A New Approach in the Treatment and Prevention of Thalassemia And Some Rare Diseases

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ABSTRACT

Recent advances in the treatment of thalassemia besides bone marrow transplant aimed to treat or improve the quality of life of the patient with frequent transfusions and chelating agents did show some convincing results in a group of patients but eventually it goes a long way to accomplish some realistic approach to treat the disease or even improve the quality of life of the patient. The role of Fagonia Cretica (FC), in Thalassemia was a chance occurrence, when a patient suffering Thalassemia Major improved in a convincing way.

Our earlier clinical trials showed that there was significant decrease in HbF and HbA2, whereas Hb A showed significant improvement after about 10±2 month treatment. It was therefore planned to conduct a clinical trial to confirm the effect of FC in Thalassemia Trait. Fagonia Cretica in doses of 120mg/kg body wt also showed statistically significant results in quite a few other rare diseases, like Myelodysplastic Syndrome (MDS) and Porphyria not responding to other conventional treatments. This study also gives case reports of patients, suffering Myelodysplastic Syndrome (MDS) and Congenital Erythropeietc Porphyria.

Could this treatment possibly give a chance of eradicating thalassemia, a crippling disease? The patients included in this trial had initially Hb A2 above 3.5 % i.e., a pathological level, on electrophoresis at the time of first diagnosis. This study explains: The patients, who had their Hb A2 ≥ 3.5%, after a treatment of 10 ± 2 month had their Hb A2 significantly ($p \leq 0.000$) reduced to normal limits.

These results encourage us to conduct multi-center long term studies of Fagonia Cretica in quite a few other hemolytic disorders as well, as this has shown very positive results in diseases like Myelodysplastic Syndrome (MDS), and Congenital Erythropeietc Porphyria, besides thalassemia.

Key Words:
Fagonia Cretica; Thalassemia trait; Myelodysplastic Syndrome (MDS); Congenital Erythropeietc Porphyria; Genetic Mutation

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1: INTRODUCTION

This paper probably meets the expectation in overcoming the challenges in the prevention and clinical management of thalassemia and a few rare diseases, like Myelodysplastic Syndrome (MDS) and Congenital Erythropeietc Porphyria.

Thalassemia besides being a crippling disease also results in quite a lot of social and cultural implications. Family bonds are also at risk and invariably culminate in break up. The new generation with better quality of treatment is often school going and is well aware of the ultimate consequences of the disease. Despite intensive treatment, various complications, which an educated child is well aware, are always haunting their minds. Above all, invariably, it is not possible for the family to follow the strict guide lines of treatment, because of the obligatory socio-cultural and socio -economic conditions.

Our earlier studies (16,17) clearly elucidate the impact of this crippling disease not only on the family life but also on the personal behavior of the individual. These studies had definite objectives:

1: To improve the clinical status of the individual so that he can become a healthy member of the society. (17)

2: Work out a program if we could possibly bring an end to the transfer the disease to the next generation, possibly initiating genetic mutation i.e., reduce Hb A2 to less than 3.5%.

3: Successful results of the treatment with FC in certain rare diseases like: Myelodysplastic Syndrome (MDS); Congenital Erythropeietc Porphyria persuade us to perform detailed studies on the exact mode of action of Fagonia Cretica (F C) and its different ingredients alone or in combination in all these conditions.

F C, a member of the family Zygophyllaceae, is a small wild spiny under-shrub found mostly in dry calcareous rocky and sandy soils throughout Indo-Pakistan sub continent. Its medicinal value is well documented [1-5]. The plant tastes bitter and is usually used for the treatment of fever, dysentery, asthma, skin infection and liver troubles [2, 3, 5]. Its active ingredients are reported [1-3] and the effect of each ingredient, like bitter alkaloids – especially the Saponin-1 and Saponin-2, has been studied on various animals [6-10]. A lot of work has already been done to dig out the active ingredients of Fagonia cretica. Some of its chemical constituents are: *Triterpenoid Saponins: Saponin 1 and Saponin 11, besides it contains beta- sitosterol; ceryl-alcohol; chinovic acid; water soluble saponins, i.e., glucose rhamose; xylose; arabinose; fagogenine and lipids 0.3-1.14%: Campesterol; aglycone; fagonin; oleanolic acid; betulic acid, the later four are derived from the saponins fraction* [1-3].

The greatest difficulty in the recognition of this plant (F C) is experienced, when it comes to differentiate it from Fagonia Arabica (F A). We don't know for sure the quality and the difference of ingredients between the two but the results of the two i.e., FC and FA when mixed, are quite confusing. We need to cultivate this wild herbal plant on a mass scale in a definite area so that there is no chance of mixing the two.

The second problem arises after the plant collection.

Collection of this plant is advisable from 15th February to 15th May of the year. This being a wild plant grown in the deserts and dry calcareous lands in extreme weather conditions, but after collection it becomes susceptible to destroy, if not dried and stored under carefully well organized conditions. It needs to be stored and dried completely in shadow. If pounded or powdered, when still wet, it is prone to fungus infestation. During the last thirty seven years of our experience the best results of the plant are accomplished when the coarsely pulverized aerial parts of the plant when soaked in water for twelve hours then finely minced and rinsed through fine muslin. People have used the tea of the plant in cancer patients but that's not so effective in thalassemia or other hemolytic disorders. Liquid form is very bitter and the patient compliance is very difficult.

This plant is quite safe in infants and also in pregnancy and can be safely administered during ill health with other medicines. The only side effect observed is occasional gastroenteritis, when taken on empty stomach.

During the last couple of years it has become clear that Thalassemia is extremely heterogeneous and that its clinical picture can result from the interaction of many different genetic defects all of which result from a reduced rate of production of one or more of the globin chain(s) of hemoglobin. Because Thalassemia occurs in populations in which structural hemoglobin carriers are common, it is not at all unusual for an individual to receive a Thalassemia gene from one parent and a gene of structural hemoglobin variant from the other, besides structural hemoglobin variants from the donor's blood. These different interactions produce an extremely complex and clinically diverse series of genetic disorders, which range in severity from patient to patient and this could possibly result in varying response of FC in some patients

Our earlier studies [16,17] showed:

1: **The** patients belonging to Thalassemia Major, Sickle Cell Thalassemia and Thalassemia Intermedia, who had initially Hb A present on electrophoresis before any transfusion, when they started the treatment of FC, there was hardly any need of transfusion.

2: **The patients, who initially had no HbA present on electrophoresis, but their liver and spleen**, were enlarged; this group of patients experienced increase in the interval between two transfusions 30±8 days to 75 ±12 days.

3: **The patients, who initially had no HbA present on electrophoresis and their liver and spleen were not quite palpable** below costal margin did not show significant change in the transfusions needs. But we observed that there was considerable improvement in the quality of life in these patients.

The challenging question in the last group of patients was:

Where was the iron going, if not in the Spleen, Liver, bones or other Viscera?

This has yet to be answered.

The response to the serum ferritin level to FC was quite variable in different patients. Some of patients experienced a rapid decrease in the serum ferritin level, whereas in others there was no significant change. We need to work on this aspect of the subject more dispassionately because FC is quite safe and also less expensive.

1. MATERIAL AND METHODS

This study is composed of two parts. The first component has been designed as a usual treatment case controlled-study based on the encouraging results from our earlier studies (16, 17).

In the first part we conducted a statistical analysis for a total of 68 Thalassemia Minor patients, who were randomly picked from Dr. A. Q. Khan Thalassemia Research Center. Their ages ranged from 17 to 45 years, but only twenty four patients completed the study. These patients were given FC in 120mg/kg body wt* dose for 10 ± 2 month and their Hb electrophoresis was done just before the start and at the end of study to evaluate Hb A2. These patients were not on transfusion, so there was no fear of overlap of the donor's blood. Besides measuring Hb A2, we also measured total Hb for academic sake. We used the paired t-test for the comparison of means to see the effect of the treatment of FC on the two parameters of Thalassemia Minor.

** This dose of the herbal product was calculated after a period of thirty five years of experience with this herbal product*

We had a chance observation of the effect of FC in certain other Hemolytic disorders like Myelodysplastic Syndrome (MDS) and Congenital Erythropeietc Porphyria. The second part of study included one patient each belonging to these rare diseases. There is no Known Treatment of these two rare diseases. Perhaps the detailed study of these cases might give a clue to the possible mode of action of this herbal preparation.

Case Report 1: Myelodysplastic Syndrome MDS or (Evan's Syndrome)

A fifty five years old diabetic patient [**Fig 1**] was suffering from ischemic heart failure. In early 2012 he experienced severe weakness because of sudden fall in his haemoglobin. He was diagosed suffering Mylodysplastic Syndrome (MDS) [Table 3, 4]. He was clearly told the prognosis of the disease with ultimate eventualities and complications. Knowing all that he visited Dr. A. Q. Khan Thalassemia Research Center OPD on 5th Aug 2013. *He had a personal experience of FC in a patient of thalassemia in his family.* When he visited the Research center: He had massive ascites with pretibeal edema and cardiomegaly. His Hemoglobin was 7.8 gm. He had not been responding to the conventional treatment for IHD and Diabetes. He was receiving fortnightly transfusions for severe anemia resulting from MDS. After explaining our limitations for MDS, the FC was given in 120mg/kg body wt doses. Subsequently when he visited the Center on 18th Nov 2013. He had not used any transfusions after he started using FC. His Hb was 12.5 gm and later 12.5±1.5 gm on the subsequent visits. He also showed a convincing response to the conventional treatment for his diabetes and IHD. His ascites and pretibeal edema also cleared. His bone marrow biopsy and peripheral blood reports are given in **[table 3**,**4]** that were suggestive of ineffective hemopoiesis. He was never in need of transfusion till his last breath, when he died of acute myocardial infarction after about one year.

Case Report 2: **Congenital Erythropeietic Porphyria**: A twelve years old patient **[Fig 2]** was clinically evaluated and studied at Bedford Hospital London (U.K) in mid July 2013, and diagnosed an extremely rare, congenital porphyria, also known as erythropoietic porphyria or Gunther's disease.

"Porphyria was first described by Schultz in 1874. Since then, less than 100 cases have been reported. Solar sensitivity is the most striking symptom but systematic effects may also be severe. The disorder is inherited in a Mendalian autosomal recessive manner. There is evidence that the defect lies at the level of uroporphyrinogen cosynthetase in the biosynthetic pathway. Boys and girls are equally affected and symptoms usually begin during the first few years of life, although the disease can occasionally present in middle age. In addition to these integumentary lesions, a number of patients also develop anemia and splenomegaly. The anemia is caused by ineffective erythoropoiesis with shortened red cell survival. The bone marrow reveals normoblastic hyperplasia; a proportion of erythrocyte precursors fluoresce red in ultraviolet light due to high porphyrin content. The peripheral blood film shows normocytic normochromic anaemia with polychromasia. There is usually a moderate reticulocytosis with Howell – Jolly bodies; leucopenia and thrombocytopenia may occur when the spleen is large. Spleenectomy may improve the anemia and can reduce the degree of photosensitization. Although not described until 1961, this form of erythropoietic porphyria, also known as erythrohepatic protoporphyria, is much more common than congenital porphyria. It is inherited as an autosomal dominant and symptoms may occur at any age, including infancy and childhood. Ferrochelatase activity is reduced in peripheral blood, liver, bone marrow, and skin. Diagnosis can be made by demonstrating fluorescence in a proportion of red cells (fluorocytes) in the peripheral blood and confirmed by measurement of greatly increased erythrocyte and fecal protoporphyrin."

This patient reported in the OPD of Dr. A. Q. Khan Thalassemia Research Center on 28th June 2014. Patient, when initially evaluated at Bedford Hospital London**,[Table 5A; 5B]** was suggested 'Bone Marrow Transplant', but was also told the unpredictability of the outcome, because no previous reports were available on the subject. Patient was getting regular transfusions at monthly intervals. Patient started Fagonia Cretica treatment on 29th June 2014. This was the first patient of *Congenital Erythropeietc Porphyria* we had ever seen, therefore, his parents were very clearly told to use the medicine at their own risk. An email message from Riyadh, Saudi Arabia: "*Respected Dr. Abdul Rashid Seyal Sahib. My son Hassan Mohammad Saeed (file no.923) is under your treatment and having 4 tablets of Casemia 3 times a day since 30th June 2014 .Like I wrote you before that Alhamdulillah since July 2014 he did not need any blood transfusion because his hemoglobin has been above 10 which is required by the doctors in UK."*

His routine blood report **[table6]** after about four month's treatment shows a normal haemoglobin level. Patient is now free of any symptoms referring the disease besides having no need of any transfusion.

Results

The response to the treatment with Fagonia Cretica *family Zygophyllaceae* in Thalassemia Minor patient in terms of their total hemoglobin and HbA2 were measured at the start of the study and again at the end of study [Table 1] after about 10±2 months. These patients were not on transfusion at any stage in their lives. The mean values of different parameters are given in [Table 2]. The total hemoglobin significantly improved from 1.12 to 1.98g ($p<.000$). The mean values for hemoglobin before and after the treatment were 10.81g and 12.36g respectively. The average values for HbA2 were 4.42 and 3.25 before and after treatment, respectively and the reduction was from 0.96 to 1.37 ($p<0.000$) significant. It is also observed that the mean value of HbA2 at the start of the study is 4.42, which is significantly higher than 3.6($t=7.47$, $p=0.000$), which indicates that patients were suffering Thalassemia Minor, whereas at the end of ten months the mean value reduced to 3.25, which is significantly lower than 3.4 ($t=2.19$, $p<0.05$) indicating: they were no more in carrier state.

2. Discussion

DNA not only can make people sick but also can alter the original parent sick gene to normal [11-14]. The classic views assumed that what are termed "silent" mutation were inconsequential to health, because such changes in DNA would not alter the composition of the proteins encoded by genes. Proteins function in virtually every process carried out by cells, from catalyzing biochemical reactions to recognizing foreign invaders. Hence, if a protein's makeup ends up being correct, any small glitches in the process leading to its construction could do no harm to the body, but instead will provide a relief to the sick masses [15-17].

A total of 68 parents of the patients suffering Thalassemia Minor were randomly picked from Dr. A. Q. Khan Thalassemia Research Center. Their ages ranged from 27 to 45 years, but only twenty four completed the study. These subjects were given FC in 120mg/kg body wt dose for 10±2 months and their Hb electrophoresis was done just before the start and at the end of study to evaluate Hb A2. These patients were not on transfusion. Besides measuring Hb A2, total Hb was also measured for academic sake. We used the paired t-test for the comparison of means to see the effect of the treatment of FC on the two parameters of Thalassemia Minor. The results as discussed before clearly elucidate the role of Fagonia Cretica in Thalassemia Trait patients, besides the most encouraging results in the treatment of two very rare diseases that had no known treatment before this moment. The purpose of including these two cases with thalassemia trait is to dig out the possible mode of action of Fagonia Cretica in causing genetic mutation and correcting the transfusion needs in these two rare diseases.

8: References

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Table 1: Thalassemia Minor (They were not on transfusion at any stage before or after the treatment of Fagonia Cretica was started. Follow up period is 10±2 months)

The Total Hemoglobin and HbA2, before and ten months after the study in 24 patients

S#	Age/Sex	Total HB		Hb A2	
		Before	After	Before	After
1	**42m**	**11.4**	**12.3**	**4.7**	**3.3**
2	**32f**	**10.6**	**11.5**	**4.5**	**3.2**
3	**26f**	**9.6**	**12.6**	**5.2**	**2.9**
4	**29f**	**8.9**	**11.3**	**4.9**	**3.5**
5	**26m**	**12.6**	**12.4**	**5.2**	**3.6**
6	**39m**	**11.2**	**12.8**	**4.8**	**3.4**
7	**28f**	**10.8**	**11.4**	**5.2**	**3.8**
8	**31f**	**9.8**	**10.9**	**4.7**	**3.2**
9	**33m**	**11.2**	**13.8**	**4.2**	**3.1**
10	**22f**	**10.4**	**12.2**	**3.8**	**3.2**
11	**32f**	**10.6**	**12.4**	**3.7**	**3.0**
12	**32f**	**9.8**	**11.4**	**3.9**	**3.2**
13	**42m**	**10.2**	**12.5**	**4.2**	**3.1**
14	**45m**	**12.2**	**13.5**	**5.2**	**3.4**
15	**17f**	**11.2**	**12.5**	**4.4**	**3.5**
16	**22m**	**11.4**	**12.7**	**3.9**	**3.2**
17	**32m**	**10.5**	**12.2**	**3.8**	**3.3**
18	**44m**	**11.2**	**13.5**	**3.7**	**3.5**
19	**42m**	**12.2**	**14.5**	**3.9**	**2.9**
20	**35m**	**11.4**	**10.4**	**4.2**	**3.5**
21	**36f**	**10.4**	**12.5**	**5.2**	**3.4**
22	**30f**	**10.2**	**13.3**	**4.6**	**3.5**
23	**27f**	**11.2**	**12.6**	**3.9**	**3.1**
24	**19f**	**10.4**	**13.4**	**4.3**	**3.4**

Table2: Thalassemia Minor

Mean and standard deviation of the different parameters selected at the time of first diagnosis for (Hb; HbA2) and at the end of study for all the parameters

Parameter	Intermediate Mean		95% Confidence Interval	
	Before	After		
HB	10.81	12.36	1.12-1.98	Significantly Improved
HBA2	4.42	3.25	0.96-1.37	Significantly Reduced

Table 3: Bone Marrow Biopsy Report in a Myelodysplastic Syndrome (MDS) Patient

Clinical Information: Pancytopenia.

Site (s): Right posterior iliac crest.

Trephine: Histological sections show a moderately cellular marrow with prominent fat spaces. Hemopoietic activity is observed. Erythropoiesis is somewhat prominent. Megakaryocytes with a hypolobulated nucleus are seen. There is no evidence of extramedulláry infiltrate. Imprint smears are in accordance with histological sections and aspirate smears.

Peripheral Blood Film: RBC's are showing moderate anisocytosis, spherocytosis.

There is leucopenia with relative lymphocytosis. There is moderate thrombocytopenia.

These features are suggestive of ineffective hemopoiesis. Most probable cause is myelodysplastic syndrome. Second possibility is Evan syndrome with secondary foliate deficiency. Relevant work up is suggested.

TABLE 4: Abdominal Ultra- Sonographic Report

Inhomogeneous echo-textured liver with splenomegaly and mild abdomino-pelvic ascites. Possibly tiny adherent calculus in gall bladder

Table 5.A:

Bedford Hospital
NSH Trust
Kempston Road
Bedford
MK42 9DJ

Department of Clinical Biochemistry
Direct Line: 01234 792166
Fax: 01234 795915

PROPHYRIA REPORT

Date: 24th July 2013
Chemical Pathology
Great Ormond Street Hospital for Children NHS Trust
Great Ormond Street
London
WCIN 3JH

PRELIMINARY REPORT

Re Saeed Hassan Mohammed Hosp No: 862597 D.O.B: 06/04/2005
Clinical Details: Myelofibrosis, Hirsutism

Random urine (Date: 14/06/2013) ,		Your Ref: 13C088617
5-aminolaevulinic acid (ALA)	= 5.53	µmol/mmol creatinine (Normal: less than 3.8)
Prophobilinogen (PBG)	= 0.70	µmol/mmol creatinine (Normal: less than 1.5)
Total Porphyrin	= 7233.5	µmol/mmol creatinine (Normal: less than 35)
Uroporphyrin-I	= 3457.5	µmol/mmol creatinine
Uroporphyrin-III	= 536.5	µmol/mmol creatinine
Total Uroporphyrin (I+III)	= 3994	µmol/mmol creatinine (Normal: less than 3)
Heptacarboxylate porphyrin	= 303.6	µmol/mmol creatinine (Normal: less than 1)
Coproporphyrin-I	= 2849.2	µmol/mmol creatinine (Normal: less than 6)
Coproporphyrin-III	= 86.7	µmol/mmol creatinine (Normal: less than 24)
% Coproporphyrin-III	= 3	% (Normal: 68-89)
Creatnine	= 7.2	mmol/L
Hexa – and pantacarboxylate porphyrins are raised.		

Table 5.B:

FINAL REPORT

Re Saeed Hassan Mohammed Hosp No: 862597 D.O.B: 06/04/2005
Clinical Details: Myelofibrosis, Hirsutism

Random urine (Date: 14/06/2013) ,		Your Ref: 13C112394
Total Porphyrin	= 1602.9	µmol/g (Normal: less than 50)
Coproporphyrin-I	= 1433.4	µmol/g (Normal: less than 5.5)
Coproporphyrin-III	= 131.5	µmol/g (Normal: less than 2.5)
% Coproporphyrin-III	= 8.4	% (Normal: 16-35)
Dicarboxylate porphyrins (“protoporphyrin”)	= 38.0	µmol/g (Normal: less than 1)

The pattern of faecal porphyrin excretion is consistent with congenital ERYTHROFELETIC PORPHYRIA (CEP), coproporphyrin type-I was predominantly raised. These data and previous report dated 2th July 2013 are typical of CEP.

Dr. W S Wassif
Consultant chemical Pathologist

Table 6:

Al-osrah medical clinic center
Riyadh-Cross of BBatha St. Whith Prince
Moh'd Bin Abdulrahman Road (Sitten)
Al-Owaidah Bld
Tel: 4572417 – Fax: 4572446

Invoice No.	823650		
Case Ser	135056	Sex	MALE
Patient File	269538	Age	9
Patient Name	Hassan Mohammad Saeed Tariq		
Date	15-11-2014		

(The above mentioned clinical data could not be done in Riadh)

Test	**Result**
mplet Blood Cells Count	
WBC	5.2
RBC	3.51
HB.	10.7
HCT	31.4
PLT	160
MCV	89.5
MCHC	30.5
Differential count:-	34.1
LYMP	45
NEUT.	44
MONO.	9
EOSINO.	2
BASO.	0

- **Fig 1 .** +92 301 759 7030

The patient of MDS responded immediately to the treatment and never needed blood transfusions thereafter.

(Patient unfortunately died of acute myocardial infarction in mid 2014)

- **Fig 2.** + 96 6500431494
  + 92 333 4134758

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