Original Article

Silvery gray hair syndromes: An insight into diagnosis

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ABSTRACT

Context: Silvery gray hair syndromes consist of three conditions, Chediak–Higashi syndrome (CHS), Griscelli syndrome (GS), and Elejalde syndrome (ES), which are rare autosomal recessive diseases presenting in childhood. They have similar skin and hair changes but differ with respect to the other clinical findings. **Aim:** This retrospective study was taken up to identify the spectrum of silvery gray hair syndromes diagnosed over a period of 10 years (2006-2015) in a super specialty pediatric institute and also to identify characteristic clinical and diagnostic findings. **Methods:** A total of 17 children diagnosed to have silvery gray hair syndrome over a period of 10 years (2006-2015) at Indira Gandhi Institute of Child Health Hospital, Bengaluru and were studied retrospectively. Complete blood counts, peripheral smear study, and microscopic examination findings of the hair were analyzed. Radiological findings were also considered. **Results:** The most common syndrome in the study was GS Type II (10 cases) followed by CHS (5 cases), with one case each of GS Type III and ES. **Conclusion:** This study emphasizes the importance of silvery gray hair as a clue to diagnose these rare genetic disorders with proper clinical examination and simple diagnostic procedures.

Key words: *Autosomal recessive, Chediak–Higashi syndrome, Elejalde syndrome, Griscelli syndrome*

Silvery gray hair syndromes consist of three conditions: Chediak–Higashi syndrome (CHS), Griscelli syndrome (GS), and Elejalde syndrome (ES). All these three disorders are rare autosomal recessive disorders presenting in childhood. All the three conditions present with similar skin and hair changes but differ with respect to immunological and neurological manifestations. These disorders are often fatal in childhood as a result of accelerated phase. Therefore, only a few patients live to adulthood. CHS and GS Type II predominantly present with immune dysfunction which may progress to hemophagocytic lymphohistiocytosis (HLH) in the absence of treatment [1]. CHS, GS Type I, and ES patients who live to adulthood have severe crippling neurological manifestations while GS Type III patients have only skin and hair changes without any immunological or neurological manifestations leading to their underdiagnosis [2]. Microscopic examination of the hair shaft and peripheral smear examination is diagnostic. Stem cell transplantation is the only curative treatment.

This study was undertaken to increase the awareness about these rare disorders. Till now, no comprehensive study has been undertaken to analyze these spectra of disorders in our country. We would like to highlight that these disorders can be easily diagnosed by a thorough clinical examination and simple investigations which will enable us to make an early diagnosis, improve the prognosis by initiating early treatment.

METHODS

A total of 17 children diagnosed with silvery gray hair syndrome over a period of 10 years (2006-2015) from Inpatient and Outpatient Departments of Indira Gandhi Institute of Child Health, Bengaluru were studied retrospectively. All the case files were retrieved, and all the relevant clinical details as well as the investigation findings were recorded. Complete blood count was done using Mindray BC-5200 5 part differential cell counter, and peripheral blood smear examination which was done to look for prominent granules in the leukocytes was taken into account. Biochemical investigations, done using fully automated Beckman Coulter analyzer AU480, were also analyzed. Hair shaft was examined by light microscopy to look for the melanin pigment distribution. Serum immunoglobulin levels, cytological, biochemical, and microbiological examination findings, whenever available, were also taken into account. Ultrasound scan, computed tomography (CT), and magnetic resonance imaging (MRI) findings which were mentioned in the case file were also noted. Statistical analysis was done by generating univariate frequency tables for the parameters.

RESULTS

A total of 17 children were studied. The age of the children ranged from 5 months to 8 years. Sex ratio observed was male:female=1:4.

All the children were born out of consanguineous marriage except one. All the children were born at term with delayed milestones in one of them. Out of 17 children, two of them were siblings (Fig. 1). Clinical features of all the children have been summarized in Table 1, and as it is clearly evident from the table, most of the children presented with symptoms of infection.

Eight out of 17 children had pancytopenia. Peripheral smear examination in five children showed prominent granules in the leukocytes suggestive of CHS. Bone marrow (BM) examination was done in all the children, out of which four each of CHS and GS showed features of hemophagocytic syndrome. In four children with CHS, BM also showed giant granules in myeloid series cells and in eosinophils. Hair shaft examination in all the children showed irregular large clumps of melanin, and in children with GS, the clumps were much larger in size than those in CHS (Fig. 2).

Table 1: Clinical manifestations of the children in the study

Clinical features	No of children
Silvery gray hair	17
Fever and recurrent respiratory tract infections	15
Multiple furunculosis	4
Hepatosplenomegaly	8
Bleeding diathesis	5
Ocular findings	5
Failure to thrive	3
Neurological symptoms	1

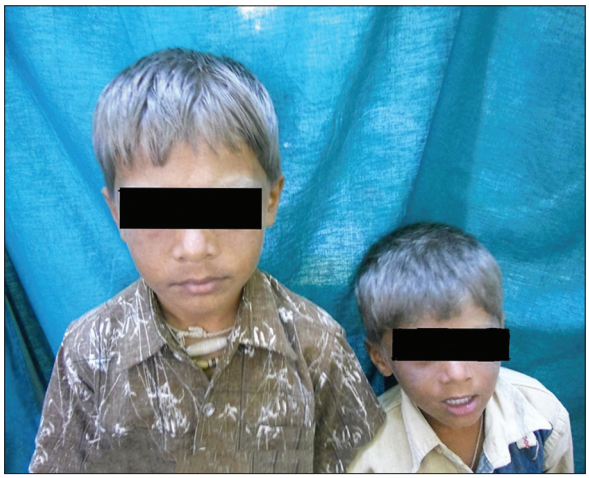

Figure 1: Two siblings with Griscelli syndrome Type III

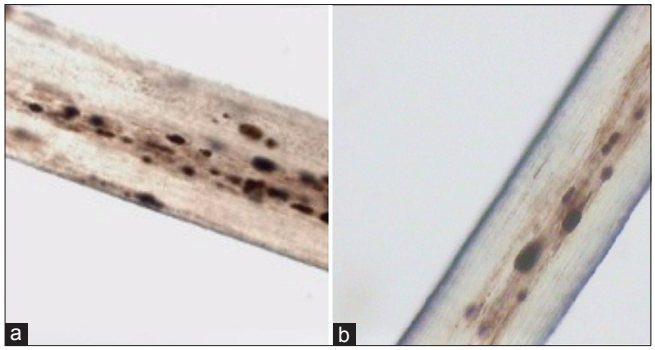

Figure 2: Microphotograph of the hair shaft showing irregular clumps of melanin in CHS & GS (X100)

CT and MRI were done in all the patients. Out of 10 cases of GS Type II, CT and MRI findings were normal in three children. In the remaining seven children, CT and MRI showed variable findings. In CT scan, two of them had findings suggestive of calcification (Fig. 3) and five children revealed findings suggestive of atrophy and cell infiltration (Fig. 4). MRI revealed areas of increased T2 signal intensity and abnormal enhancement in the subcortical white matter. CT and MRI findings were normal in one case of GS III. Out of five children with CHS, CT and MRI findings were normal in one case, and in remaining four cases, findings were suggestive of diffuse brain atrophy. In ES, CT scan revealed abnormal findings in the white matter, cerebellum, and gray matter with atrophy of the brain and MRI showed marked cerebellar atrophy. Summary of the investigations has been listed in Table 2.

Clinical diagnosis was made at the time admission on the basis of the clinical findings and laboratory investigations. It was found that GS Type II is the most common entity in the spectrum of silvery gray hair syndromes, followed by CHS, GS Type III, and ES as depicted in Table 3. Out of five children

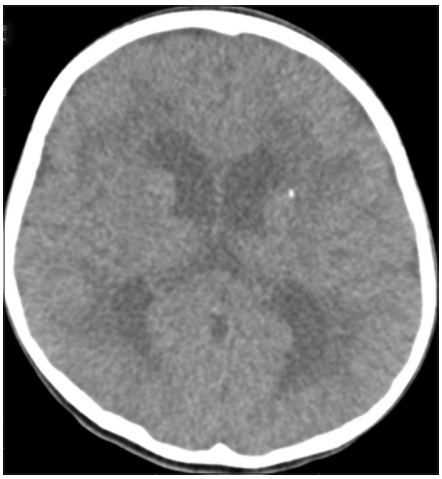

Figure 3: CT Brain (Plain) showing small calcification in the left basal ganglion region with mild ventricular dilatation

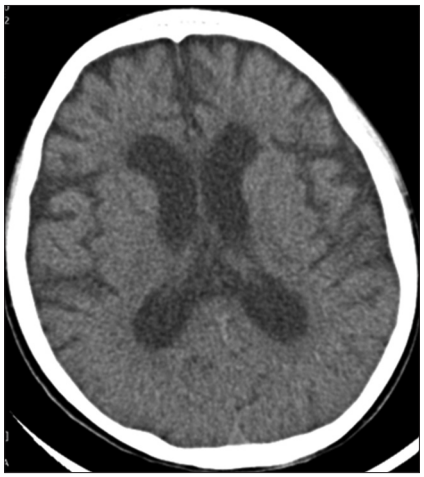

Figure 4: CT Brain (Plain) showing diffuse cerebral atrophy with mild ventricular dilatation

with CHS, two succumbed to recurrent infections and three were lost to follow-up. However, out of 10 children with GS Type II, four had presented in accelerated phase and were treated with immunosuppressive agents. Unfortunately, even with aggressive management, one child succumbed to infection and the other three children were lost to follow-up after discharge from the hospital.

Table 2: Investigation findings in the study group

Investigations	No of children
Pancytopenia on peripheral smear	8
Prominent granules in leukocytes	5
BM showing hemophagocytosis	8
Hair shaft showing irregular clumps	17
CT, MRI abnormality	12

CT: Computed tomography, MRI: Magnetic resonance imaging, BM: Bone marrow

Table 3: Final diagnosis in the spectrum of silvery gray hair syndrome

Silvery gray hair syndrome	No of children (%)
Chediak–Higashi syndrome	5 (29.5)
GS-I	0 (0)
GS-II	10 (58.9)
GS-III	1 (5.8)
ES	1 (5.8)
Total	17

GS: Griscelli syndrome, ES: Elejalde syndrome

Rest of the six children with GS Type II are on regular follow-up and are being treated with antibiotics and antiviral agents as and when symptomatic. One child with ES is on regular follow-up with symptomatic treatment. All the clinical features along with clinical diagnosis and management have been depicted in Table 4.

DISCUSSION

CHS is known to affect multiple systems of the body and is acquired by autosomal recessive inheritance. CHS is caused by mutations in a single gene characterized in 1996 as the lysosomal trafficking regulator localized to 1q42-43. To date, 40 mutations have been identified in the gene which include missense and nonsense mutations and small deletions and insertions in the coding region [3,4]. Throughout the world, <500 cases of CHS have been reported over the past 20 years [5,6]. 15 patients were diagnosed during a period of 11 years (2000-2010) in a nation-wide survey in Japan, indicating that one or two patients with CHS were diagnosed each year. There are only a few case reports from India [7-9].

Symptoms of CHS usually appear soon after birth or in children younger than 5 years, and the mean age of onset is 5.85 years. However, most of the patients die before the age of 10 years. In our study, the earliest age of presentation was 5 months, and the mean age was 2.68 years. Out of five children with CHS, four presented in the accelerated phase and two succumbed to

Table 4: Clinical diagnosis and management of children with silvery gray hair syndrome

Age	Gender	H/O consanguinity	Immunodeficiency	Neurological manifestations	Cytoplasmic inclusions	Hair shaft examination-clumps	Hemophagocytosis	Diagnosis	Outcome
8 years	Male	Yes	No	No	No	Large	No	GS III	Follow-up
1 years	Female	No	Yes	No	No	Large	No	GS II	Follow-up
6 months	Female	Yes	Yes	Yes	No	Large	Yes	GS II	Follow-up
5 years	Male	Yes	Yes	Yes	No	Large	Yes	GS II	Died
3 years	Female	Yes	Yes	Yes	Yes	Small	Yes	CHS	Died
1 years 6 months	Female	Yes	Yes	No	No	Large	No	GS II	Follow-up
1 years	Male	Yes	Yes	Yes	No	Large	Yes	GS II	Follow-up
2 years 7 months	Male	Yes	Yes	Yes	No	Large	Yes	GS II	Lost to follow-up
6 months	Male	Yes	Yes	No	No	Large	No	GS II	Lost to follow-up
7 years	Female	Yes	Yes	Yes	No	Large	No	GS II	Follow-up
11 months	Male	Yes	No	Yes	No	Large	No	ES	Follow-up
11 months	Male	Yes	Yes	Yes	No	Large	No	GS II	Lost to follow-up
10 months	Female	Yes	Yes	Yes	No	Large	No	GS II	Follow-up
5 years	Male	Yes	Yes	No	Yes	Small	No	CHS	Lost to follow-up
2 years	Female	Yes	Yes	Yes	Yes	Small	Yes	CHS	Died
5 months	Male	Yes	Yes	Yes	Yes	Small	Yes	CHS	Lost to follow-up
3 years	Male	Yes	Yes	Yes	Yes	Small	Yes	CHS	Lost to follow-up

the infection. Other three children were lost to follow-up. In our cases, unique finding was solitary giant cytoplasmic inclusion noted predominantly in the lymphocytes (Fig. 5), which is a rare occurrence. This is in contrast to the usual finding of the presence of such inclusion predominantly in the myeloid series cells and less commonly in the lymphocytes, according to the literature review.

Frequent bacterial infections and hemophagocytic syndrome are the major causes of morbidity. Fatal hemophagocytic syndrome is seen in 50-80% of patients with CHS, if untreated, which is also termed as accelerated phase. If BM transplantation is not carried out, most of the patients may die of hemophagocytic syndrome, but few patients may have relatively milder disease. Neurological manifestations such as polyneuropathy, parkinsonism, dementia, and ataxia characterize the adult form of CHS which is milder. CHS should be suspected in young adults, with a combination of these defects with oculocutaneous albinism or recurrent infections. A peripheral smear finding of giant inclusion in the leukocytes is diagnostic [10].

GS is a rare disorder acquired due to autosomal recessive inheritance, which results in pigmentary dilution of the skin and hair (silver hair) with the presence of large clumps of pigment in hair shafts and an accumulation of melanosomes in melanocytes [11]. Overall, three types of GS have been described based on clinical features and genetic mutations [12]. GS Type I manifests with primary dysfunction of central nervous system (CNS) characterized by Myosin Va mutation [13]. GS Type II commonly manifests with immunological abnormalities and develops HLH and is characterized by a defect in the Rab27a gene [14-16]. In HLH, T-lymphocytes and macrophages proliferate and infiltrate most of the organs, including the brain, leading to mortality. This immune abnormality can initially be controlled with immunosuppressive treatment, but stem cell transplantation is the only definitive curative treatment for this condition [17]. In GS Type II patients, the inability of lymphocytes and natural killer cells to lyse target cells is due to inability to consistently secrete the cytotoxic granules accounting for the severe immunological disorder, namely HLH [18]. GS Type III patients manifest with merely partial albinism with a defect in melanophilin gene [19].

In our study, a maximum number of children belonged to GS Type II, most of them had fever and upper respiratory tract infections, and one had pneumonia. 4 out of 10 had hepatosplenomegaly and BM examination revealed hemophagocytic syndrome. All four of them were recommended BM/stem cell transplantation, out of which one succumbed to infection and the others were lost to follow-up. One child who was diagnosed to have GS Type III was incidentally identified by noting her silvery gray hair and hypopigmented skin. The diagnosis was confirmed through hair shaft examination by light microscopy. This child did not have any neurological or immunological manifestations.

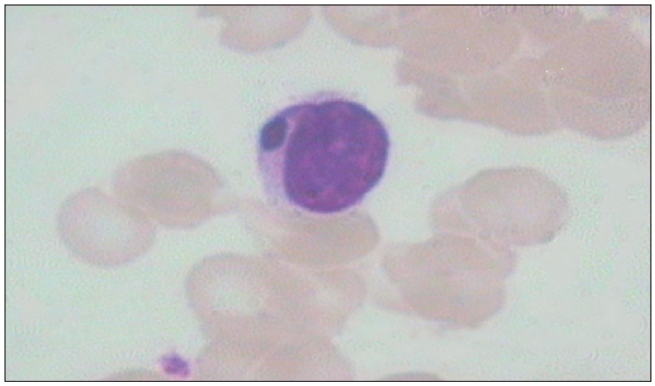

Figure 5: Microphotograph of the peripheral smear showing cytoplasmic inclusion in the lymphocyte (Leishman stain, X100)

Very few cases of GS have been reported so far from all over the world. Most reported cases are from the Turkish and Mediterranean population. However, in 2004, Manglani et al. [20] and Rath et al. [21] reported several cases from India. In most of the patients, diagnosis was done between the ages of 4 months and 7 years with the youngest occurring at 1 month [22]. In our study, diagnosis was made between the ages 6 months and 9 years. Rajyalakshmi and Chakrapani [23] have reported a case of GS Type II in a 4-year-old South Indian boy who presented in accelerated phase with features of immunodeficiency and secondary neurological manifestations caused by the involvement of the brain and also demonstrated lymphohistiocytosis in the lymph nodes. Verma et al. [24] have reported GS Type II in a 17-month-old boy who incidentally presented with severe anemia, which was due to parvovirus B19 infection, suggested by BM findings and later confirmed by DNA polymerase chain reaction.

ES was first described in 1977 [25]. It is also referred to as neuroectodermal melanolysosomal disease. It has been suggested that ES is the same disease entity as GS Type I which is caused by Myo Va gene mutation [26]. Still, the exact basis for ES remains to be defined [27]. Certain clinical features such as very early onset of neurological symptoms such as severe mental retardation, seizures, and severe hypotonia and tendency for intense tanning after exposure to the sun are used in differentiating GS Type I and ES. However, gene mutation specific to ES is yet to be defined [28]. ES is an autosomal recessive disease characterized by silvery hair and CNS dysfunction. Severe neurologic impairment either is congenital or develops during childhood. However, the function of the immune system is not impaired. ES is also featured by a wide spectrum of ophthalmologic abnormalities. Microscopic examination of the hair shaft reveals large granules of melanin which are unevenly distributed. Abnormal melanocytes, melanosomes, and abnormal inclusion bodies may be present in fibroblasts [29].

In the present study, only one child was diagnosed to have ES based on the clinical features of silvery gray hair, eyebrows and eyelashes, delayed milestones, and seizures at the age of 11 months. There was no immunological abnormality. CT and MRI findings were also supportive, and the child is on regular follow-up. The findings of hair shaft examination in CHS were the presence of uneven accumulation of large pigment granules. In GS, the hair shaft examination also revealed irregular large pigment granules, but relatively the size of pigment granules was larger compared to CHS. Sheela et al. [30] have also found that in GS, the size of clusters of melanin pigment is six times larger than in CHS. A skin biopsy specimen can be normal or may show

Table 5: Investigations which aid in the differential diagnosis of silvery gray hair syndromes

Investigations	GS	CHS	ES
Peripheral blood smear	No prominent granules	Prominent granules in leukocytes and giant organelles	No prominent granules
Light microscopy of the hair	Small and large clumps of melanin in irregular pattern	Small clumps of melanin in a regular pattern	Small and large clumps of melanin in irregular pattern
Histopathology of skin	Excess pigmentation of melanocytes at basal layer and scanty pigmentation in skin surrounding the pigmented areas	Large melanosomes in both melanocytes and keratinocytes	Irregular sized melanin granule dispersed in basal layer
Electron microscopy of skin	Mature melanosomes in melanocytes and to some extent in keratinocytes	Large melanosomes in both melanocytes and keratinocytes	Melanosomes at different stages of formation in the melanocytes

CHS: Chediak–Higashi syndrome, GS: Griscelli syndrome, ES: Elejalde syndrome

large melanin deposits and sparse dermal melanin.

The distinguishing feature of CHS on peripheral smear examination is the presence of giant intracytoplasmic granules in all granulated cells which is not seen in GS [31]. Although all the three silvery gray hair syndromes manifest with overlapping clinical and genetic findings, Table 5 helps in the differential diagnosis. Examination of hair from fetal scalp biopsy specimen and microscopic examination of the leukocytes from fetal blood samples aid in the prenatal diagnosis of silvery gray hair syndrome. Allogenic BM transplant/hematopoietic stem cell transplant is the only curative treatment, when done early, before the onset of HLH. Otherwise, the management is largely symptomatic. Once HLH develops, it is fatal [32,33]. There is also scope for prenatal diagnosis and counseling for these rare disorders.

Limitations of our study were its retrospective design and a small number of the patients. Second, we could not confirm the diagnosis by genetic analysis due to non-availability of the facility in our institution. Similarly, none of the patients underwent hematopoietic stem cell transplant as it was not available in our institution, and patients could not be taken up to higher center despite advice being given for the same.

CONCLUSION

We present the spectrum of silvery gray hair syndromes with their characteristic clinical and diagnostic findings in a tertiary care pediatric institute. The characteristic silvery gray hair of the patients is emphasized so that it is quite possible to suspect the diagnosis at the bedside and easily diagnosed by simple laboratory investigations such as hair shaft and peripheral smear examination.

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