

# Peeling skin syndrome in eight cases of four different families from India and Bangladesh

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**ABSTRACT**

Peeling skin syndrome (PSS) is a rare recessively inherited ichthyosiform genodermatoses characterized by superficial skin peeling. This has 2 subtypes, acral (APSS; OMIM 609796) and generalized form (OMIM 270300). The later has been subdivided into type A (non-inflammatory) and type B (inflammatory). Eight cases of peeling skin syndrome in 4 families were recorded over a period of 5 years. They were diagnosed clinically and confirmed histopathologically. Disease onset ranged from birth to childhood age (mean 5.25 ± 4.528 years) and age at presentation ranged from 7-35 years (mean 23.25 ± 10.471 years). Males outnumbered females (M:F - 5:3). All had non-inflammatory generalized disease of type-A PSS variety, except one who had type-B PSS. Two Muslim families (1<sup>st</sup> and 2<sup>nd</sup> family, total 5 patients) came from nearby country Bangladesh, and the 2 Hindu families were Indian. Higher severity over acral areas in generalized type, possible autosomal dominant pattern of inheritance and improvement with age as found in this series were new manifestations and possibly unreported previously. The disease was found to be poorly responsive to oral retinoids. Prevalence of the disease may be higher than expected. Importance of mutational analysis was also highlighted.

**Key words:** Bangladesh, India, peeling skin syndrome

**INTRODUCTION**

Peeling skin syndrome (PSS) or peeling skin disease (PSD) is a rare, recessively inherited ichthyosiform genodermatoses characterized by peeling of superficial skin. This condition was first described as a case report in 1921 by Fox who used the term ‘keratolysis exfoliativa congenita.’<sup>[1]</sup> Subsequently, many other terminologies were used to describe similar conditions like ‘deciduous skin’ (Bechet),<sup>[2]</sup> ‘familial continual skin peeling’ (Kurban and Azar),<sup>[3]</sup> and ‘continual skin peeling syndrome’ (Silverman *et al*).<sup>[4]</sup>

Clinically, PSS appears as ichthyosiform dermatosis with easy skin fragility that manifests as superficial skin peeling following minimal sliding forces. Severity is variable. Based on extent of involvement, this condition has been divided into acral PSS (APSS; OMIM 609796) and generalized PSS (OMIM 270300). Acral PSS was first described by Shwayder *et al*.<sup>[5]</sup> Generalized variety have been further subdivided on the basis of presence of signs of inflammation like erythema into 2 subtypes - type A (non-inflammatory) and type B (inflammatory).<sup>[6]</sup> Disease localized exclusively over face has been reported in a family.<sup>[7]</sup> Mevorah *et al* introduced a new subtype and described it as type C.<sup>[8]</sup> This subtype starts in infancy and is characterized by atopy, itching, and presence of circular erythematous patches that are encircled by areas of peeling.

In this series, 8 cases of peeling skin syndrome from 4 different families from India and Bangladesh are reported.

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**CASE REPORT**

Total 8 cases in 5 families were found (male- 3, female- 5). Age of onset varied from birth to early childhood (Mean  $5.25 \pm 4.528$  years). Age at presentation ranged from 7-35 years ( $23.25 \pm 10.471$  years). Two families (1<sup>st</sup> and 2<sup>nd</sup> family, 5 patients) came from nearby country Bangladesh. All those who were Muslims had a history of consanguinity in their parents. Three cases (3<sup>rd</sup> and 4<sup>th</sup> family) were of Indian origin and all were Hindus without any history of consanguinity [Table 1].

Demographic and disease details of the patients and their families have been mentioned in the Table 1 and some of these features were compared with 2 previous case series [Table 2].

All cases were of non-inflammatory generalized variety (PSS-A), except one (2<sup>nd</sup> family) who had prominent inflammatory changes (PSS-B).

Involvement of other family members with same disease was found in 2 families (1<sup>st</sup> and 4<sup>th</sup> family). In the 1<sup>st</sup> family (Muslim, consanguineous), PSS-A was present among 4 out of 7 siblings in one generation. There was no evidence of disease in the previous or subsequent generations. In the 4<sup>th</sup> family (Hindu, non-consanguineous), mother and daughter had PSS-A, but acral areas had higher severity than trunks. Partial or complete improvement in disease severity was reported by some members of the 1<sup>st</sup> family [Table 1]. The study was approved by the institutional ethical committee.

Details of each family are mentioned below:

**First family**

In this Muslim family from Bangladesh, 4 siblings out of total 7 were found to be affected. There was consanguineous marriage among their parents. Only one generation was found to be affected. The disease in this family was diagnosed as type A- PSS.

The eldest daughter (33 years) developed mild and localized truncal disease at the age of 10-12 years and improved completely after few years. She had no disease activity at the time of presentation.

The 2<sup>nd</sup> and 3<sup>rd</sup> sons developed the disease at around 8 years of age. In both of them, the disease improved greatly in subsequent years as mentioned by them. During examination, disease was detected only over the back and chest in both of them.

**Table 1: Profile of the patients and their families**

Family details	Position of pt in the family	Consanguinity	Family history	Sex	Age (yrs)	Age of onset (yrs)	Distribution	Seasonal variation	Erythema	Severity	Present status	Type
Family 1, Bangladesh, Muslim	1 <sup>st</sup> child	Yes	Yes, many members of single generation	Fem	33	15	Trunk,	Nil	Nil	Mild	Improved completely,	Type-A
	2 <sup>nd</sup> child			Male	30	4	Trunk,	Nil	Nil	Mild	Now less severe	Type-A
	3 <sup>rd</sup> child			Male	27	6	Trunk,	Nil	Nil	Mild	Now less severe	Type-A
	4 <sup>th</sup> child			Fem	25	Spared						
	5 <sup>th</sup> child			Male	20	3	Trunk, face, acral areas	Nil	Mild	Mod	No improvement	Type-A
	6 <sup>th</sup> child			Male	16	Spared						
	7 <sup>th</sup> child			Fem	13	Spared						
Family 2, Bangladesh, Muslim	1 <sup>st</sup> child	Yes	No	Male	22	Since birth	Trunk, acral areas	Yes, more in summer	Mild	Mod	No improvement	Type-B
	2 <sup>nd</sup> child			Male	18	Spared						
	3 <sup>rd</sup> child			Female	15	Spared						
Family 3, India, Hindu,	Single child	No	No	Male	10	2	Trunk, face	Yes, more in summer	Mild	Mild	No improvement	Type-A
Family 4, India, Hindu	Single child	No	Yes, two generations affected	Female	33	7	Palms, soles > trunk	Nil	Nil	Mild	No improvement	Type-A
	Mother			Female	7	5	Palms, soles > trunk	Nil	Nil	Mild	No improvement	Type-A

**Table 2: Demographic profiles of the patients with comparison with previous series**

	Sarma <i>et al</i> (India and Bangladesh) (n = 8)	Al-Ghamdi <i>et al.</i> (Saudi Arabia) <sup>[26]</sup> (n = 11)	Kose <i>et al.</i> (Turkey) <sup>[9]</sup> (n = 21)
Male-female	5:3	10:1	21:0
Average age of onset (yr)	5.25 ± 4.528 years	2.476 ± 4.643 years	1.545 ± 1.809 years
Onset at birth (no. of patients)	1	3	14
Age at presentation (yr)	7-35,	2-15	19-26
Type A (no. of patients)	7	4	6
Type B (no. of patients)	1	2	9
Acral (no. of patients)	0	5	6
Consanguinity (no. of patients)	5 (62.5%)	6 (54.5%)	10 (47.6%)
Positive family history (no. of patients)	6 (75%)	8 (72.7%)	10 (47.6%)

Most severely affected was the 5<sup>th</sup> son. Disease started to develop at around 3 years of age. He had extensive, asymptomatic, non-inflammatory peeling [Figure 1a]. Minor sliding force induced peeling. Almost all areas including face were affected. Palms and soles were thickened without any fissuring.

After routine investigations, systemic retinoid (isotretinoin) was advised only to the 5<sup>th</sup> patient for 3 months at the dose of 20 mg/day. It failed to show any satisfactory improvement.

#### Second family

One 22-years-old male born from a consanguineous marriage in this Muslim family from Bangladesh presented with skin peeling. None of his family members including his two other siblings were found to be affected.

Disease started as reddish morbilliform/ miliaria-like rash just after birth. Disease showed a temporary improvement during first 2 years but progressively worsened thereafter. Palmo-plantar hyperkeratosis appeared few years later and progressed rapidly. At presentation, he had persistent erythematous thick hyperkeratosis of hands and feet with transgradiens over dorsal surfaces. There was generalized easy skin peeling and prominent erythroderma [Figure 2]. Peeling occurred throughout the year, but severity increased noticeably during summer, after sweating or after contact with water for some duration. It was associated with bad body odor. Overall, skin appeared

dry and ichthyotic. The patient was diagnosed as having type B-PSS.

Oral acitretin (25 mg/ day) was given continuously for 2 months when it was stopped due to elevated liver enzyme. Only mild temporary improvement was noted.

#### Third family

This Indian non-consanguineous Hindu family had only one affected member who was a 10-year-old single male child. Disease started around 2 years of age and involved trunk, limbs, and face [Figure 1b,c]. Palms and soles were spared. Disease severity was higher in summer. There was no erythema or other signs of inflammation. It was classified as type A- PSS.

He was treated with acitretin (25 mg/ day) for 3 weeks and then 10 mg/day for 6 weeks, but it failed to show any benefits.

#### Fourth family

In this Hindu family, without any parental consanguinity, mother (age 35 years) and her only daughter (7 years) presented with peeling of thin skin, mostly from acral areas along with mild involvement of back. [Figure 1d]. No other family members were known to be affected.

The disease started around 5 years of age in both of them. Palmar skin showed persistent peeling throughout the year, but truncal areas had recurrent peeling during summer. The disease was asymptomatic and there was no erythema or bulla. It was diagnosed as type A- PSS. These patients could not be treated as they were lost early to follow-up.

Pedigree diagrams of 1<sup>st</sup> and 2<sup>nd</sup> family have been depicted in Figure 3. None of the patients had any hair, nail, and dental abnormality. Systemic examination did not reveal any abnormality in any of the patients. Routine systemic investigations like complete hemogram, liver function and kidney function tests, thyroid profile were normal in all. Any further metabolic and biochemical profile evaluation was not attempted in any of them.

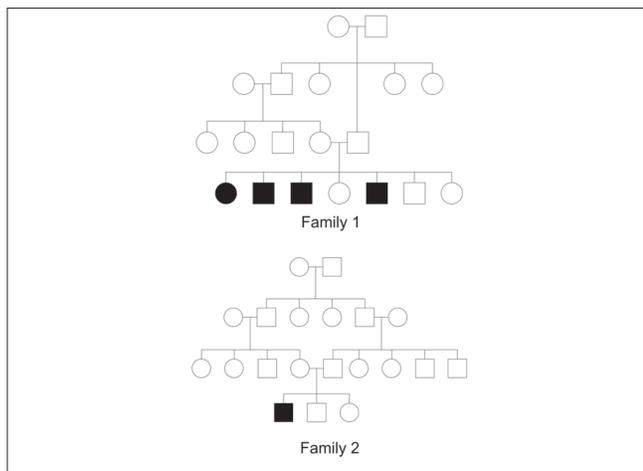
Histology was done from the affected areas in one member from 1<sup>st</sup> 3 families (1<sup>st</sup> family - 5<sup>th</sup> child, 2<sup>nd</sup> family - 1<sup>st</sup> child, 3<sup>rd</sup> family - Single affected child). [Table 2].



**Figure 1:** (a) Extensive peeling that developed following slight sliding force on trunk in a Muslim boy aged 10 years (1<sup>st</sup> family, 5<sup>th</sup> patient). (b, c) The young Hindu boy showed presence of superficial peeling over sides of cheek (3<sup>rd</sup> family). (d) Persistent superficial peeling of acral areas since early childhood in the Hindu mother (4<sup>th</sup> family)



**Figure 2:** Thick keratoderma with transgradiens over hands (a) and feet (b) along with prominent erythema (2<sup>nd</sup> family, 1<sup>st</sup> patient). Lower leg showed diffuse erythema and skin peeling. Area over back of the same patient showed erythroderma and superficial peeling after mild rubbing (c)

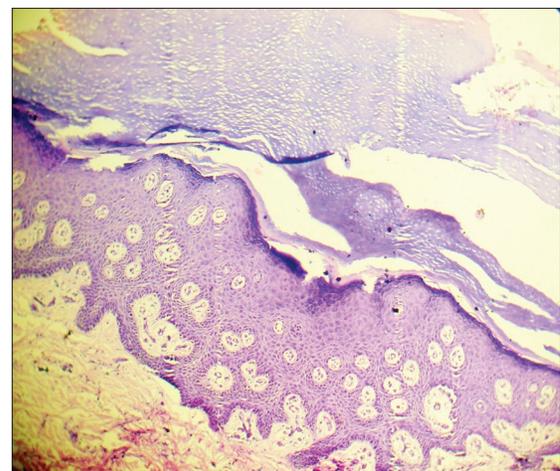


**Figure 3:** Pedigree diagram of 1<sup>st</sup> and 2<sup>nd</sup> family

Histology in cases of type A-PSS revealed presence of orthokeratosis and intra-corneal cleft of lower part of stratum corneum in patients. In patient with type B-PSS, prominent hyperkeratosis and acanthosis and distinct sub-corneal split was detected [Figure 4].

**DISCUSSION**

Diagnosis of PSS is often delayed due to late presentation in less severe and asymptomatic cases. For same reasons, misdiagnosis is also frequent. These factors might contribute to the apparent rarity of this



**Figure 4:** Photomicrograph (H and E stain, x400) showing histology of skin from back in patient with type B PSS (2<sup>nd</sup> family- 1<sup>st</sup> child) demonstrated hyperkeratosis, acanthosis, and sub-corneal split

condition. Many cases in the current series were diagnosed in adulthood despite a childhood onset. The patients in 4<sup>th</sup> family presented only when two successive generations developed the same problem. With this report, prevalence of the disease in this part of the globe appeared to be higher than predicted from previous isolated case reports.

PSS has clinical resemblance to subcorneal pustular dermatosis, epidermolysis bullosa simplex superficialis, congenital ichthyosiform erythroderma, Netherton syndrome, pemphigus foliaceus, erythrokeratoderma like erythrokeratolysis hiemalis (Oudtshoorn disease), keratolysis exfoliativa and even exfoliation following dyshidrosis.<sup>[9]</sup> However, differentiation from most of these diseases can be done with almost certainty only on clinical ground supported by histological findings. Netherton's syndrome is characterized by presence of atopy, trichorrhexis invaginata ("bamboo hairs") and is caused by SPINK5 (MIM 605010) mutations,<sup>[10]</sup> which is different from the known mutations of PSS. Epidermolysis bullosa superficialis is characterized by presence of bulla, dominant mode of transmission, absence of spontaneous peeling, and onset before 2 years.<sup>[11]</sup>

Histological features are characteristic in PSS. Type-A shows orthohyperkeratosis while type B shows a psoriasiform pattern. Separation within keratinocyte at the junction of stratum granulosum and stratum corneum (Levy and Goldsmith, 1982)<sup>[12]</sup>, (Brusasco *et al*, 1998)<sup>[13]</sup> is considered unique and can differentiate PSS from all forms of epidermolysis bullosa (EB) where the separation is at the level of basal keratinocyte (EB simplex type), dermo-epidermal junction (junctional EB), and superficial dermis (dystrophic EB). Although EBS superficialis has similar level of cleavage or separation in the epidermis, they can be distinguished clinically. In this series, patients with non-inflammatory generalized disease (type A) showed presence of orthokeratosis and intra-corneal cleft at lower part of stratum corneum. Patient with type B PSS had prominent hyperkeratosis, acanthosis, and sub-corneal split. These findings corroborated with classical findings and were helpful in confirmation of the diagnosis.

Rare associations have been reported with fragile hair and koilonychia, distal onycholysis, chapping, keratoderma, sexual dysfunction, anosmia, short stature, primary amenorrhea, poor development of sexual characters, cheilitis, keratosis pilaris, melanonychia, clubbing and hyperhidrosis.<sup>[9]</sup> Abnormal biochemical parameters like altered tryptophan levels, aminoaciduria,<sup>[14]</sup> high serum copper level, higher IgE and higher ceruloplasmin, iron and iron-binding capacity, and abnormal epidermal retinoid metabolism are also reported.<sup>[15]</sup> As

mentioned, extensive investigations to detect any rare biochemical alterations were not done in this study.

PSS is known to have an autosomal recessive transmission. New mutations involving profilaggrin/filaggrin gene causing poor aggregation and disruption of keratin is also reported to cause PSS.<sup>[16]</sup> Ultrastructural studies have detected abnormal and cribriform keratohyalin granules, reduced desmosomal plates, intracellular cleavage, and intercellular electrondense globular deposits in the stratum corneum.<sup>[6,4,16]</sup> For acral PSS (APSS), the genetic defect has been mapped to the 10.2-Mb region on chromosome 15 (at 15q15.2)<sup>[17]</sup> that contains transglutaminase (TGM) gene cluster (containing TGM5, TGM7, and EPB42). TGM5 is expressed in the skin between granular layer and the stratum corneum and this gene is now known to play the key pathogenic role in APSS. TGMs are extremely important in terminal differentiation of the epidermis where it assists in cross-linking of keratins and structural proteins like involucrin, loricrin, filaggrin, and forms the cornified cell envelope<sup>[18]</sup> through formation of gammaglutamyl- lysine isodipeptide bonds between adjacent polypeptides.<sup>[19,20]</sup> The mutation in PSS-B involves corneodesmosin (CDSN) gene as found in two different studies.<sup>[21,22]</sup> Corneodesmosome maintains the structural integrity of stratum corneum. For PSS-A, the affected genes are yet to be found out. In this series, no mutation analysis was performed because of infrastructural limitation. Evaluation of clinico-histological parameters appeared to be sufficient enough to confirm the diagnosis in all cases.

There has been a male preponderance in all the reports including the current one. However, we have found a lesser gap in sex ratio [Table 1].

In addition to presenting the case series on this rare disease, this present report highlighted few important yet previously unreported aspects of the disease.

Permanent improvement with age (many members of 1<sup>st</sup> family) or temporary improvement for some years in the very early stage before a progressive course as seen in the patient with type B PSS (1<sup>st</sup> patient, second family) is *hitherto* unreported.

Acral disease is known to be rarer, categorized as a separate entity, has specific mutation and develop without any involvement of other non-acral areas.<sup>[5,23]</sup>

On the contrary, acral involvement can be sometimes present in generalized type where the severity in acral areas is generally milder than non-acral areas.<sup>[24]</sup> One of the cases in this present series with generalized disease of type-A pattern had higher severity in acral areas than in trunk. This pattern was neither reported previously nor can be explained with currently available knowledge.

So far, only autosomal recessive mode of transmission was known for PSS. However, this cannot explain the involvement of mother and her daughter (4<sup>th</sup> family, Hindu). Possibility of autosomal dominant mode of transmission cannot be ruled out. Unfortunately, these particular patients were lost early to follow-up even before histological evaluation or genetic confirmation could be attempted.

Retinoids failed to show any significant benefit in any of the cases this series. This supported the previous observations where methotrexate, retinoids, and steroids were found to be ineffective.<sup>[25]</sup>

Only two case series are published so far in the world literature; one from Saudi Arabia<sup>[26]</sup> and one from Turkey.<sup>[9]</sup> In 2011, Kose published the so far largest case series of 21 cases from Turkey.<sup>[9]</sup>

Some isolated cases of PSS has been reported from India.<sup>[10,27-35]</sup> Total number of cases reported so far is 81 and it consisted of 20 cases of type A PSS, 27 cases of type B PSS, 2 cases of type C PSS, 21 cases of acral PSS, and 11 of undefined phenotypes. In 2010, two separate groups published single case report and reviewed the literature in the same issue of one journal.<sup>[25,27]</sup> Kharfi *et al* in their review of world literature found 51 cases till then, including their own single case of type A PSS.<sup>[25]</sup> Garg *et al*<sup>[27]</sup> authored a single case report and reviewed the Indian literature. The present series of 8 patients of PSS is the first case series from south-east Asian countries.

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