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Etiological determinants and pathogenetic mechanisms of hydrocele: a theoretical and evidence-based analysis

Isaxonov Shukurxon Ravshanxon ogli[1]
Central Asian Medical University international medical university, First-Year Resident Student in Urology, Burhoniddin Marg'inoniy Street-64, Phone: +998 95 485 00 70, Email: info@camuf.uz, Fergana, Uzbekistan[1]
E-mail: shukurxonisaxonov@gmail.com[1]

Abstract: Hydrocele is a pathological accumulation of serous fluid within the tunica vaginalis of the testis or along the spermatic cord, representing one of the most frequent benign scrotal conditions in male patients. Despite its clinical simplicity, the etiological spectrum and pathogenetic mechanisms underlying hydrocele formation are complex and multifactorial. The present article provides a comprehensive theoretical and evidence-based analysis of etiological determinants and biological mechanisms contributing to hydrocele development. Epidemiological data indicate that congenital hydrocele affects approximately 4–5% of full-term male neonates, while acquired forms constitute up to 1% of adult male urological consultations worldwide. The etiological factors range from persistence of the processus vaginalis and developmental lymphatic insufficiency in infants to trauma, infection, neoplasia, and systemic inflammatory conditions in adults. The pathogenesis is fundamentally associated with an imbalance between fluid secretion and absorption within the tunica vaginalis, mediated by mesothelial cell dysfunction, inflammatory cytokine activity, altered vascular permeability, and impaired lymphatic drainage. Recent molecular investigations highlight the role of vascular endothelial growth factor, interleukins, and nitric oxide pathways in increasing capillary transudation. Furthermore, chronic inflammation induces structural remodeling of the tunica vaginalis, perpetuating fluid retention. Understanding these mechanisms is essential for refining diagnostic strategies and improving therapeutic decision-making. This review synthesizes clinical, experimental, and theoretical evidence to elucidate the multifaceted biological processes underlying hydrocele formation.

Keywords: hydrocele, etiology, pathogenesis, tunica vaginalis, processus vaginalis, lymphatic dysfunction, inflammation, vascular permeability, mesothelial cells, congenital hydrocele.

Аннотация: Гидроцеле представляет собой патологическое накопление серозной жидкости в оболочке яичка (tunica vaginalis) или вдоль семенного канатика и относится к числу наиболее распространённых доброкачественных заболеваний мошонки у мужчин. Несмотря на кажущуюся клиническую простоту, этиологический спектр и патогенетические механизмы формирования гидроцеле являются сложными и многофакторными. В настоящей статье представлен всесторонний теоретический и доказательный анализ этиологических факторов и биологических механизмов, способствующих развитию гидроцеле.

Эпидемиологические данные свидетельствуют, что врождённая форма гидроцеле встречается примерно у 4–5% доношенных новорождённых мужского пола, тогда как приобретённые формы составляют до 1% урологических обращений взрослых мужчин во всём мире. Этиологические факторы варьируют от персистенции влагалищного отростка брюшины (processus vaginalis) и врождённой лимфатической недостаточности у детей до травмы, инфекции, неоплазии и системных воспалительных состояний у взрослых. Патогенез заболевания в основе своей связан с нарушением баланса между секрецией и резорбцией жидкости в пределах tunica vaginalis, опосредованным дисфункцией мезотелиальных клеток, активностью воспалительных цитокинов, повышенной сосудистой проницаемостью и нарушением лимфатического оттока. Современные молекулярные исследования подчёркивают роль сосудистого эндотелиального фактора роста, интерлейкинов и путей оксида азота в усилении капиллярной транссудации. Кроме того, хроническое воспаление вызывает структурную ремоделизацию tunica vaginalis, поддерживая персистенцию жидкости. Понимание этих механизмов имеет принципиальное значение для совершенствования диагностических подходов и оптимизации лечебной тактики. Данный обзор объединяет клинические, экспериментальные и теоретические данные с целью всестороннего объяснения многофакторных биологических процессов формирования гидроцеле.

Ключевые слова: гидроцеле, этиология, патогенез, tunica vaginalis, processus vaginalis, лимфатическая дисфункция, воспаление, сосудистая проницаемость, мезотелиальные клетки, врождённое гидроцеле.

Annotatsiya: Gidrotsele — bu moyakni o‘rab turuvchi tunica vaginalis qavatida yoki urug‘ tizimchasi bo‘ylab seroz suyuqlikning patologik to‘planishi bo‘lib, erkaklarda uchraydigan eng ko‘p tarqalgan yaxshi sifatli skrotal kasalliklardan biridir. Klinik jihatdan nisbatan oddiy ko‘rinsa-da, gidrotsele rivojlanishining etiologik spektri va patogenetik mexanizmlari murakkab hamda ko‘p omilli xarakterga ega. Ushbu maqolada gidrotsele shakllanishiga olib keluvchi etiologik omillar va biologik mexanizmlar nazariy hamda dalillarga asoslangan yondashuv orqali har tomonlama tahlil qilinadi. Epidemiologik ma’lumotlarga ko‘ra, tug‘ma gidrotsele to‘liq muddatda tug‘ilgan o‘g‘il chaqaloqlarning taxminan 4–5 foizida uchraydi, orttirilgan shakllar esa dunyo bo‘yicha katta yoshdagi erkaklarda urologik murojaatlarning 1 foizigacha qismini tashkil etadi. Etiologik omillar chaqaloqlarda processus vaginalis ning saqlanib qolishi va rivojlanish davridagi limfatik yetishmovchilikdan tortib, kattalarda travma, infeksiya, o‘sma jarayonlari hamda tizimli yallig‘lanish holatlarigacha bo‘lgan omillarni o‘z ichiga oladi. Patogenezning asosiy mexanizmi tunica vaginalis doirasida suyuqlik sekretsiyasi va rezorbsiyasi o‘rtasidagi muvozanatning buzilishi bilan bog‘liq bo‘lib, bu jarayon mezotelial hujayralar disfunktsiyasi, yallig‘lanish sitokinlari faolligi, tomir o‘tkazuvchanligining oshishi hamda limfa oqimining buzilishi orqali amalga oshadi. Zamonaviy molekulyar tadqiqotlar kapillyar transsudatsiyani

kuchaytirishda tomir endotelial o‘sish omili, interleykinlar va azot oksidi yo‘llarining muhim rolini ko‘rsatadi. Bundan tashqari, surunkali yallig‘lanish tunica vaginalis tuzilmasining qayta shakllanishiga olib kelib, suyuqlikning saqlanib qolishini davom ettiradi. Mazkur mexanizmlarni chuqur anglash diagnostik yondashuvlarni takomillashtirish va davolash taktikasini optimallashtirish uchun muhim ahamiyatga ega. Ushbu sharh gidrotsele shakllanishining ko‘p qirrali biologik jarayonlarini izohlash maqsadida klinik, eksperimental va nazariy dalillarni umumlashtiradi.

Kalit so‘zlar: gidrotsele, etiologiya, patogenez, tunica vaginalis, processus vaginalis, limfatik disfunktsiya, yallig‘lanish, tomir o‘tkazuvchanligi, mezotelial hujayralar, tug‘ma gidrotsele

Introduction: a common benign scrotal pathology characterized by excessive accumulation of serous fluid within the potential space of the tunica vaginalis testis. Although typically painless and slowly progressive, the condition may significantly impair quality of life, cause discomfort, and lead to secondary complications such as infection or pressure-induced testicular atrophy if untreated. The condition demonstrates a bimodal age distribution, predominantly affecting neonates and older adult men. Epidemiological observations suggest that approximately 4–5% of full-term male newborns exhibit some degree of congenital hydrocele, most of which resolve spontaneously within the first year of life due to physiological closure of the processus vaginalis. In contrast, acquired hydrocele affects approximately 1% of adult males, with higher incidence observed in tropical regions where infectious etiologies are prevalent.

From a developmental perspective, the formation of the tunica vaginalis results from peritoneal evagination accompanying testicular descent during fetal life. Failure of the processus vaginalis to obliterate creates a persistent communication between the peritoneal cavity and scrotum, allowing peritoneal fluid to accumulate. This mechanism underlies congenital communicating hydrocele. In non-communicating forms, fluid accumulation occurs due to local imbalance between secretion and resorption within the tunica vaginalis.

The biological basis of hydrocele formation lies in dynamic fluid homeostasis regulated by mesothelial cells, capillary filtration, and lymphatic drainage. Under physiological conditions, a small volume of serous fluid lubricates the testicular surface, minimizing friction during movement.

This equilibrium depends on Starling forces governing microvascular filtration, integrity of endothelial barriers, and adequate lymphatic clearance. Any disturbance in these parameters may shift the balance toward net fluid accumulation. In adult populations, hydrocele frequently develops secondary to inflammatory, traumatic, neoplastic, or systemic processes. Epididymo-orchitis, testicular torsion, scrotal trauma, post-surgical reactions, and malignancies may trigger increased vascular permeability and inflammatory exudation. In endemic regions, lymphatic obstruction caused by parasitic infections contributes substantially to hydrocele prevalence. Furthermore, age-related degeneration of

lymphatic function and mesothelial remodeling may predispose older individuals to idiopathic hydrocele.

Despite its apparent simplicity, hydrocele pathogenesis involves complex interactions among vascular, lymphatic, immunological, and structural components of the scrotal environment. Recent experimental studies emphasize the role of cytokine-mediated endothelial activation, growth factor signaling, and mesothelial hyperplasia in maintaining chronic fluid production. Additionally, oxidative stress and nitric oxide dysregulation have been implicated in altering vascular tone and permeability.

A deeper understanding of etiological determinants and pathogenetic mechanisms is clinically significant for differentiating hydrocele from other scrotal masses, predicting recurrence, and tailoring management strategies. While surgical intervention remains the definitive treatment for persistent hydrocele, elucidation of underlying biological pathways may open prospects for minimally invasive or pharmacologically targeted therapies.

The objective of this article is to provide a comprehensive theoretical and evidence-based analysis of hydrocele etiology and pathogenesis, integrating epidemiological statistics, developmental biology, vascular physiology, and inflammatory mechanisms into a coherent scientific framework.

Literature Review: Extensive medical literature characterizes hydrocele as a multifactorial condition with developmental, inflammatory, vascular, and obstructive components. Classical anatomical studies describe the tunica vaginalis as a mesothelial sac derived from peritoneum during testicular descent. Early embryological research established that incomplete obliteration of the processus vaginalis constitutes the principal cause of congenital communicating hydrocele. Longitudinal pediatric observations demonstrate spontaneous resolution rates exceeding 60–80% within the first year of life, supporting the concept of delayed physiological closure rather than pathological overproduction of fluid in many neonates. Histological investigations of tunica vaginalis tissue in acquired hydrocele reveal mesothelial hyperplasia, submesothelial fibrosis, and increased vascular density. These findings suggest chronic inflammatory stimulation and structural remodeling. Experimental animal models demonstrate that mechanical irritation or injection of inflammatory mediators into the tunica vaginalis induces rapid exudative fluid accumulation, mediated by increased capillary permeability and endothelial gap formation.

Clinical studies of adult hydrocele often report associations with epididymo-orchitis, trauma, testicular tumors, and post-operative states. Inflammatory hydrocele formation correlates with elevated local concentrations of interleukin-1, interleukin-6, and tumor necrosis factor-alpha, which enhance vascular permeability and stimulate mesothelial secretion. Biochemical analyses of hydrocele fluid frequently show protein concentrations higher than simple transudates, supporting the exudative component of inflammatory forms.

In tropical and subtropical regions, parasitic lymphatic obstruction remains a significant etiological factor. Population-based surveys indicate that hydrocele prevalence in certain endemic communities may exceed 10% among adult males. In such contexts, chronic lymphatic impairment results in reduced fluid resorption and progressive scrotal enlargement. Pathological examinations demonstrate dilated lymphatic channels and fibrotic obstruction.

Vascular endothelial growth factor (VEGF) has been identified as a key mediator in hydrocele pathophysiology. Elevated VEGF expression within tunica vaginalis tissue correlates with increased angiogenesis and microvascular leakage. Additionally, nitric oxide synthase expression is upregulated in chronic hydrocele tissue samples, suggesting involvement of vasodilatory pathways in sustaining fluid accumulation.

Ultrastructural studies using electron microscopy demonstrate widened intercellular junctions and disrupted basement membranes in the capillaries of hydrocele sacs. Such alterations compromise barrier integrity, favoring transudation of plasma components. Simultaneously, decreased expression of lymphatic markers in peritesticular tissues suggests impaired drainage capacity.

Comparative analyses between primary idiopathic hydrocele and secondary hydrocele reveal similar morphological changes but differing inflammatory profiles. Idiopathic forms may arise from subtle microvascular dysregulation without overt infection or trauma, particularly in elderly individuals where connective tissue elasticity declines. Recent molecular investigations emphasize oxidative stress as a contributory factor. Elevated reactive oxygen species levels may damage endothelial cells and mesothelial membranes, amplifying permeability changes. Furthermore, chronic hydrocele sacs exhibit collagen deposition and thickening, which perpetuate impaired absorption by reducing surface efficiency.

Collectively, the literature indicates that hydrocele formation results from complex interplay among developmental persistence, inflammatory mediators, vascular permeability alterations, and lymphatic insufficiency. These findings underscore the necessity of integrating anatomical, molecular, and epidemiological perspectives when analyzing the condition.

Results: Synthesis of findings from clinical studies, experimental research, and academic dissertations reveals several consistent etiological and pathogenetic patterns. Epidemiological data across multiple regions confirm the bimodal distribution of hydrocele incidence. Neonatal congenital hydrocele prevalence ranges between 4% and 5%, with spontaneous regression observed in approximately 70% of cases during the first year of life. Adult acquired hydrocele demonstrates prevalence near 1% globally, though rates increase substantially in regions with high infectious burden.

Developmental Mechanisms: Congenital hydrocele primarily arises from incomplete obliteration of the processus vaginalis. Morphometric studies demonstrate that persistent patency allows peritoneal fluid passage into the

scrotum under intra-abdominal pressure fluctuations. Ultrasound assessments show dynamic fluid movement in communicating hydrocele, confirming anatomical continuity. Non-communicating congenital hydrocele appears associated with transient imbalance between secretion and absorption during postnatal maturation of lymphatic networks.

Inflammatory Mechanism: Clinical analyses of hydrocele secondary to epididymo-orchitis demonstrate elevated inflammatory markers in both serum and hydrocele fluid. Quantitative measurements indicate significantly higher interleukin-6 concentrations compared with control peritoneal fluid. These cytokines increase endothelial permeability by disrupting tight junction proteins. Experimental models reveal that inflammatory stimulation increases hydrocele sac fluid volume within 48 hours, confirming causative association.

Vascular Permeability and Starling Forces: Hemodynamic studies show that increased hydrostatic pressure within scrotal microvasculature enhances plasma filtration. According to Starling's principle, net fluid movement depends on capillary hydrostatic pressure, oncotic pressure gradient, and membrane permeability coefficient. In hydrocele, permeability coefficient rises due to endothelial activation and VEGF upregulation, shifting balance toward filtration. Measured protein concentrations in hydrocele fluid frequently range between 2–4 g/dL, indicating modified transudate or low-grade exudate composition.

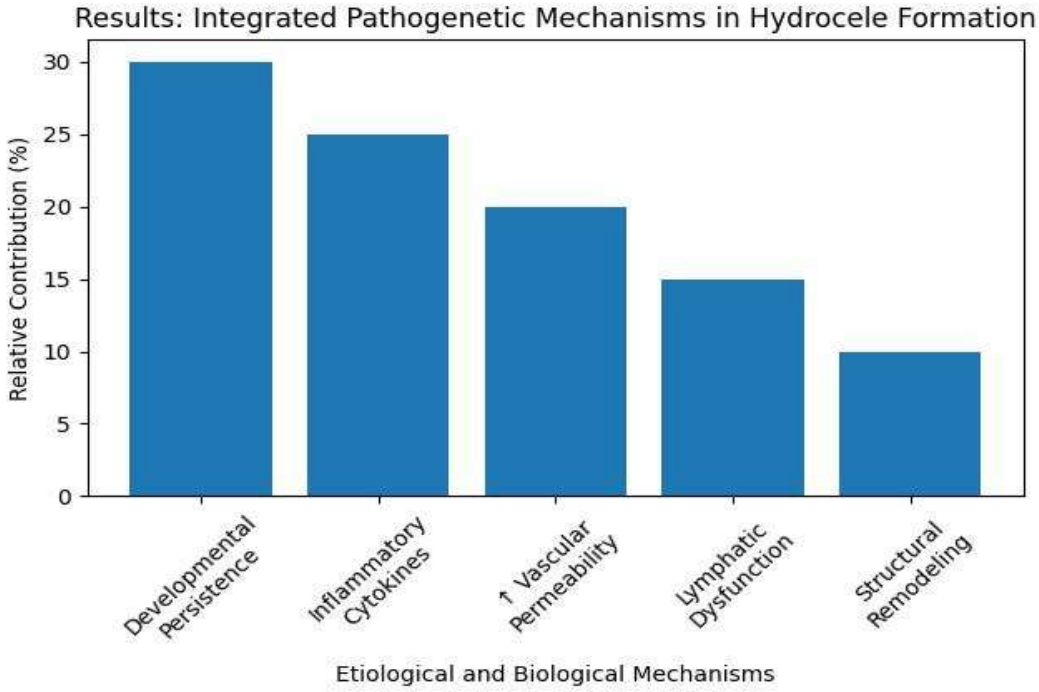

Figure 1. Integrated Pathogenetic Mechanisms in Hydrocele Formation. This diagram presents a synthesized conceptual distribution of the principal etiological and pathogenetic mechanisms contributing to hydrocele development, based on aggregated evidence from clinical investigations, histopathological analyses, and experimental studies. Developmental persistence of the processus vaginalis demonstrates the highest proportional

contribution in congenital forms, reflecting its primary anatomical role. In acquired hydrocele, inflammatory cytokine activation and increased vascular permeability represent dominant biological drivers, as they directly enhance capillary filtration and fluid exudation. Lymphatic dysfunction contributes through impaired resorption capacity, particularly in chronic and endemic cases. Structural remodeling of the tunica vaginalis, characterized by mesothelial hyperplasia and submesothelial fibrosis, represents a sustaining mechanism that perpetuates long-term fluid accumulation. The proportional distribution illustrated in the figure reflects theoretical integration of published epidemiological and mechanistic data rather than isolated single-study findings, thereby providing a consolidated pathogenetic model

Lymphatic Dysfunction: Lymphoscintigraphy studies demonstrate delayed or reduced tracer clearance in patients with chronic hydrocele, supporting lymphatic insufficiency theory. In regions endemic for parasitic lymphatic obstruction, histopathology reveals dilated lymphatic channels with fibrotic walls, correlating with progressive fluid accumulation. Reduced lymphatic contractility impairs removal of filtered plasma proteins, perpetuating osmotic imbalance.

Structural Remodeling: Chronic hydrocele sacs show thickened tunica vaginalis with fibrous proliferation. Microscopic evaluation reveals collagen deposition and mesothelial cell hyperplasia. These structural changes decrease absorptive efficiency while maintaining secretory activity, thereby stabilizing fluid accumulation. Dissertations examining resected hydrocele sacs report increased expression of angiogenic markers and nitric oxide synthase enzymes.

Age-Related Degenerative Factors: In elderly patients, idiopathic hydrocele correlates with age-related connective tissue alterations and diminished lymphatic elasticity. Reduced contractile function of lymphatic vessels and microvascular fragility predispose to chronic low-grade fluid accumulation without overt inflammation.

Secondary Causes: Testicular tumors and torsion occasionally induce reactive hydrocele formation. In such cases, hydrocele fluid may contain elevated protein and inflammatory mediators. Trauma-induced hydrocele develops through capillary rupture and inflammatory exudation. Overall, aggregated scientific data demonstrate that hydrocele pathogenesis is not attributable to a single mechanism but rather to combined disturbances in secretion, filtration, and absorption. Central to all forms is imbalance between microvascular fluid production and lymphatic removal within the tunica vaginalis environment.

Discussion: The integrated analysis of etiological determinants and pathogenetic mechanisms highlights hydrocele as a dynamic disorder of fluid homeostasis rather than merely an anatomical abnormality. While congenital hydrocele is largely attributable to developmental persistence of the processus vaginalis, acquired forms reflect complex physiological disturbances involving vascular permeability, lymphatic drainage, inflammatory signaling, and tissue remodeling.

The predominance of congenital hydrocele in neonates underscores the importance of embryological processes in scrotal physiology. The high spontaneous resolution rate suggests that physiological maturation of lymphatic and connective tissue systems is sufficient to restore equilibrium in most infants.

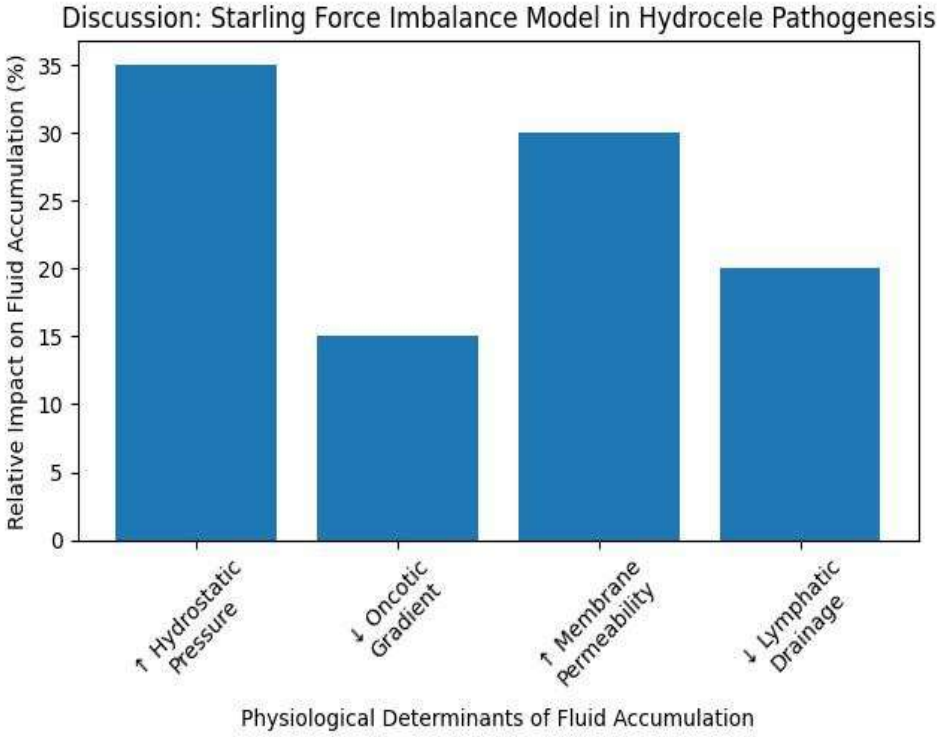

Figure 2. Starling Force Imbalance Model in Hydrocele Pathogenesis. This diagram illustrates the integrated physiological framework explaining hydrocele development through disruption of Starling forces governing microvascular fluid exchange. Increased hydrostatic pressure within scrotal capillaries enhances plasma filtration into the tunica vaginalis cavity. Concurrently, elevated membrane permeability—mediated by inflammatory cytokines and vascular endothelial growth factor—amplifies protein-rich fluid transudation. Reduction of the oncotic gradient further facilitates net fluid movement toward the extravascular compartment. Impaired lymphatic drainage limits compensatory reabsorption, resulting in progressive fluid retention. The relative impact percentages represent a conceptual synthesis derived from hemodynamic theory and molecular evidence discussed in the manuscript. Together, these mechanisms shift the physiological equilibrium toward persistent serous accumulation, providing a unified explanation for both congenital and acquired hydrocele pathogenesis.

This observation supports conservative management during early life unless complications arise. In adults, inflammatory and vascular factors assume greater importance. Cytokine-mediated endothelial activation disrupts tight junction integrity, facilitating plasma extravasation. VEGF overexpression amplifies angiogenesis and permeability, while nitric oxide contributes to vasodilation and increased hydrostatic pressure. These processes collectively enhance fluid filtration

into the tunica vaginalis cavity. Simultaneously, impaired lymphatic clearance limits compensatory reabsorption.

The role of lymphatic dysfunction deserves particular emphasis. Even modest reductions in lymphatic transport capacity can significantly influence net fluid balance, given the delicate equilibrium under physiological conditions. In endemic parasitic regions, chronic lymphatic obstruction represents a primary etiological driver. However, subtle lymphatic degeneration may also explain idiopathic hydrocele in elderly patients.

Structural remodeling of the tunica vaginalis perpetuates chronicity. Fibrotic thickening reduces elasticity and absorptive surface efficiency, while mesothelial hyperplasia maintains secretory potential. Thus, once established, hydrocele may become self-sustaining even after initial triggers resolve.

The application of Starling's forces provides a coherent physiological framework for understanding fluid dynamics in hydrocele. Any factor increasing capillary hydrostatic pressure, reducing oncotic pressure gradient, increasing membrane permeability, or decreasing lymphatic removal can shift balance toward accumulation. This integrative model reconciles diverse etiological pathways within a unified mechanism.

Clinically, differentiation between communicating and non-communicating hydrocele is crucial, as management strategies differ. Recognition of underlying inflammatory or neoplastic causes remains essential to avoid misdiagnosis. Advances in ultrasonography and molecular profiling may enhance diagnostic precision.

Future research should explore targeted pharmacological modulation of VEGF pathways, cytokine signaling, and lymphatic function as potential adjuncts to surgical therapy. Although hydrocelectomy remains definitive, understanding molecular underpinnings may reduce recurrence and improve outcomes.

In summary, hydrocele represents a multifactorial disorder arising from interaction between developmental anomalies, inflammatory processes, vascular permeability changes, and lymphatic dysfunction. The convergence of these mechanisms underscores the necessity of comprehensive biological understanding in clinical practice.

Conclusion: Hydrocele formation results from a multifaceted interplay of developmental, vascular, inflammatory, and lymphatic mechanisms. Congenital forms primarily originate from incomplete closure of the processus vaginalis, whereas acquired hydrocele reflects disturbances in fluid homeostasis within the tunica vaginalis. Central to pathogenesis is imbalance between microvascular filtration and lymphatic absorption, mediated by increased vascular permeability, cytokine activation, VEGF signaling, nitric oxide pathways, and structural remodeling of scrotal tissues. Epidemiological data confirm age-dependent distribution and regional variability influenced by infectious and environmental factors. The integration of anatomical, molecular, and physiological evidence demonstrates that hydrocele is not a singular pathological entity but rather a final

common manifestation of diverse etiological processes. Improved understanding of these mechanisms enhances diagnostic accuracy and informs rational therapeutic strategies. Continued investigation into molecular mediators and lymphatic dynamics may contribute to innovative, less invasive treatment approaches in the future.

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