! (Thank you ma'am!)

Report from Congo in 2015 by Joseph Ndebo Balikwisha

Since several months, we are treating the sickle cell anaemia with PC 1436n. In nine cases that we followed up, there is a very meaningful progress. Their health improves and the crises disappear.

Dr. Kapinga in Goma treated two cases. He saw the rate of haemoglobin increase from 5 to 10 resp. 11 g/dL.

As proof of the efficiency of this remedy, some parents go as far as telling me that, if one day we can't provide this remedy to their children, they will sue us.

A soldier of the Congolese army who came back from the front line to Beni passed directly by my office to inquire about the product that relieved his daughter and saved her life. He and his wife had lost their two eldest children because of the illness.

In Uganda, all four cases of SCD that are under treatment of PC 1436n evolve well.

Since 2010 to 2014, we treated 15-year-old Miss B.M. without success for SCD. Tests showed that 100% of her red blood cells had the abnormal sickle cell structure. Her haemoglobin was 8.2 g/dL and the white blood cells were 28.400/mm^3 (normal: 4–10.000).

After 8 months of treatment with PC1436n, she presented with 95% of abnormal cell structure (5% of cells became normal) with a haemoglobin rate of 10.2 g/dL and the white blood cells equivalent to 9.700/mm^3. It is a very meaningful progress.

Report from Kenya in 2015 by Yuanita Hongo, Clinician

I visited a sickle cell client in hospital (►**Fig. 4**) who improved so well with a haemoglobin level that went up from 3 to 8.1 g/dL.

[Transcript video recorded by Nico Beentjes on PC1436n sickle cell anaemia; Kisumu 2015]

Patient: 'I'm M.A. I'm 27 years old by now. I'm born in a family of 5. I'm the 3rd born. We used to be 6, but then we lost our 1st born because of sickle cell disease. I was diagnosed of sickle cell disease when I was 8 months old; that was when I was very, very young. So, it has been a challenge living with

Fig. 4 A.M. hospitalised.

the sickle cell disease, because most of the time, I'm admitted, I stay at the hospital. And when the crisis comes, when the attack comes, you feel like it's a no-go zone because of the pain. The pain is very, very severe; fever, there's dehydration, you have to be transfused. And at times, the challenge I normally get is that there is no blood in the hospital when I'm admitted. Because I'm blood group A-positive, and at times that A-positive is not in the hospital. So before even transfusion itself, I feel so bad because of the joint pains, the fever, the headache, and the pain is very, very severe. Actually, it has not been easy for me, even at my workplace, because of my condition. Even when I was at school, it was not very, very easy for me, because I had to move in and out, in and out. But then, a few months earlier, that was in the month of July 2015, my supervisor came with a drug called PC. And she introduced me to the drug, the PC Malaria and the PC Sickle cell. So, I've been taking PC Malaria and PC Sickle cell, and at least I can say I have improved, because my haemoglobin has never gone down again and the pain has not been very, very severe, like it was before. And since then, I can say that, I've never had that attack, that crisis. I'm very grateful and I'd wish that they continue bringing us the drug so that we can live a long and better life. Thank you' (►**Fig. 5**).

Yuanita Hongo: 'Yes. I'm a clinician, and M is my client, my patient. She has been having sickle cell disease for long. Recently, she was in the ward, but before that, her haemoglobin level was 3 g/dL, but after being admitted in the ward, it didn't improve, so I came up with the remedy PC Sickle cell, which I introduced to her. She has been taking PC Sickle cell and PC Malaria, and currently, she has really improved. Her haemoglobin level is 8.1 g/dL, and her general condition, as you can see, has really improved. The pains have reduced; no more headache, no more fever; abdominal pain and joint pain, very, very minimal. Therefore, I feel that the PC remedy for sickle cell actually is working for her, and she is continuing taking the PC remedy as we sit now'.

Fig. 5 A.M. after treatment (still from video).

Another 'sickler', a boy called J. has not had major crisis since I gave PC sickle cell in February 2015. He joined Maseno University on 5 September 2015 for a degree course.

Mighty Ray—Report from Nigeria (2015–2017) by Dr. Elton Akpabio, Homeopathic Physician

On Saturday, 11 July 2015, a single mother brought her little son of 4 years old by name 'R.' to me for a case of sickle cell anaemia. The situation was very acute and she thought the little R. was going to die. I thought the same.

I prepared PC1436n in a 75cl bottle water and gave to the mother (Ms. W.). I instructed her to do vigorous succussion and give to the boy as many times as possible in a day since the situation was very acute. He was close to death. The extreme high fever with deep seating excruciating pains along with complete prostration defied all initially given medications.

But surprisingly, she called me today and told me that the boy is alive. The fever has gone and the pain has reduced to bearable standard.

I want the boy to continue with the remedy for 30 days after which we will examine the haemoglobin level under microscope. Then I will send my report.

I have another boy of around 18 years with three other adults of not less than 25 years each that I am following up. Soon I will send their reports.

January 2016

It is my great pleasure to welcome you and the entire team of ARHF into 2016. Yesterday, our Amma4africa—Nigeria office—was full to capacity as we celebrated the successful case of sickle cell anaemia of a 4-year-old boy R. that I wrote to you before.

Master R. lives with his single mother Ms. W. The sickle cell condition of master R. broke the marriage of his parents leaving R. in the care of his jobless single mother. The agony of a single

mother taking care of a sickle cell boy subjected her into terrible humiliating conditions. She was referred to me last year by a pharmacist friend who has been assisting in some medications when there was a health crisis with little R.

R. has been able to stay healthy without a single health crisis for 90 days. This is an incredible and a remarkable result because he used to experience health crises almost at weekly basis before. Yesterday we celebrated with him.

We received report from Bela Medical Laboratory. His current haemoglobin is 4.6 mmol/L while the PCV (packed cell volume) is 14% (normal = 31–44%). The initial report before the commencement of PC sickle cell was haemoglobin of 3.1 mmol/L and PCV of 10%. The result is not as impressive as I thought and we are very surprised that this improvement has been enough to prevent any crisis for 3 months—what a miracle! A reason could be that this is the peak period of mosquito bites and a recent bout of malaria could have caused a dip in his haemoglobin. It may also be an indication that PC sickle cell goes far beyond mere increases of physical haemoglobin and PCV into the dynamics of sustaining *life* regardless of the level of haemoglobin and PCV.

My next line of treatment is to combine PC sickle cell and PC malaria at intermittent rate for optimum result.

Fig. 6 Mighty Ray, December 2017.

December 2017

The combination of PC 1436n (sickle cell) with PC 240m (malaria) has really proven wonders in the life of boy 'R.'—fondly called *Mighty Ray*. On Friday, 8 December 2017, the mother brought the boy to my office from school with a test result indicating PCV of 20% and Hb of 9.8 mmol/L. The greatest wonder is that since 21 January 2016 till date, the boy has experienced only two crises compared to the monthly and sometimes biweekly crises he used to have. Please see pictures of *Mighty Ray* as he came back from school on Friday (8th December 2017; ►**Fig. 6**).

Report from Kenya (2017) by Monica Atieno Omondi

Since April 2016, thirty-eight cases of SCD have been treated with PC1436n. Their age at intake ranges from 4 months to 18 years old. The main complaints that are being reported are yellow discoloration of the eyes, joint pains, general weakness, oedema of the legs and ascites. Many patients feel very hot before the start of the treatment. The typical story is that they have crises very regularly and are often brought to the hospital, where they would get painkillers or blood transfusion. Some were so weak that they had to be carried to come for consultation. Others were ill so often that they stopped going to school. In many cases, a 'sickler' in the family causes a lot of strain on the parents and their finances.

It is important to state that gathering data in Africa is not an easy job. The below data are far from complete but nevertheless very impressive considering the effects reported and measured.

Results

To complete their data, all patients who had not come for follow-up were contacted. In total, there are follow-up data available of 34 patients. The follow-ups show that most symptoms completely disappear after a few weeks of treatment. Joint pains and yellow discoloration of the eyes can remain, but with a much lesser extent. Measured on a visual analogue scale, all symptoms are reduced by 75% or more. The most objective parameter though is the level of haemoglobin. For children a normal haemoglobin level ranges from 11 to 16 g/dL, and the average level in this group at intake was 4.33 g/dL, while after treatment the average raised to 8.15 g/dL (►**Table 1**).

Although still below the norm, the raise is impressive and statistically significant, but more importantly experientially significant as it means a child is free from symptoms.

Two patients appeared to have died—one from malaria and the other probably also from malaria. At the time of their death, both patients had discontinued treatment with PC1436n for about 9 months and they were not taking PC240m to prevent malaria.

None of the patients who kept using PC1436n needed to return to hospital for sickle cell crisis. As a consequence, many had no longer tested their blood for haemoglobin; so, we do not have follow-up levels.

Conclusion

It is with great gratitude and joy that we witness and share these life-changing results in children who were once cursed by SCD now live a normal life. Our deepest wish and hope is that this treatment may reach many more. May you find it in your heart to join us, please contact us through the Web site of ARHF: www.arhf.nl. In November this year, ARHF is planning a volunteers training in Muenster, Germany. You are most welcome.

Table 1 Symptoms and haemoglobin (Hb) levels before and after treatment

	Number #	Intake 0 - 5		Follow-up 0 - 5		Change percent
		average	range	average	range	
Headache	8	2.33	1–4	0.25	0–1	<89%
Hot feeling	15	2.72	1–4	0.19	0–1	<93%
Sweating	3	2.75	2–3	0	0–0	<100%
Swollen legs	13	2.57	2–4	0.15	0–1	<94%
Ascitis	8	2.5	2–4	0.25	0–1	<90%
Joint pains	19	2.9	2–4	0.37	0–1	<87%
Weakness	21	2.33	1–4	0.14	0–2	<94%
Nosebleeds	3	1.67	1–3	0	0–0	<100%
Yellow eyes	29	2.84	1–4	0.72	0–3	<75%
Hb g/dl	22	4.33	2.3–7.8	8.15	4.3–12.0	>88%

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