Pilonidal Recurrence: Hypoxia in Cleft as a Source

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Dear Sir,

Søndenaa et al. [1] identify chronic inflammatory processes as the source of pilonidal recurrence. They advance pilonidal management with their careful review and report the problems created by recurrent pilonidal disease and its treatment.

Recurrences appear only in the midline whether it is covered with shifted skin of any thickness, surgical scar, or native tissue. It must follow that weakness of midline tissue is not the essential defect. Instead, unnamed agents in the midline attack any tissue that lies there. Wounds will heal when factors change in the cleft. Marks et al. [2] identified anaerobic bacteria as one agent of delayed healing. Leaving airless folds in the cleft after surgery creates localized anaerobic conditions, bacterial growth, and recurrences. Leaving a suture line there further provokes delayed healing.

Conventional pilonidal surgery removes tissues that are deformed yet innocent; understanding this concept calls for a paradigm shift in our thinking [3]. These are normal tissues responding to attack. The true culprits are invisible. We cannot see, as we spread the cleft, that the air within is low in oxygen. We cannot see bacteria that would be eradicated if oxygen bathed a pilonidal recurrence. We cannot see tethering tissues that pull the cleft to the sacrum which increases cleft airlessness in the upright patient. Simply cleansing the cleft [4] can alter the factors such that deformed normal tissues heal in many cases. Even the most difficult recurrence can be healed by constructing a shallower and ventilated cleft [5]. However, the final suture line, asymmetric in position, must lie on a ventilated, convex surface and not within a hazardous, anaerobic fold.

References

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