INTERNATIONAL JOURNAL OF MEDICAL SCIENCE AND DENTAL HEALTH
(ISSN: 2454-4191)
www.ijmsdh.org (2026)

Check for updates

Received: 30 Sep 2026
Revised: 12 Oct 2026
Accepted: 20 Oct 2026
Published: 27 Oct 2026

2026: OCTOBER - IJMSDH
Page No – 71-76
DOI - 10.55640/ijmsdh-spc-10

Article Citation: Dr. Aris Nugraha. (2026). Molecular Mechanisms and Clinical Outcomes of Tobacco-Associated Pathologies: A Comparative Review of GPI Transamidase Dysregulation in Head and Neck Squamous Cell Carcinoma and Hemodynamic Prognosis in Abdominal Aortic Aneurysm. International Journal of Medical Science and Dental Health, 1(01), 71-76. https://doi.org/10.55640/ijmsdh-spc-10

Copyright: © 2026 The Authors. Published by IJMSDH under the Creative Commons CC BY License

Molecular Mechanisms and Clinical Outcomes of Tobacco-Associated Pathologies: A Comparative Review of GPI Transamidase Dysregulation in Head and Neck Squamous Cell Carcinoma and Hemodynamic Prognosis in Abdominal Aortic Aneurysm

Dr. Aris Nugraha

Faculty of Medicine, Universitas Indonesia, Jakarta, Indonesia

Abstract

Background: Tobacco consumption remains a pervasive global health challenge, driving distinct pathologies across physiological systems. This study examines two divergent consequences of systemic tobacco exposure: the molecular upregulation of Glycosylphosphatidylinositol (GPI) transamidase in Head and Neck Squamous Cell Carcinoma (HNSCC) and the hemodynamic failure associated with Abdominal Aortic Aneurysm (AAA).

Methods: A comprehensive review was conducted analyzing genomic data regarding GPI biosynthesis subunits (PIG-U, GPAA1) in HNSCC and clinical outcomes data for AAA screening and repair. Data sources included The Cancer Genome Atlas (TCGA), MIMIC-IV databases, and recent randomized trials comparing endovascular versus open repair.

Results: In HNSCC, overexpression of the PIG-U subunit acts as a critical oncogene, facilitating the anchoring of proteins that drive cellular proliferation and immune evasion within the tumor microenvironment. Tobacco smoke was found to specifically activate protein association and tyrosine phosphorylation of GPI-transamidase subunits. Conversely, in AAA, mortality remains high despite advances in screening; however, updated US Preventive Services Task Force guidelines and predictive scoring systems like the Glasgow Aneurysm Score have improved risk stratification. Endovascular repair continues to show survival benefits over open repair in ruptured cases.

Conclusion: While HNSCC requires a molecular approach targeting GPI-anchored protein pathways and immunomodulation, AAA management relies on early detection and precise surgical intervention. Understanding these distinct pathways highlights the multifaceted impact of tobacco and underscores the need for organ-specific therapeutic strategies.

Keywords: Head and Neck Squamous Cell Carcinoma, GPI Transamidase, Abdominal Aortic Aneurysm, PIG-U, Tumor Microenvironment, Endovascular Repair, Tobacco Pathology.

Introduction

The systemic burden of tobacco consumption manifests through a complex array of pathophysiological mechanisms, affecting cellular regulation and structural tissue integrity in profoundly different ways. While the link between tobacco and lung pathologies is ubiquitously recognized, its role in driving the distinct etiologies of Head and Neck Squamous Cell Carcinoma (HNSCC) and Abdominal Aortic Aneurysm (AAA) represents a critical area of medical research. According to GLOBOCAN 2022 estimates, the global incidence of cancer continues to rise, with HNSCC representing a significant portion of preventable morbidity [1]. Simultaneously, vascular pathologies such as AAA remain a leading cause of sudden death in older adults, necessitating rigorous screening and intervention protocols [15][16].

This article seeks to bridge the gap between these two disparate fields—molecular oncology and vascular surgery—by exploring them as divergent downstream consequences of a shared risk factor profile. In HNSCC, the pathology is characterized by unmitigated cellular proliferation and genomic instability. Recent genomic characterizations have identified specific alterations in the glycosylphosphatidylinositol (GPI) transamidase complex as pivotal drivers of malignancy [4][12]. The subunits of this complex, particularly PIG-U and GPAA1, are responsible for attaching GPI anchors to proteins, a post-translational modification essential for the surface expression of various receptors and adhesion molecules [5][6]. The upregulation of these subunits, often triggered by tobacco smoke, leads to an aberrant tumor microenvironment (TME) that fosters immune evasion and metastasis [2][13].

Conversely, the pathology of AAA is defined not by proliferation, but by degradation and structural failure. The aortic wall, subjected to chronic hemodynamic stress and inflammatory damage from tobacco toxins, weakens over time, leading to dilation and potential rupture. The management of AAA focuses heavily on early detection through screening and the careful selection of surgical repair techniques—open versus endovascular [19][23]. Unlike the molecular targets sought in HNSCC therapy, AAA management relies on predictive scoring systems and intensive care unit (ICU) prognostication to reduce mortality rates [21][22].

By juxtaposing the molecular upregulation of GPI transamidase in HNSCC with the clinical management of AAA, this review provides a comprehensive overview of how a single environmental insult can drive both intracellular chaos and macroscopic structural failure. We aim to synthesize current knowledge on the biochemical pathways of GPI-anchored proteins (GPI-APs) and contrast this with the evidence-based guidelines for AAA screening and repair, ultimately highlighting the multifaceted approach required for patients with tobacco-associated comorbidities.

Methods

To ensure a robust analysis of both molecular and clinical dimensions, a dual-pronged literature review strategy was employed. For the oncological section, databases including PubMed, Scopus, and The Cancer Genome Atlas (TCGA) were queried for terms such as "GPI transamidase," "PIG-U," "HNSCC," "Tumor Microenvironment," and "Immunomodulation." Special attention was given to studies elucidating the biochemical structure and function of the GPI transamidase complex, specifically references spanning from foundational cell biology studies in 2003 to structural breakthroughs in 2024 [5][9].

For the vascular surgery section, the review focused on clinical guidelines and randomized controlled trials. Keywords included "Abdominal Aortic Aneurysm," "Screening Guidelines," "Endovascular Repair," "Open Repair," and "Mortality Prediction." The search prioritized high-impact guidelines from the US Preventive Services Task Force and large-scale meta-analyses of patient outcomes [15][18].

Inclusion criteria for the molecular section required studies to demonstrate a mechanistic link between GPI subunits and cancer progression or to provide structural insights into the transamidase complex. For the clinical section, studies were selected based on their relevance to current screening standards and the statistical power of their mortality analyses. Data extraction focused on identifying the specific gene amplifications associated with HNSCC prognosis and the statistical hazard ratios associated with different AAA repair modalities.

Results and Comprehensive Analysis

Part I: Molecular Pathogenesis in HNSCC

The Architecture of GPI Transamidase

The attachment of GPI anchors to proteins is a critical post-translational modification that occurs within the

endoplasmic reticulum (ER). This process is catalyzed by the GPI transamidase complex, a multi-subunit membrane-bound enzyme. Understanding the structural biology of this complex is essential for grasping its oncogenic potential. The complex is composed of five subunits: PIG-K, PIG-S, PIG-T, PIG-U, and GPAA1 [5]. PIG-K serves as the catalytic component, cleaving the C-terminal signal peptide of the precursor protein, while the other subunits function to stabilize the complex and maintain the fidelity of the transfer reaction [7].

Recent structural analyses using cryo-electron microscopy have illuminated the precise arrangement of these subunits, revealing how PIG-U and GPAA1 facilitate the recognition of the GPI lipid substrate [8][9]. PIG-U, identified as the fifth subunit, is particularly crucial as it is believed to be involved in recognizing the long-chain fatty acids of the GPI anchor, bridging the hydrophilic protein substrate with the hydrophobic membrane environment [5]. The integrity of this complex is vital; if any subunit is downregulated or dysfunctional, the surface expression of GPI-anchored proteins (GPI-APs) is severely compromised, often leading to cellular lethality or dysfunction. However, in the context of cancer, the reverse is observed: the complex is hyper-active.

PIG-U and GPAA1 as Oncogenic Drivers

The transition from normal cellular physiology to malignancy in HNSCC is often driven by copy number variations (CNVs). Comprehensive genomic characterization of HNSCC has revealed that the chromosomal region 20q11-13 is frequently amplified [4][12]. This region houses the genes for both PIG-U (CDC91L1) and GPAA1. When these genes are amplified, the resulting overexpression of the GPI transamidase complex leads to an overabundance of GPI-APs on the cell surface.

This overexpression is not merely a bystander effect but a driver of oncogenesis. Research has demonstrated that PIG-U acts as an oncogene in various carcinomas, including bladder and head and neck cancers [11]. The mechanism involves the upregulation of specific GPI-APs that function as receptors for growth factors and cytokines. For instance, the urokinase plasminogen activator receptor (uPAR) is a GPI-AP that, when overexpressed, promotes tissue invasion and metastasis. Similarly, GPAA1 overexpression has been linked to the enhancement of the ERBB signaling pathway, further driving cell proliferation [14].

Crucially, tobacco smoke has been identified as a potent activator of this system. Exposure to tobacco smoke extract has been shown to induce the tyrosine phosphorylation of GPI transamidase subunits, enhancing their association and catalytic efficiency [13]. This suggests a direct molecular link between the carcinogens in tobacco smoke and the biochemical machinery responsible for displaying growth-promoting proteins on the tumor cell surface. This phosphorylation event stabilizes the complex, preventing its degradation and ensuring a sustained high level of GPI-AP presentation, which in turn feeds into the aggressive phenotype typical of smoking-associated HNSCC.

Immunomodulation and the Tumor Microenvironment

The implications of GPI transamidase dysregulation extend beyond the tumor cell itself, profoundly influencing the tumor microenvironment (TME). The TME in HNSCC is a complex ecosystem comprising immune cells, fibroblasts, and extracellular matrix components. The profile of proteins displayed on the tumor surface dictates how the immune system perceives and interacts with the malignancy [2].

GPI-anchored proteins play a significant role in this immunological "handshake." Many immune regulatory proteins, such as CD55 and CD59, are GPI-anchored. These proteins function as complement regulatory factors, protecting cells from complement-mediated lysis. In HNSCC tumors where GPI transamidase is upregulated, there is often a concomitant increase in the surface density of CD55 and CD59 [10]. This creates a "shield" around the tumor cells, rendering them resistant to the innate immune system's attempts to eliminate them.

Furthermore, the TME in HNSCC is characterized by significant immunosuppression. Strategies to reverse this suppression are the cornerstone of modern immunotherapy [3]. However, the efficacy of agents such as pembrolizumab or nivolumab (PD-1 inhibitors) can be modulated by the density of other surface markers. The overexpression of GPI-APs can physically alter the landscape of the cell membrane, potentially affecting the accessibility of target antigens or clustering with other receptors to trigger alternative signaling pathways that bypass immune checkpoints [6].

Expanding the Genomic Landscape of GPI Biosynthesis

To fully appreciate the role of GPI transamidase in HNSCC, one must consider the lipid remodeling that occurs alongside protein attachment. The biosynthesis of GPI anchors is not a static process; it involves distinct lipid remodeling steps that tailor the anchor for specific membrane domains, such as lipid rafts [6]. These rafts are signaling hubs. When PIG-U and GPAA1 are overexpressed, they not only increase the number of GPI-APs but may also alter the specific lipid composition of the anchors, forcing a redistribution of receptors into these signaling hotspots. This clustering effect amplifies signaling cascades related to cell survival and migration, independent of ligand binding [7].

The genetic amplification of PIGU and GPAA1 in HNSCC is statistically significant when compared to healthy tissue, serving as a potential biomarker for poor prognosis [12]. Patients exhibiting high expression levels of these subunits often present with more advanced disease stages and higher rates of lymph node metastasis. This correlation underscores the potential of targeting the GPI biosynthesis pathway as a therapeutic strategy. Small molecule inhibitors that could disrupt the interaction between PIG-U and the GPI lipid substrate, or prevent the assembly of the transamidase complex, could theoretically strip the tumor cells of their protective and proliferative surface coat, rendering them vulnerable to standard chemotherapies and immune attack [8][9].

Part II: Clinical Management of Abdominal Aortic Aneurysm (AAA)

Screening and Diagnosis

Shifting focus from the microscopic to the macroscopic, the impact of tobacco on the abdominal aorta results in the weakening of the arterial wall, leading to aneurysm formation. The mortality associated with ruptured AAA is catastrophic, making screening a priority for at-risk populations. The US Preventive Services Task Force (USPSTF) has updated its evidence report and recommendations, emphasizing the importance of one-time ultrasonography screening for men aged 65 to 75 who have ever smoked [15][16]. This recommendation is grounded in the high prevalence of AAA in this demographic and the clear survival benefit associated with elective repair prior to rupture.

However, the screening guidelines remain nuanced. For men who have never smoked, the benefit is less clear, and for women, the prevalence is generally too low to warrant routine screening, although tobacco use in women significantly increases risk. The guidelines reflect an understanding of the epidemiological shift; while smoking rates have declined in some regions, the lag time for aneurysm development means the burden of disease remains high among the current geriatric population.

Surgical Intervention: EVAR vs. Open Repair

Once an aneurysm is detected and reaches a size threshold (typically 5.5 cm in men), intervention is required. The debate between Endovascular Aneurysm Repair (EVAR) and Open Surgical Repair (OSR) has defined vascular surgery for the past two decades. EVAR, a minimally invasive technique involving the placement of a stent-graft through the femoral arteries, has become the preferred modality for anatomically suitable candidates.

Meta-analyses of randomized trials have consistently shown that EVAR offers a significant reduction in perioperative mortality compared to open repair [18][23]. The minimally invasive nature of EVAR reduces physiological stress, blood loss, and ICU length of stay. However, the long-term durability of EVAR is a concern, with higher rates of re-intervention required due to complications such as endoleaks. Conversely, open repair, while carrying a higher initial risk, generally provides a more durable solution with fewer long-term surveillance requirements.

In the context of ruptured AAA (rAAA), the stakes are significantly higher. Individual-patient meta-analyses of trials comparing EVAR versus open repair for rupture suggest a survival benefit for EVAR, provided the patient's anatomy allows for rapid deployment of the device [18]. The "endovascular-first" strategy for rupture has been adopted by many high-volume centers, leading to a gradual decline in overall rAAA mortality [20].

Prognostic Scoring and Intensive Care

The management of AAA extends into the postoperative period, particularly for patients admitted to the Intensive Care Unit (ICU) following repair. Predicting mortality in these high-risk patients is essential for guiding clinical decision-making and resource allocation. Various scoring systems have been evaluated for their predictive accuracy. The Glasgow Aneurysm Score (GAS) is a specific tool designed for this purpose, incorporating factors such as age, shock, and renal disease [22].

Recent studies utilizing large datasets like MIMIC-IV have compared general ICU scoring systems (such as APACHE II or SOFA) against disease-specific scores like GAS [22]. The findings suggest that while general scores are useful for assessing organ failure, aneurysm-specific scores often provide better discrimination for mortality related to the unique hemodynamics of aortic pathology. Factors such as admission lactate levels, hemodynamic stability, and renal function are critical determinants of 28-day mortality [21]. The integration of these scoring systems into clinical practice allows for more personalized care plans and better communication of prognosis to families.

Post-Surgical Hemodynamics and Comorbidities

The survival of AAA patients is not solely determined by the technical success of the repair. These patients often possess a heavy burden of cardiovascular comorbidities—largely driven by the same history of tobacco use that caused the aneurysm. Hypertension, coronary artery disease, and chronic obstructive pulmonary disease (COPD) are ubiquitous in this population [17]. The management of these comorbidities in the ICU is as critical as the surgical repair itself. For instance, rigorous blood pressure control is vital to protect the repair site, while careful fluid

management is necessary to prevent renal failure and pulmonary edema.

Discussion: The Convergence of Tobacco Pathologies

The juxtaposition of GPI transamidase upregulation in HNSCC and the clinical management of AAA illustrates the pervasive and multifaceted toxicity of tobacco. In the head and neck, tobacco smoke acts as a molecular accelerant. It chemically modifies the enzymatic machinery of the cell (GPI transamidase), leading to the overexpression of surface proteins that drive division and hide the cell from the immune system [13]. This is a pathology of addition—genomic amplification, protein overexpression, and tissue hyperplasia.

In the abdominal aorta, tobacco acts as a destructive force. It triggers inflammatory cascades that degrade elastin and collagen, leading to the structural failure of the vessel wall. This is a pathology of subtraction—loss of elasticity, loss of structural integrity, and eventual rupture.

Despite these opposing cellular mechanisms (hyper-proliferation vs. tissue degradation), the clinical reality is that these conditions frequently coexist or occur sequentially in the same patient population. A patient successfully treated for HNSCC may succumb years later to a ruptured AAA if surveillance is neglected. Conversely, a patient surviving AAA repair faces a significantly elevated risk of developing malignancies due to their smoking history.

Therapeutic Implications

The insights from the molecular biology of HNSCC offer potential avenues for therapeutic intervention that go beyond standard chemotherapy. Targeting the GPI transamidase complex, specifically the PIG-U subunit, represents a novel strategy. If small molecule inhibitors can be developed to block the assembly or function of this complex, it may be possible to strip tumor cells of their "armor" (CD55/CD59) and their "engines" (growth factor receptors) [9]. This would potentially sensitize the tumor to immunotherapy, enhancing the efficacy of checkpoint inhibitors by altering the TME [3].

For AAA, the focus remains on refinement of screening and surgical technique. The data supports a continued shift toward endovascular approaches, but also highlights the need for rigorous post-operative surveillance. The improvement in ICU prognostic scoring [22] allows clinicians to better identify patients who require more aggressive supportive care following repair.

Limitations

This review is limited by the heterogeneity of the studies analyzed. The molecular data for HNSCC is largely derived from genomic databases and in vitro studies, which may not perfectly reflect in vivo biology in all patients. Similarly, the clinical data for AAA is drawn from trials that may exclude patients with complex anatomies or severe comorbidities, potentially limiting the generalizability of the findings. Furthermore, while tobacco is a common link, the specific genetic susceptibility that predisposes one smoker to cancer and another to aneurysm remains an area of active investigation.

Future Directions

Future research should aim to bridge the divide between these specialties. Prospective studies tracking inflammatory markers and genomic profiles in AAA patients could reveal early biomarkers for malignancy. Conversely, HNSCC patients should perhaps undergo more rigorous cardiovascular screening than the general population, given their shared risk profile. Understanding the shared and divergent pathways of tobacco toxicity is essential for developing holistic treatment strategies that address the patient as a whole, rather than treating organ systems in isolation.

Conclusion

The impact of tobacco exposure is systemic, yet its manifestations are organ-specific and mechanistically distinct. In Head and Neck Squamous Cell Carcinoma, tobacco smoke drives the oncogenic upregulation of the GPI transamidase complex, specifically the PIG-U and GPAA1 subunits, facilitating tumor survival and immune evasion. In contrast, in the abdominal aorta, tobacco exposure precipitates structural degradation leading to aneurysm formation, necessitating vigilant screening and complex surgical management. This comparative review underscores the necessity of a multidisciplinary approach to the tobacco-exposed patient. While molecular biologists unravel the complexities of GPI-anchor biosynthesis to develop new cancer therapies, vascular surgeons must continue to refine predictive models and surgical techniques to prevent catastrophic aortic failure. Ultimately, cessation of tobacco use remains the single most effective intervention to halt the progression of both the molecular machinery of cancer and the hemodynamic collapse of the vasculature.

References

1. Bray F, Laversanne M, Sung H, Ferlay J, Siegel RL, Soerjomataram I, Jemal A: Global cancer statistics 2022: GLOBOCAN estimates of incidence and mortality worldwide for 36 cancers in 185 countries. CA Cancer J Clin 2024, 74:229-263.
2. Ruffin AT, Li H, Vujanovic L, Zandberg DP, Ferris RL, Bruno TC: Improving head and neck cancer therapies by

immunomodulation of the tumour microenvironment. Nat Rev Cancer 2023, 23:173-188.
3. Miyauchi S, Kim SS, Pang J, Gold KA, Gutkind JS, Califano JA, Mell LK, Cohen EEW, Sharabi AB: Immune Modulation of Head and Neck Squamous Cell Carcinoma and the Tumor Microenvironment by Conventional Therapeutics. Clin Cancer Res 2019, 25:4211-4223.
4. Cancer Genome Atlas N: Comprehensive genomic characterization of head and neck squamous cell carcinomas. Nature 2015, 517:576-582.
5. Hong Y, Ohishi K, Kang JY, Tanaka S, Inoue N, Nishimura J, Maeda Y, Kinoshita T: Human PIG-U and yeast Cdc91p are the fifth subunit of GPI transamidase that attaches GPI-anchors to proteins. Mol Biol Cell 2003, 14:1780-1789.
6. Kinoshita T, Fujita M: Biosynthesis of GPI-anchored proteins: special emphasis on GPI lipid remodeling. J Lipid Res 2016, 57:6-24.
7. Kinoshita T: Biosynthesis and biology of mammalian GPI-anchored proteins. Open Biol 2020, 10:190290.
8. Xu Y, Li T, Zhou Z, Hong J, Chao Y, Zhu Z, Zhang Y, Qu Q, Li D: Structures of liganded glycosylphosphatidylinositol transamidase illuminate GPI-AP biogenesis. Nat Commun 2023, 14:5520.
9. Li D: Structure and Function of the Glycosylphosphatidylinositol Transamidase, a Transmembrane Complex Catalyzing GPI Anchoring of Proteins. Subcell Biochem 2024, 104:425-458.
10. Gamage DG, Hendrickson TL: GPI transamidase and GPI anchored proteins: oncogenes and biomarkers for cancer. Crit Rev Biochem Mol Biol 2013, 48:446-464.
11. Guo Z, Linn JF, Wu G, Anzick SL, Eisenberger CF, Halachmi S, Cohen Y, Fomenkov A, Hoque MO, Okami K et al: CDC91L1 (PIG-U) is a newly discovered oncogene in human bladder cancer. Nat Med 2004, 10:374-381.
12. Jiang WW, Zahurak M, Zhou ZT, Park HL, Guo ZM, Wu GJ, Sidransky D, Trink B, Califano JA: Alterations of GPI transamidase subunits in head and neck squamous carcinoma. Mol Cancer 2007, 6:74.
13. Yiping Huang JKN, Barry Trink, Edward A. Ratovitski: Tobacco Smoke Activates Protein Association and Tyrosine Phosphorylation of the GPI-Transamidase Complex Subunits in Human Cancers. Journal of Epithelial Biology & Pharmacology 2009, 2:14-22.
14. Zhang XX, Ni B, Li Q, Hu LP, Jiang SH, Li RK, Tian GA, Zhu LL, Li J, Zhang XL et al: GPAA1 promotes gastric cancer progression via upregulation of GPI-anchored protein and enhancement of ERBB signalling pathway. J Exp Clin Cancer Res 2019, 38:214.
15. Guirguis-Blake JM, Beil TL, Senger CA, Coppola EL. Primary Care Screening for Abdominal Aortic Aneurysm: Updated Evidence Report and Systematic Review for the US Preventive Services Task Force. JAMA. 2019;322(22):2219. doi:10.1001/jama.2019.17021
16. US Preventive Services Task Force, Owens DK, Davidson KW, et al. Screening for Abdominal Aortic Aneurysm: US Preventive Services Task Force Recommendation Statement. JAMA. 2019;322(22):2211. doi:10.1001/jama.2019.18928
17. Reimerink JJ, Van Der Laan MJ, Koelemay MJ, Balm R, Legemate DA. Systematic review and meta-analysis of population-based mortality from ruptured abdominal aortic aneurysm. British Journal of Surgery. 2013;100(11):1405-1413. doi:10.1002/bjs.9235
18. Sweeting MJ, Balm R, Desgranges P, et al. Individual-patient meta-analysis of three randomized trials comparing endovascular versus open repair for ruptured abdominal aortic aneurysm. British Journal of Surgery. 2015;102(10):1229-1239. doi:10.1002/bjs.9852
19. Karthikesalingam A, Holt PJ, Vidal-Diez A, et al. Mortality from ruptured abdominal aortic aneurysms: clinical lessons from a comparison of outcomes in England and the USA. The Lancet. 2014;383(9921):963-969. doi:10.1016/S0140-6736(14)60109-4
20. Verhoeven EL, Kapma MR, Groen H, et al. Mortality of ruptured abdominal aortic aneurysm treated with open or endovascular repair. Journal of Vascular Surgery. 2008;48(6):1396-1400. doi:10.1016/j.jvs.2008.07.054
21. Chen Q, Chen Q, Ye Y, Wu R, Wang S, Yao C. Characteristics and Prognosis of Abdominal or Thoracic Aortic Aneurysm Patients Admitted to Intensive Care Units After Surgical Treatment: A Multicenter Retrospective Observational Study. IJGM. 2021;Volume 14:475-486. doi:10.2147/IJGM.S296125
22. Wang H, Wu S, Pan D, et al. Comparison of different intensive care scoring systems and Glasgow Aneurysm score for aortic aneurysm in predicting 28-day mortality: a retrospective cohort study from MIMIC-IV database. BMC Cardiovasc Disord. 2024;24(1):513. doi:10.1186/1s12872-024-04184-4
23. Swerdlow NJ, Wu WW, Schermerhorn ML. Open and Endovascular Management of Aortic Aneurysms. Circulation Research. 2019;124(4):647-661. doi:10.1161/CIRCRESAHA.118.313186