Hence, PCR amplification followed by Mst II digestion confirms the sickle cell mutation, both in homozygous as well as heterozygous states.

Hence, using PCR technique and restriction digestion it is possible to diagnose antenatally single gene mutation. For this, no radioactive material or any external probe is used. The diagnosis is possible by the second day after the biopsy and it can be performed in the first trimester of pregnancy. Hence, medical termination of pregnancy if required, can be recommended. This technique (PCR), coupled with non-radioactive probing can also be effectively used for the prenatal diagnosis of thalassemia.

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False Positive Widal Reaction in Malaria

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Both malaria and typhoid fever are endemic in Surat and the surrounding areas of South Gujarat. The diagnosis of malaria is clinical and confirmed by the presence of malarial parasite in the peripheral smear. Widal test inspite of its nonspecificity and unreliability, is still used as the gold standard for the diagnosis of typhoid fever(1). A positive widal reaction in malaria poses a diagnostic dilemma in the evaluation of a

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TABLE I–*Data of 8 Subjects with Positive Malarial Parasite and Widal Reaction*

	1	2	3	4	5	6	7	8
History:								
Age (years)	4	8	8	12	10	14	8	11
Sex	M	F	M	F	M	M	M	F
Past history of recurrent malaria	+	+	+	-	+	-	+	+
Prior treatment before admission:								
Antimalarial	+	-	+	-	+	-	-	+
Antibiotics	-	-	-	-	+	+	-	-
Laboratory								
Total WBC	6×10	6.4×10	8.2×10	9×10	7×10	5.2×10	7.4×10	6.8×10
PS for MP on admn.	P. falci.	P. Vivax	P. falci.	P. falci.	P. falci.	P. falci.	P. falci.	P. vivax.
After treatment	-	-	-	-	-	-	-	-
Urine R/E/C7S	NAD	NAD	NAD	NAD	NAD	NAD	NAD	NAD
s WIDAL	H O	H O	H O	H O	H O	H O	H O	H O
On admn.	1/160 1/160	1/240 1/240	1/320 1/320	1/240 1/240	1/160 1/160	1/240 1/240	1/240 1/240	1/160 1/160
Repeat after 4 weeks	Same	Same	Same	Same	Same	Same	Same	Same
X-ray chest	-	NAD	-	-	-	-	-	-
Stool culture	Negative	Negative	Negative	Negative	Negative	Negative	Negative	Negative
Blood culture	Negative	Negative	Negative	Negative	Negative	Negative	Negative	Negative

TABLE II–*Prevalence of Titers of Salmonella H and O Antibodies in Control Group*

Age (yrs)	No. of children tested	No. of Children without antibody	No. of Children with Antibody							
			H antibody titre				O antibody titre			
			20	40	80	160	20	40	80	160
5-9	12	6	4	2	0	0	0	3	3	0
10-12	9	5	2	2	0	0	0	2	2	0
13-15	4	2	2	0	0	0	0	1	1	0

The reasons for a false positive Widal in our cases could be because of: (*a*) Infection by non typhoidal *Salmonella* of group B and D which have cross-reacting O antigen(5) or cross reactivity of *Salmonella* antigen with antibodies, produced in response to infection with other Gram negative bacilli(6). None of the 8 subjects from the study group were having either diarrhea or stool culture growing *salmonella*; (*b*) Subclinical *Salmonella* infection is a possibility that cannot be ruled out, but because, repeat Widal titers were identical with previous ones and all subjects were clinically asymptomatic after 3 months of follow up, it is unlikely; (*c*) Malaria derived mitogens may be responsible for polyclonal B-cell activation, which can result in hypergamaglobulinemia in repeated malarial infection in an endemic area(7). Only small proportion of these immunoglobulins (5% IgG) constitute antibodies reacting with malarial antigen and the main portion is formed by antibodies which react with other antigens such as heterophilic antigen or show autoimmune reactivity(7). Thus, recurrent malaria which was seen in 6 study subjects, can theoretically be associated with false positive Widal test.

We conclude that one should be aware of the possibility of false positive Widal reaction in a case of malaria in endemic area. We advise that in such a situation one should treat for malaria only with possibility of resistant case in mind, unless blood culture shows growth of *Salmonella typhi* or repeat Widal shows rising titers in a nonresponsive febrile child.

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Congenital Erythropoetic Porphyria

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Congenital erythropoetic porphyria (CEP) or Gunther's disease is a rare autosomal recessive disorder that causes chronic photosensitivity with severe mutilating lesions(1). Of the various types of porphyria, CEP is the least common and about 70 cases have been reported from different parts of the world(2). From India, Bhutani *et al.* previously reported a case of photodermatosis due to erythropoetic protoporphyria(3). We describe a case of CEP which was diagnosed on the basis of clinical, histological and bio-chemical features at our hospital.

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Case Report

An 18-month-old Muslim boy born of consanguineous marriage was admitted in the Pediatric ward of North Bengal Medical Hospital with complaints of diffuse skin lesions since birth. His parents also noticed that he passed normal urine which became reddish (burgundy red) on standing. History revealed that the skin rash appeared first over the face and gradually spread to the upper part of the trunk, mainly the back.

The boy weighed 6 kg, length was 77.5 cm and had a head circumference 41 cm (all below one fifth percentile for age). Examination of the skin revealed diffuse areas of hyperpigmented macules with few areas of hypopigmented patches interpersed within them. The skin lesions were seen mostly over the photosensitive areas, being prominent on the face, scalp, neck, shoulder, extensor, extensor surface of arms and back (*Fig. 1*). The involvement of the skin was progressive with successive bouts of bullous formation followed by healing and scarring. On exposure to sunlight even for a few minutes the child felt extremely uneasy and irritated possibly due to intense itching. The sclap showed cicatrised alopecia and there was no evidence of hypertrichosis in any part of the body. There was abnormal yellowish mottling of the teeth with hypertrophic gums. The liver was palpable 2 cm and spleen 1 cm below the costal margin. The nervous system examination was normal.