The article by Pradhan\textsuperscript{[1]} on the description of the diamond sign in dysferlinopathy is refreshing and interesting. In the modern times, medical advances have moved away from the bedside, into laboratories which...
use complex gadgets to probe the depth of scientific problems. More and more time is being spent by medical practitioners huddled around the radiological view boxes and with instruments, discussing the problems of their ‘i patients’ from the files, while the real patient gets only a brief and often cursory examination.

In this context, returning to the careful clinical observations, as Dr Pradhan[1] has so elegantly and repeatedly shown, is very helpful. Starting with the valley sign in Duchenne Muscular Dystrophy [DMD],[2,3] he has been bringing out peculiarities of clinical features of various muscular dystrophies encountered in India. The valley sign is particularly useful in patients of DMD who are examined late in the disease course when the calf hypertrophy has disappeared. The poly hill sign delineates the specific hypertrophy and atrophy of the muscles around the shoulder, helping the diagnosis of Facio scapuloperoneal muscular dystrophy [FSD].[4] The shank sign in myotonic dystrophy[4] helps to differentiate it from other myotonic syndromes. The diamond sign recently described by him[6] is a useful indicator of the subgroup of patients with deficiency of dysferlin. The specificity and sensitivity of the sign need further work, as he mentions in the paper. At this stage, perhaps a note needs to be made on the control population which comprises of patients having FSHD and myotonic muscular dystrophy which have a very different pattern of muscular involvement.

It is important to put the role of clinical signs in perspective of molecular mechanisms of disease. The observation of the hip abduction sign in limb girdle muscular dystrophies is an example. Initially, I felt the sign is seen in sarcoglycanopathies alone,[5] only to observe later that this pattern of muscular weakness is shared by more limb girdle muscular dystrophies. The clinical observation is relevant because it points to inherited myopathies, probably dystrophies, narrowing down the differential diagnosis. In some ways, a corollary could be drawn with the hereditary spinocerebellar degenerations and slow eye movements. Slow eye movements direct us to the inherited nature of the process but do not give a specific genotype. The genotype phenotype correlation is proving to be very complex in myopathies and it is well known that one gene can exhibit many phenotypes and vice versa. We can not expect clinical signs to accurately predict the genotype, but they can certainly lead the clinician in the right direction of inquiry and minimization of investigative resources. To this effect these observations by Dr Pradhan have great merit.

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