During Oncologic Oral Surgery, the Endothelial Glycocalyx Sheds Independently of Systemic Tryptase Release

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Received: 08-Oct-2022, Manuscript No. SCR-22-19669; **Editor assigned:** 9-Oct-2022, Pre QC No. SCR-22-19669(PQ); **Reviewed:** 15-Oct-2022, QC No. SCR-22-19669 (Q); **Revised:** 16-Oct-2022, Manuscript No. SCR-22-19669 (R); **Published:** 19-Oct-2022, DOI: 10.35248/2161-1076.22.12(10).415

Abstract

Several vascular activities depend on the endothelium surface layer and endothelial glycocalyx. Various clinical situations, including significant surgery, can cause the glycocalyx to tear or shed. One potential "sheddase" has been suggested, and that is mast cell tryptase. Glycocalyx shedding during oncologic oral surgery may be harmful because it causes oedema in the musculocutaneous flap graft and a loss of vascular barrier function. In 16 patients having oncologic oral surgery, the blood serum concentrations of tryptase, heparan sulphate, and syndecan-1, as well as their levels before and after surgery, were determined. The concentrations of these compounds on postoperative day 1 and day 2 served as secondary measurements. During surgery, the level of heparan sulphate increased from 692 ng/mL. From 35 ng/mL to 138 ng/mL, syndecan-1 levels increased. Tryptase levels were almost same before and after surgery, at 4.2 ng/mL and 4.2 ng/mL, respectively. During surgery, serum levels of syndecan-1 and heparan sulphate rose, indicating glycocalyx shedding. Tryptase levels remained constant, indicating that damage to the glycocalyx may have been caused by sheddases other than systemic tryptase release. A possible way to enhance flap viability and patient outcomes during oncologic oral surgery is to look into protective measures for the glycocalyx.

Keywords: Oral surgery • Glycocalyx • Tryptase

Introduction

A thin layer of glycoproteins known as the endothelial glycocalyx covers the endoluminal surface of the vasculature. The Endothelial Surface Layer (ESL) is formed when it combines with plasma proteins. On the luminal surfaces of endothelial cells, syndecan-1 is a transmembrane molecule having attachment sites for glycosaminoglycans. Additionally, it contributes to transmembrane signaling. The primary glycosaminoglycan of the endothelium glycocalyx, heparan sulphate, is linked to syndecan-1 in the vascular lumen. Numerous artery activities, such as vascular permeability, vascular tone, and coagulation, depend on the ESL. Recent studies have shown that a variety of pathologic situations, including as sepsis, severe injury, or cardiac arrest, can lead to the breakdown of the glycocalyx and the release of its constituents into the bloodstream, including heparan sulphate and syndecan-1. Additionally, it has been demonstrated that major surgery is linked to glycocalyx shedding. The ESL's functions are disturbed with if the glycocalyx is shed.

Large fluid infusions and ischaemia reperfusion during flap surgery, two situations typically seen during oral cancer surgery, are also recognized as factors to glycocalyx shedding. On the other hand, ESL disruption symptoms are typically seen during oncologic oral surgery, specifically a loss of vascular barrier function. High fluid amounts that must be infused postoperatively are one indication of this. There is still much to learn about the precise mechanisms of glycocalyx shedding and the molecules responsible, the so-called "sheddases". Mast cell activation and the release of their enzyme tryptase have been mentioned as potential mechanisms, among others. We predicted that severe oncologic oral surgery, especially when free or pedicled flap reconstruction was involved, would result in tryptase release from mast cells and glycocalyx shedding. The perfusion of flaps used for reconstruction after oral cancer surgery can be endangered by fluid overload, so it may be possible to improve flap viability and the medical and surgical perioperative course by examining how the glycocalyx sheds during oral surgery and developing protective measures. Therefore, we designed a study to evaluate tryptase concentration and glycocalyx shedding in patients having oral cancer surgery, as indicated by levels of the glycocalyx components heparan sulphate and syndecan-1.

Shedding of glycocalyx components

Prior to surgery, the blood serum concentration of heparan sulphate was 692 ng/mL; upon admission to the critical care unit, it was 810 ng/mL. The Wilcoxon matched-pair signed-rank test yielded a statistically significant result for this increment (p=0.024). On the first and second postoperative days, the heparan sulphate concentrations stayed at 789 ng/mL and 787 ng/mL, respectively. According to the ROUT technique with Q=1%, there were two cases with exceptional values. The difference remained statistically significant once these outliers were taken into account (paired t-test p=0.005, Wilcoxon matched-pair signed-rank test p=0.005).

As previously mentioned, the concentration of syndecan-1 in blood serum (n=12) increased from 35 ng/mL prior to surgery to 138 ng/mL upon admission to the intensive care unit and decreased to 123 ng/mL on the first and 61ng/mL on the second postoperative day.

Concentration of tryptase

Before surgery and after admission to the ICU, the serum tryptase concentration was 4.2 ng/mL and 4.2 ng/mL, respectively. Tryptase concentration was 3.7 ng/mL and 4.3 ng/mL on the first and second postoperative days, respectively. One patient was identified by the ROUT technique as having exceptionally high tryptase readings at all four measurements. There were no discernible changes in the perioperative tryptase concentration even after this outlier was taken into account.

Discussion

We were able to verify our theory that following major oral surgery, the endothelium glycocalyx sheds and its constituent parts are discharged into the bloodstream. This aspect of the study hypothesis was not supported because the rise in tryptase activity did not coincide with the rise in glycocalyx-shedding products, suggesting that the shedding of the glycocalyx in this circumstance occurs independently of mast cell activation and tryptase release. Heparan sulphate and syndecan-1 concentration time courses follow different paths. Heparan sulphate remained high until the completion of our measures on the second postoperative day, although syndecan-1 fell near to its preoperative level in our patients, despite the fact that both rise after surgery. This is similar to the time courses seen in patients undergoing straightforward abdominal surgery or abdominal aortic repair. Syndecan-1 and heparan sulphate, on the other hand, returned to normal levels more quickly following cardiac surgery, while patients who remained septic after abdominal surgery experienced delayed returns to normal levels.

During oncologic oral surgery, a number of variables could cause glycocalyx to shed: free-flap surgery involves ischaemia and reperfusion of the graft. In numerous clinical and experimental settings, it has been demonstrated that ischemia-reperfusion results in the glycocalyx being shed. The free-flap graft may lose glycocalyx due to ischaemia and subsequent reperfusion, according to a supposition. Additionally, the glycocalyx of the vasculature beyond the graft may also be harmed if mediators that cause the glycocalyx to shed build up in the graft and are then released into the circulation following anastomosis. Larger fluid injections could be another significant aspect. Volume loading causes the release of atrial natriuretic peptide, which causes the glycocalyx to shed. However, massive fluid infusions may not only contribute to glycocalyx shedding but also be a result of it. Damages to the glycocalyx result in endothelial leakage because maintaining vascular barrier function is one

of the glycocalyx's and the endothelial surface layer's primary roles. This leads to extravasation, which can worsen intravascular hypovolemia, and is therefore frequently treated with high infusion volumes to maintain adequate mean arterial pressure for organ perfusion. Vascular hyperpermeability brought on by glycocalyx shedding has been established in patients with burn injuries, a mouse model of abdominal sepsis, and a cell-culture model of Dengue-virus-induced glycocalyx shedding.

The specific impact of vascular leakage during oral flap surgery may raise concerns in addition to the general deleterious effects of the condition, such as generalized oedema, pulmonary oedema, or pleural effusion. Capillary leaking around the flap graft could cause tissue oedema and endanger the viability of the flap. Even though there are no research specifically looking into a connection between flap viability and endothelial glycocalyx status, there are indications that one might exist. The injection of large amounts of fluid has been found as a risk factor for postoperative problems, including flap failure, in an older analysis of predictors of complications after major head and neck surgery. Later studies looked examined how surgical and medical complications following head and neck surgery were affected by goal-directed infusion treatment, which implies infusion of lower volumes than with standard fluid management. Higher flap survival and lower rates of flap necrosis were the results of goal-directed therapy, respectively. According to some writers, the relationship between high infusion rates and worse flap outcomes may be due to the glycocalyx.

Tryptase levels in this research were stable during the operation. Therefore, it seems unlikely that systemic release of tryptase—also known as "sheddase"—plays a significant role in the glycocalyx shedding that we saw in our patients. Though not sufficient to raise systemic blood concentrations, local release by perivascular tissue mast cells may nonetheless contribute to glycocalyx shedding. The pathophysiological mechanisms and sheddases, such as matrixmetalloproteinases or heparinase, that destroy the glycocalyx following oral surgery are still unknown.

Conclusion

In this pilot investigation, we discovered a rise in the blood levels of syndecan-1 and heparan sulphate, which suggests that the endothelium glycocalyx was shed after the significant oncologic oral surgery. As systemic tryptase release did not change throughout surgery, it suggests that additional sheddases other than tryptase are responsible for this glycocalyx shedding.

The results of this pilot study motivate more, larger studies to clarify the processes that cause glycocalyx to shed during oral surgery. Further research should be done into strategies for preserving the glycocalyx, such as normovolemia, heparanase inhibitor infusion, steroids, or doxycycline. Due to possibly increased macro- and microcirculation, protecting the glycocalyx may have the potential to hasten postoperative recovery.

Cite this article: Mathew C. During Oncologic Oral Surgery, the Endothelial Glycocalyx Sheds Independently of Systemic Tryptase Release. Surg Curr Res, 2022, 12(10), 001-002.