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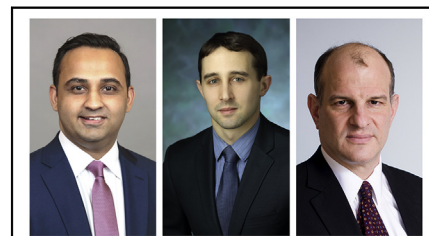


Commentary: Predicting surgical-site infections following cardiac surgery? Perhaps the “NOSE” knows

Sameer A. Hirji, MD, MPH, Jake A. Awtry, MD, MA, and George Tolis, Jr, MD

Despite advances in local wound care and standardized perioperative treatment protocols, surgical-site infection (SSI) continues to be a significant contributor to morbidity, mortality, and cost after cardiac surgery.^{1,2} To limit SSI and improve outcomes, previous work has primarily focused on improving perioperative care through stewardship of perioperative antibiotic use, optimization of glycemic control, and, in some cases, preoperative prophylactic treatment of intranasal *Staphylococcus* decolonization with mupirocin.^{3,4} Although screening for and treating *Staphylococcus* colonization before surgery is known to reduce SSI, to our knowledge no previous study has specifically assessed the relative contributions of other nasopharyngeal organisms to development of SSI after cardiac surgery.⁵

Takami and colleagues,⁶ in this issue of the *Journal*, address this timely question and retrospectively reviewed 1226 consecutive patients undergoing cardiac surgery via median sternotomy with preoperative nasopharyngeal or nasal cultures. Specifically, their study evaluated the relationship between microbial patterns and SSI and demonstrated that cultures positive for abnormal nasopharyngeal flora were an independent predictor of SSI, in addition to known predictors such as female sex, diabetes mellitus, and use of tracheostomy. While the study findings were



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CENTRAL MESSAGE

Abnormal nasopharyngeal flora may be a risk factor for SSIs in patients after cardiac surgery, but further investigations under optimal SSI-prevention conditions are warranted.

hypothesis-generating, the authors suggested that the use of both preoperative nasal and nasopharyngeal cultures, which was found to have high sensitivity and specificity for SSI, respectively, would allow for possible identification of high-risk patients to help tailor the use of prophylactic measures (eg, wound vac therapy) or guide initial therapy for infections.

Takami and colleagues⁶ should be applauded for this extensive study and for providing a framework to identify additional readily identifiable and potentially actionable risk factors. Interpretation of their results, however, should take into account the following limitations. First, local protocols in the studied time period included use of sternal bone paste and frequent use of tracheostomies. Both these have been previously shown to be associated with SSI and may have likely contributed to the high rate of SSI observed in the study (6.5%). Second, intraoperative gentamicin was not used, which may have contributed to the sizable number of SSIs caused by gram-negative rods. Furthermore, although abnormal flora in cultures was a risk factor for SSI, there was no statistical concordance between the organisms grown in culture and those that caused the associated SSI.

Although some clinicians have postulated that infections in the surgical population are not simply related to the presence of virulent organisms, it is possible that some infections are a result of more broad disturbances in the host–microbiome relationship that promote bacterial virulence.⁷ Thus, the causative relationship between the

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aberrant nasopharyngeal microbes and SSI as well as the relevance of the association in the setting of more stringent SSI prevention protocols remains unclear at this time. Possible further studies may include validating the proposed association in a prospective cohort of patients, mechanistic investigations into how alterations in the skin and nasopharyngeal microbiome contribute to SSI, and adjusting perioperative antibiotics or local wound care approaches for individuals determined to be high risk based on preoperative cultures. Time will tell whether the “nose” knows.

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