Splinter haemorrhages: facts and fiction

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Ritual inspection of the hands may reveal much useful clinical information. One readily identified sign is the finger nail splinter haemorrhage (SH). However, not all splinters are the same. Though most are dark, distal and painless, others are red, proximal and painful. They may occur singly or in clusters and the pattern of distribution is variable. Splinter haemorrhages are therefore unlikely to represent a single clinico-pathological entity. They often arouse clinical interest but their interpretation is frequently uncertain. Standard medical texts variously infer that splinter haemorrhages are useful, sometimes useful or seldom useful as an indicator of systemic disease. What is the basis of this uncertainty, and are splinter haemorrhages important as a physical sign?

Historical background

Infective endocarditis

The first description of splinter haemorrhages is ascribed to Sir (later Lord) Thomas Horder. He noticed ‘... a minute petechia, in the form of a vivid linear splash of red at the side of the bed of a finger nail...’ in two patients with infective endocarditis [1]. He later added that they were exquisitely tender, inferring that they were akin to an Osler’s node arising beneath the nail plate [2]. The sudden painful onset of these lesions caused these patients to examine their nails carefully to ensure that a thorn or splinter had not caused the sensation. The term 'splinter haemorrhages' was first coined by Blumer [3]. He described two patients with subacute bacterial endocarditis (SBE) who had painful linear haemorrhages underneath the distal part of the nail plate, looking exactly like wood splinters under the nails.

Blumer found splinters in only two out of 48 patients with SBE. Other workers have also noted that SH were uncommon in this disease. Wedgwood [4] found SH in only four of 65 cases of SBE and later surveys showed that they occurred in only 13 percent of patients [5,6]. Although one report regarded SH as ‘the commonest superficial evidence of embolism’ [7], several major surveys of SBE published over the last four decades covering over 1300 patients, made no mention of SH [8-11].

Nonetheless, subungual SH have come to be considered one of the classical signs of bacterial endocarditis [12]. Thomas Lewis [13], whilst acknowledging that SH occurred in endocarditis, thought that their clinical importance was diminished by their frequent appearance in many other conditions.

Other disorders

Diverse diseases have been linked with SH. The current edition of French’s Index of differential diagnosis [14] lists six systemic diseases, and trauma, as causative factors. Other putative associations have been reported (eg peptic ulcer, hypertension, anaemia, neoplasia), but there is no evidence to substantiate them.

In other conditions, the evidence rests on limited case reports from which it is difficult to judge the closeness of the association. For example, Fraga and Mintz [15] reported SH in four patients with systemic lupus erythematosus and suggested that the sign might be a useful marker for disease activity. However, they cautioned their conclusion by stating that ‘splinter haemorrhages have been carefully looked for in all of our patients with SLE for the last two years and no more cases have been found.’

The greatest difficulty when evaluating the usefulness of splinter haemorrhages in individual patients arises from the high background frequency of the sign. Surveys have shown SH to be present in 26-56 percent of healthy subjects [16, 17] and a wide ranging but high frequency of 19-60 percent has also been found in hospital patients [14, 18-24]. In the hospital studies, no definite disease associations were identified. In several of the studies, infective endocarditis was specifically looked for, but no relationship with SH emerged.

Pathogenesis

The observation that SH are carried forward with nail growth indicates that they are formed and contained within the epidermis of the nail bed. When they have moved into the free margin of the nail they can be scraped away and give positive colour tests for altered blood [21, 25].

Fastidious self-documentation for six months of the
incidence, site and movement of SH in a trained observer’s digits [25] reliably demonstrated that SH resulting from trauma arise in the distal third of the nail, in the region where the nail plate separates from the nail bed. Immediately beneath the surface of this region are delicate specialised spiral capillaries. Detailed histological studies of SH in cadaver specimens have revealed that it is the rupture of these vessels which produces the SH [26]. There was no evidence for emboli in the microcirculation of the cadaver specimens examined. In view of the close proximity of the thin walled vessels to the surface, minor trauma to the nail is the likely cause of capillary damage. Leverage, whereby the nail plate is pushed away from the underlying nail bed, would injure the distal part of the nail where it is least supported [26]. Evidence for trauma as a common causative factor for SH is inferred from several surveys. The evidence is mostly indirect but is consistent and compelling:-

1. Splinters are common in the finger nails but rare in toe nails (which are protected by shoes) [25].
2. Splinters predominantly appear in the right thumb and index finger [17,21,22].
3. Splinters are more frequent in manual workers [18,19,22].
4. Splinters are commoner in men [16,21,22].
5. Splinters occur in sports involving jarring hand trauma [16,21,27].
6. Splinters are associated with the use of walking aids in the elderly [23].
7. Splinters become less common the longer a patient stays in hospital [18].

Although trauma is the commonest cause, increased capillary fragility, microemboli and capillaritis have also been advanced as possible mechanisms to account for SH. Increased capillary fragility is the least satisfactory explanation because capillary integrity assessed by Hess’s test is normal in patients with SH [18,27], and because they are not a feature of generalised purpura [28].

In some instances multiple small emboli may be a more plausible explanation than trauma. For example, unilateral SH have followed the insertion of indwelling radial artery catheters [29]. The high frequency of SH in patients with a recent major arterial embolus (86 percent) and in mitral stenosis (44 percent) would also support an embolic origin for the lesions [27]. However, in the latter group, there was the paradoxical finding that SH were independent of the presence of atrial fibrillation, and this must temper the embolic proposal.

Possible immunological mechanisms for SH have been inferred by their apparent association with rheumatoid arthritis [30] (where the SH were unrelated to the activity of the disease); systemic lupus erythematosus [15] and polyarteritis nodosa [31]. However, SH are only an occasional feature of these conditions: other surveys of the cutaneous manifestations of rheumatoid disease [32,33] and of polyarteritis nodosa [34,35] make no mention of them. Until we have further data the role of immunological or inflammatory processes in the genesis of SH must remain speculative.

The patterns of nail splinters in specific conditions

If SH can be produced by different mechanisms in a variety of diseases it is possible that the site, size, shape, distribution and other features of SH may be characteristic.

Trauma

Traumatic nail haemorrhages are usually linear but can be punctate [25]. They occur distally and are 1–3 mm long [21,27] with an average length of 1.5 mm. Some people have single splinters but the mean is 2.5 per person [19]. The patient is not usually aware of their presence [27] — unlike the symptomatic splinters of trichiniasis and arterial embolism. Traumatic splinters are plum coloured but after a day or two turn brown and then black [26,27]. They do not fade but grow distally out of the nail.

Infective endocarditis

Horder’s first description was of patients with linear ‘splash’ at the side of the bed of a finger nail. The SH arose suddenly ‘with sharp pain, as though the finger had been pricked’ [2]. Blumer [3] also commented on the soreness of the fingers of patients with SBE who had SH, but argued that Horder’s description did not correspond to the distal linear haemorrhages that he had observed, which simulated precisely the appearance of a splinter of wood.

Although it is often taught that proximal splinters are more suggestive of SBE, there are no published data to confirm this. Indeed, very few surveys have documented the site of the splinters. In Dawling’s account [19] of 200 inpatients with SH, seven (2.1 percent) had SH in the proximal third of the nail but the author does not mention whether any of these patients had SBE. One of the four patients who did have SBE also had an Osler node in the pulp of the affected finger. He developed fresh subungual petechiae whilst in hospital. A photograph of this patient’s nail shows a large SH in the distal third of the nail and two other minute SH in the middle third. There are no proximal SH.

The association of SH and Osler nodes in occasional patients with SBE suggests that SH may indeed sometimes occur as a result of this condition. We do not know whether the clinical features of SH in SBE are distinguishable from those produced by trauma. Until the picture is clearer, it seems prudent to continue to document the presence, number and site of SH on admission to hospital and to consider SBE in those who develop fresh SH in the absence of trauma [19,36,37].

Indwelling arterial catheters

Splinters in the nails of one hand sometimes occur in patients who have had catheters inserted into the radial or brachial arteries. Multiple splinters have been observed distally in the nail beds after the insertion of a brachial artery cannula [29]. These may occur after non-infective arterial puncture [38].
A report of splinters occurring downstream from a radial artery catheter infected with *Staphylococcus aureus* [39] is of particular interest. Arcades of subungual splinters occurred in the thumb, ring and forefingers. The onset was rapid, six days after insertion of a teflon arterial catheter, in a setting where there could be no trauma to the hand. The splinters occurred at the same time as an Osler’s node in the right ring finger and Janeway lesions on the dorsum of the hand. No lesions were found elsewhere and there were no signs of endocarditis. At subsequent operation a large septic thrombus was found in a false aneurysm. This temporal association of splinters with an Osler node and Janeway lesions support the belief that splinters may indeed be a direct result of infected emboli.

**Trichiniasis**

Splinters occurring in the larval migrating phase of infestation with *Trichinella spiralis* are said to be transverse [40]. This belief has arisen because of misinterpretation of one of the first descriptions of splinters in this disease. Sheldon [41] discovered that 10 per cent of patients with trichiniasis had ‘a peculiar type of haemorrhage along the distal end of the nail bed, following the curve of the nail’. This broke up into parallel lines ‘looking as if a number of black splinters had got under the nail.’

Splinters in fingers and toes of patients with active trichiniasis were mentioned in an earlier paper, in which a photograph clearly shows longitudinal splinters forming an arcade across several finger nails [42]. Splinters in trichiniasis are associated with soreness at the tips of the fingers. They are initially red, then plum coloured, and finally black. They are 2 mm wide and 4–5 mm long [41] and up to six may be seen in a single finger [42]. They are not transverse.

**High altitude**

Chronic mountain sickness (Monge’s disease) is a complex condition which predominantly affects young and middle aged men who live at 3000 m or more above sea level. In this condition (which has haematological, cardiovascular, respiratory and neuropsychiatric features) distinctive splinter haemorrhages occur throughout the nail including the base [43]. Nail haemorrhages in this disorder are of both splinter and dot configuration, and large numbers can occur in a single nail. Heath and Williams postulated that these nail haemorrhages are associated with a raised haematocrit [43].

Haemorrhages in the nails of those living on high mountains differ from those developing in lowlanders who climb to high altitude. One mountaineer spontaneously developed numerous splinters on an expedition to Nepal. He counted 46 splinters (11 of which were beneath each thumb nail) which were thin, red, longitudinal streaks arranged in a ‘corona’ near the distal part of the nail bed. The haemorrhages were 0.5–4.0 mm long, and when they grew out were found to be in the soft thin layer on the undersurface of the nail [44]. These haemorrhages differ from those occurring in trichiniasis in that they seem to arise painlessly, are present in larger numbers, and are narrower. They may be a result of a rising level of haemoglobin and repeated trauma [43].

**Discussion**

This review of the pattern of splinters in different conditions shows that the term ‘splinter haemorrhage’ holds no pathogenetic or diagnostic information. Although the words are perfectly descriptive, splinters cannot be considered an entity. The term splinter haemorrhage is so clinically convenient that it is unlikely to be displaced by more specific pathological terms. It is uncertain how far the anatomy of the subungual vessels determines a single appearance from a variety of pathological procedures. However, it may be important to discriminate between the different patterns of these lesions. We would therefore urge investigators to make more detailed reports of SH in different medical conditions. In particular, it might be noted whether splinters are proximal or subterminal, single or multiple, arcuate or random in their distribution, painful or painless at onset. Future accounts could usefully include photographs as well as descriptions of the size, site, shape, colour and number of SH, as well as details of associated clinical features.

SH can be useful in alerting one to the possibility of a diagnosis. They are contributory but not confirmatory evidence of a pathological process.

**Conclusions**

SH are common and the main cause is trauma. Most people with traumatic SH are not aware of their presence; those few patients with SBE, trichiniasis and arterial catheters who develop splinters may notice pain under the nails. A small proportion of patients with uncommon diseases exhibit specific patterns of splinter haemorrhages. The long-held belief that transverse splinters occur in trichiniasis is erroneous. The notion that proximal splinters are indicative of SBE has yet to be confirmed.

It is nearly 60 years since SH were first recognised: the task of defining their origin and usefulness is a contemporary challenge.

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