# Hypergranulation tissue: evolution, control and potential elimination

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## Introduction

Granulation tissue typically consists of an abundance of blood vessels mixed with fibrous connective tissue. Granulation tissue grows from the base of a wound and is able to fill the wound, facilitating eventual epithelialisation and wound closure. Continued wound healing will only take place once internal inter- and intracellular signalling notify keratinocytes and epithelial cells that the tissue is ready for their cellular migration.[1-4] Prolonged stimulation of fibroplasia and angiogenesis results in hypergranulation, a potential problem for the wound healing process.

## Pathophysiology

Granulation tissue is light red or dark pink in colour, with a profusion of often friable capillary loops. This results in an overgrowth of clustered, cobblestoned, bleeding material with a red, raised, glistening surface. It is moist and soft to the touch. It appears uneven and is usually raised from the base of the wound bed. It may be present in a cavity (often foreign material, sutures, etc) or in a wound tract (pilonidal sinus-type of situation). Aside from the bleeding that is commonplace, the patient may complain of disproportionate pain. The make-up of granulation tissue is essentially that of immature proliferative tissue consisting of an extracellular matrix of proteins, such as collagen type III, a precursor to type I collagen which replaces it as the tissue matures.[3-5] The chief cellular components are macrophages, neutrophils and fibroblasts which normally phagocytose older, dead and foreign tissue material, aiding in the defence against septic invasion. This is especially important as the skin has been breached and no longer has the immune benefits of an intact stratum corneum. To eliminate the cellular waste products and to effect cell renewal following apoptosis, blood vessel networks are established, forming an integral and dominant component of the granulation tissue. The fibroblastic component of the cellular make-up controls the deposition of this granulation tissue and the synthesis of collagen components – thus it controls formation of the extracellular matrix.[3,4]

Hypergranulation prevents epithelialisation and the healing process is arrested. The point at which hypergranulation tissue replaces normal healthy granulation tissue has not been clearly defined, but a convenient temporal definition would be when epithelialisation stops and the healing process is halted. This is directly as a result of the nature of the hypergranular tissue which impedes epithelial migration, either by virtue of the constituent change of the tissue itself physically impeding epithelial movement (raised rolled overgrowth), or as a result of changes in extracellular signalling 'switching off' movement of epithelial cells.[5-7] This exact mechanism has yet to be defined.

The 'trigger point' for this transformation is also unclear, but certain common background characteristics have been identified. Moist areas from exudates or bleeding which are also subject to prolonged physical irritation or friction with continued repetitive minor trauma or pressure appear to be particularly vulnerable; excessive inflammation, bacterial bioburden (critically colonised wounds), undercurrent infection, and a possible new scenario of negative pressure suction with microdeformation particularly applicable to large pore foam dressings constitute these background characteristics.[5-8] Additionally it has been suggested that low oxygen levels and high moisture can stimulate granulation tissue formation and, for this reason, some companies advocate the use of occlusive dressings to encourage granulation tissue formation. The converse approach has therefore been used to treat hypergranulation – vapour permeable dressings (increased oxygen) with low moisture (absorbent dressing) to replace any occlusive dressing that may have been used.[6,7,9-11]

The obvious factors that often herald the start of hypergranulation tissue need to be looked for: increased exudate volume; infected or critically colonised (?biofilm) wound; location of the wound (umbilicus, stoma, and pilonidal sinus often indicate epithelial tracts or hidden foreign materials or hair); concomitant treatment (negative pressure, totally occlusive dressings, ill-fitting dressings or garments).

Conventional treatment regimens involve:

1. Silver nitrate: a caustic technique that is extremely destructive, and when activated, will oxidise organic matter, coagulate tissue and destroy bacteria; tissue dies almost immediately.[6-12] Unfortunately this sets up further inflammation and exudate formation and if ancillary treatment methods are not instituted

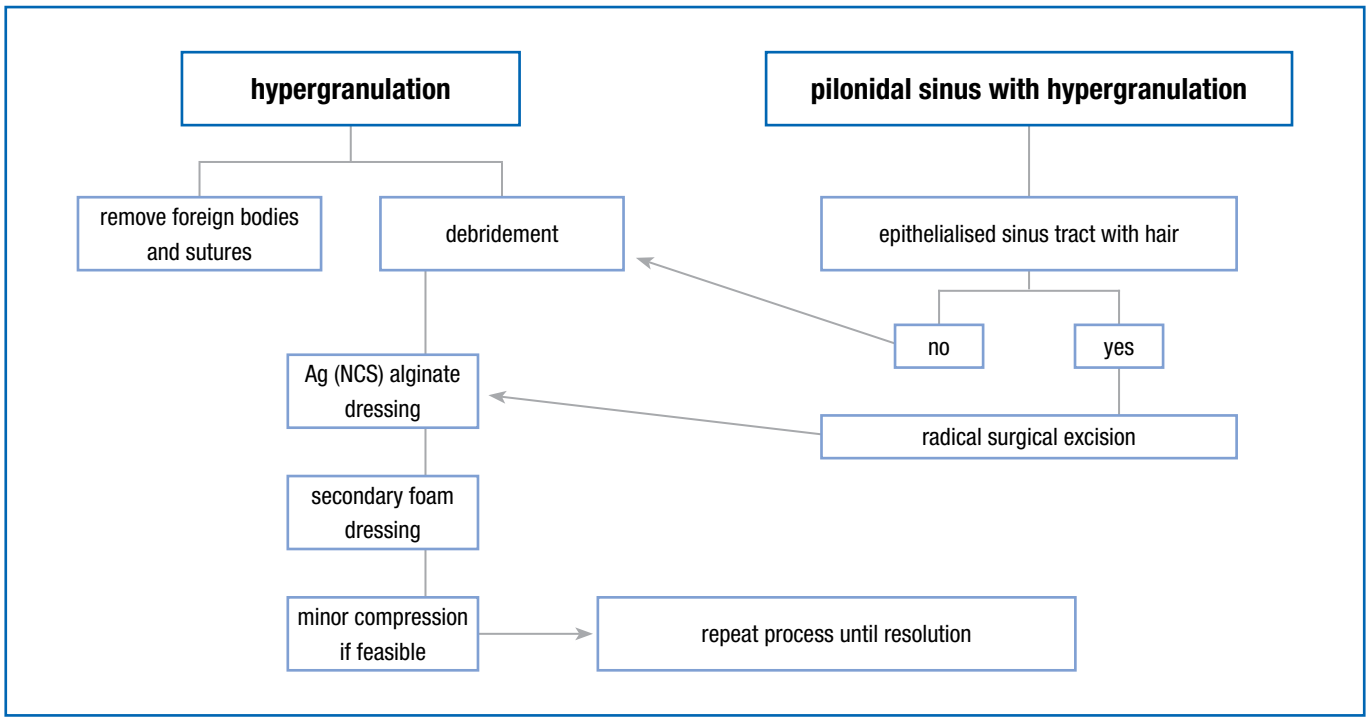


**Figure 1:** Algorithm for management of hypergranulation tissue

(exudate management, infection control, minor compression to avoid friction) the condition is liable to recur. One must also be sure to exclude foreign materials, such as sutures, being the primary cause of hypergranulation. If the material is not removed, no treatment modality will work. Thus silver nitrate may be acceptable as a part of the debridement routine rather than a treatment solution.[6-11]

2. Low dose cortisone cream (Kenalog®, Pevisone®) to promote collagen breakdown.[5,10,13] Topical corticosteroids are not approved or indicated for open wounds or hypergranulation tissue.[13] This method of treatment is rarely successful. Impregnated steroid tapes have been used successfully to eradicate hypergranulation tissue – unfortunately problems with recurrence are still valid.[8]
3. Vapour permeable dressing: non-occlusive,[6] with light pressure application. A foam dressing (Lyofoam®) resulted in a reduction of hypergranulation tissue within a two week period in one study. This was considered a combined result of a non-occlusive, moisture vapour permeable, absorbent dressing.[6]
4. Hypertonic NaCl dressing products, such as Mesalt® or Curasalt®, use oncotic pressure to promote drying by managing exudates, promoting movement of fluid away from the wound and reduces tissue oedema.[6–13] Neither method (foam, hypertonic saline) directly deals with the inflammation or potential bacterial component.
5. Surgical or sharp debridement of the area is extremely successful at removing the hypegranular tissue, but not successful at preventing recurrence and does require the clinician to have skill and competence in wound debridement.

## Multitarget approach to hypergranulation tissue removal and prevention of recurrence

Based on the pathophysiology of hypergranulation tissue as described above, there appear to be a few common background wound circumstances that need to be addressed and controlled:

- Excess moisture
- Critical colonisation or true infection
- Friction/movement at wound interface
- Foreign material

As with other multifactorial pathologies, it is wise to introduce a treatment regimen that is multitargeted, aimed at all steps in the pathogenesis rather than one step.[13] Thus in the past, silver nitrate may have been used to remove the tissue, but this did not address the exudates, infection or friction and recurrence was common.

The basic suggested routine which has been used successfully in the treatment of pilonidal sinus[14] would involve the following steps (Figure 1):

- Initial debridement of the hypergranulation: this can take the form of dry swab wipe removal (painful, used if clinician is untrained at sharp debridement), sharp debridement or curettage (preferable), or silver nitrate (not primary choice, due to destructive nature of this modality – stimulates further inflammation).
- Ag (nanocrystalline, NCS) alginate dressing (Acticoat® absorbent): the calcium facilitates clotting, controlling the bleeding, the alginate provides excellent exudate control and the nanocrystalline silver has an ideal antimicrobial spectrum.[15,16]

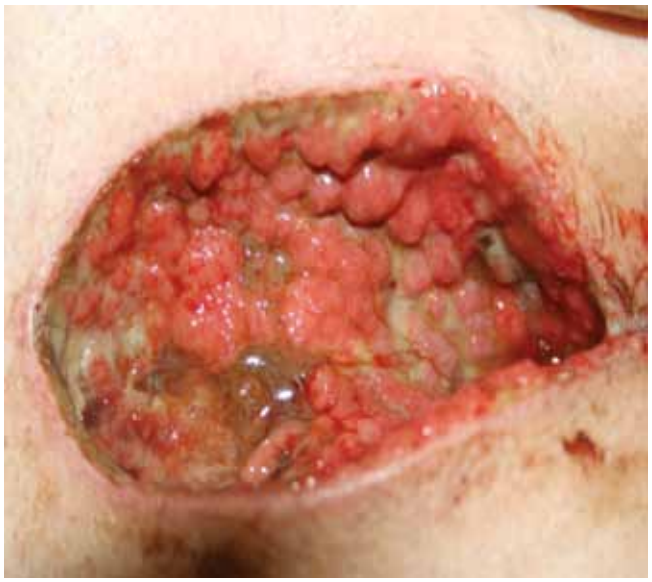

**Figure 2:** Hypergranulation in the base of a pilonidal sinus

- A secondary foam dressing (Allevyn® adhesive) is added to the mix to mop up excess exudate and, more importantly, to provide minor compression and prevent shear and friction at the wound interface and the surrounding skin.
- Minor compression (where possible) can be added to secure dressings and prevent friction.

This routine has been used successfully to treat pilonidal sinus,[14] a situation that commonly involves hypergranulation tissue with the exact background causes elucidated in Figures 2 and 3 – moisture, possible infection and friction.[14,17] This regimen is extremely patient-friendly (as opposed to other modalities) and appears to be successful, as it concentrates on all elements of the background pathophysiology.

## Conclusion

Hypergranulation tissue is a condition that occurs commonly, can halt healing and re-epithelialisation and presently lacks a coherent approach to its eradication and prevention. The systematic approach to each individual background pathophysiologic contributor to the process is a logical approach to the problem. This has been successfully applied to hypergranulation tissue arising in pilonidal sinus, and is suggested as a good practical solution to hypergranulation tissue in general.

## Conflict of interest

Professor Widgerow is consultant to Sirius Scientifica; Smith & Nephew; Omnimed; LITHA healthcare, Southern Medical. No part of this publication or study was influenced by any of these companies.

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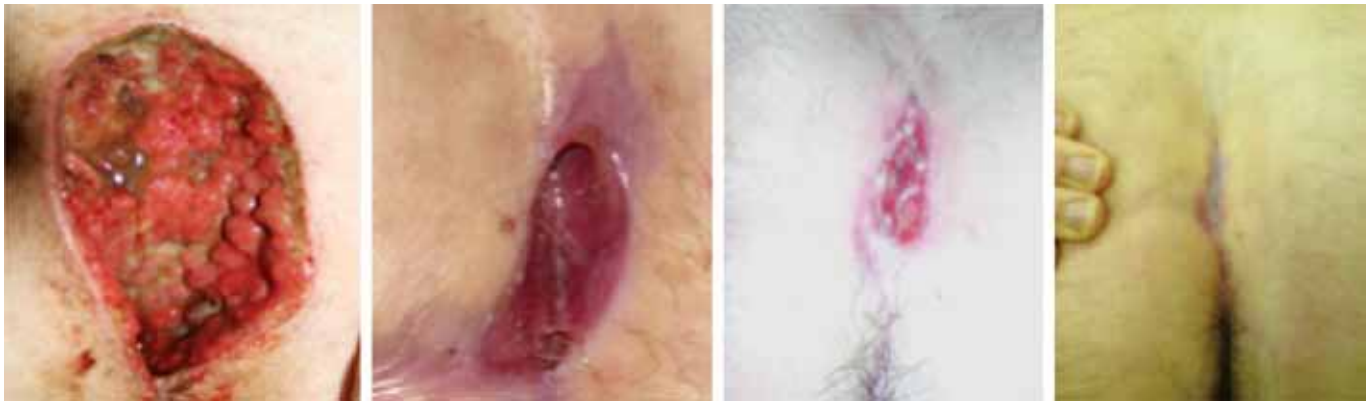

**Figure 3:** Pilonidal sinus treatment progression, from hypergranulating wound base (shown in Figure 2) to full healing