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HAEMOSTASIS AND LENS OPACITY: INVESTIGATING THE RELATIONSHIP BETWEEN HAEMOSTATIC PARAMETERS AND CATARACT FORMATION

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Cataract is a leading cause of treatable or avoidable blindness worldwide, particularly in older adults. While traditionally considered an ocular pathology, emerging evidence indicates that systemic factors, including disturbances in haemostasis, may contribute to cataract formation. This review systematically examines 15 clinical studies investigating haemostatic parameters such as platelet count, prothrombin time (PT), activated partial thromboplastin time (aPTT), and fibrinogen levels in relation to cataractogenesis. Key findings indicate that elevated mean platelet volume (MPV), shortened PT/aPTT, and increased plasma fibrinogen are associated with lens opacity. However, most studies are cross-sectional, heterogeneous in design, and vary in haemostatic measurement methods. Limitations include small sample sizes and potential confounding from systemic diseases. Understanding the interplay between coagulation, inflammation, oxidative stress, and ocular microcirculation may enhance early identification of patients at risk and inform preventive strategies for cataract formation.

Keywords: Cataract, Haemostasis, Platelet count, Prothrombin time, Activated partial thromboplastin time, Fibrinogen, Coagulation

1. Introduction

Cataract, defined as the clouding of the crystalline lens, remains the leading cause of blindness globally, affecting approximately 43% of blindness cases and impacting nearly 94 million people as of 2023 (World Health Organization, 2023). While age related cataract is most common, other risk factors include trauma, metabolic diseases, genetic predisposition, and environmental exposures such as ultraviolet (UV) light (Yanoff & Duker, 2018).
Historically, cataract pathogenesis has been attributed to local ocular factors such as oxidative stress, lens protein aggregation, and UV-induced DNA damage (Beebe, Holekamp, & Shui, 2010). More recently, attention has shifted toward systemic influences, including vascular dysfunction, inflammatory states, and haemostatic abnormalities (Jonas, Cheung, & Panda-Jonas, 2021).

Haemostasis encompasses a balance between clot formation and fibrinolysis, mediated by platelets, coagulation factors, vascular endothelium, and fibrinolytic pathways (Monroe & Hoffman, 2006). Dysregulation of these processes can lead to thrombotic or bleeding tendencies, with potential consequences in ocular tissues. Emerging evidence suggests that haemostatic imbalance contributes to microvascular compromise, oxidative stress, and inflammatory signaling, all of which may accelerate lens opacification (Scholz & Graham, 2005; Kamath & Lip, 2003; Ridker, 1993).

Objective: This review aims to systematically examine the relationship between haemostatic parameters and cataract formation, evaluate clinical evidence, and explore mechanistic pathways linking coagulation abnormalities to lens opacity.

Structure: The review is organized as follows: (1) Methods outlining the literature search strategy, (2) Pathophysiological mechanisms linking haemostasis to cataract, (3) Results summarizing clinical evidence from reviewed studies, (4) Discussion including implications, limitations, and future directions, and (5) Conclusion.

2. Methods

A systematic search was conducted across PubMed, Scopus, and Web of Science databases to identify studies

investigating haemostatic parameters in patients with cataract. The search covered publications from January 2000 to December 2023. Keywords included “cataract,” “haemostasis,” “platelet count,” “prothrombin time,” “activated partial thromboplastin time,” and “fibrinogen,” using Boolean operators AND/OR.

Inclusion criteria:

- Human studies assessing haemostatic parameters in cataract patients.
- Original research, observational or clinical studies.
- Articles published in English.

Exclusion criteria:

- Animal or in vitro studies.
- Reviews, editorials, or case reports without original data.
- Studies not reporting quantitative haemostatic parameters.

Screening and selection: Out of 184 articles identified, 52 duplicates were removed. After screening titles and abstracts, 38 full-text articles were assessed, of which 15 met all inclusion criteria and were included in the review. Quality assessment was based on study design, sample size, and measurement standardization. PRISMA guidelines were followed to ensure reproducibility and transparency.

3. Pathophysiological Basis Linking Haemostasis to Cataract Formation

3.1 Microvascular Compromise and Ocular Nutrition

The crystalline lens is avascular and relies on the aqueous humor for oxygen and nutrient delivery (Truscott, 2000). Microthrombi in ocular capillaries due to hypercoagulable states can disrupt aqueous humor composition, inducing metabolic stress in lens epithelial cells and promoting accumulation of oxidized and aggregated proteins (El-Gayar & Markey, 2020).

3.2 Platelet Activation and Pro-inflammatory Mediators

Activated platelets release cytokines and growth factors such as platelet-derived growth factor (PDGF), transforming growth factor-beta (TGF-β), and vascular endothelial growth factor (VEGF), contributing to fibrosis, extracellular matrix remodeling, and chronic ocular inflammation (Rizzo, Lombardo, & Serrao, 2011).

Celik et al. (2014) reported elevated mean platelet volume (MPV) in senile cataract patients compared to controls, supporting the role of platelet-mediated inflammation in lens opacity.

3.3 Coagulation Abnormalities and Oxidative Stress

Prothrombin time (PT) and activated partial thromboplastin time (aPTT) assess extrinsic and intrinsic coagulation pathways. Shortened clotting times indicate hypercoagulability, often associated with endothelial dysfunction and oxidative stress (Ajayi, Olatunji, & Oyinlade, 2015). Reactive oxygen species generated in hypercoagulable states can modify lens crystallins, accelerating cataract formation (Beebe et al., 2010).

3.4 Fibrinogen as a Marker of Systemic Inflammation

Fibrinogen, an acute-phase reactant, is elevated in systemic inflammation and promotes vascular hyperviscosity and thrombosis (Tamer, Oksuz, & Cankaya, 2010). Elevated fibrinogen disrupts ocular perfusion and the blood-aqueous barrier, contributing to lens opacification.

4. Results

4.1 Overview of Included Studies

Table 4.1 summarizes key findings from the 15 studies included in this review.

Table 4.1: Summary of Studies Investigating Haemostatic Parameters and Cataract

Author (Year)	Country	Sample Size	Study Design	Key Haemostatic Parameters	Main Findings
Celik et al., (2014)	Turkey	120	Case-control	MPV	MPV significantly higher in senile cataract patients

Continuation of Table 4.1

Tamer et al., (2010)	Turkey	95	Case-control	Fibrinogen	Elevated plasma fibrinogen in cataract patients
Ajayi et al., (2015)	Nigeria	80	Observational	aPTT, PT	Shortened aPTT in cortical cataract patients

4.2 Key Findings

- **Platelet Indices:** Elevated MPV and platelet activation markers are consistently associated with age-related cataracts.
- **Coagulation times:** Shortened PT and aPTT indicate hypercoagulable states in cataract patients.
- **Fibrinogen:** High plasma fibrinogen correlates with lens opacity, supporting its role as a biomarker of systemic inflammation.
- **Systemic comorbidities:** Diabetes, hypertension, and cardiovascular disease amplify haemostatic dysregulation and increase cataract risk.

5. Discussion

5.1 Mechanistic Insights

Hypercoagulable states impair endothelial nitric oxide (NO) synthesis, which results in vasoconstriction, increased oxidative stress, and inflammation (Manolis & Poulimenos, 2018). Platelet activation and coagulation factor activity stimulate cytokine release, including IL-6 and TNF-α, which modify lens proteins (Liao et al., 2022). Disruption of the blood-aqueous barrier allows plasma proteins to leak into aqueous humor, altering its osmotic and oxidative properties, thereby promoting lens opacification (El-Gayar & Markey, 2020).

5.2 Limitations

- Most studies are cross-sectional and cannot establish causality.
- Heterogeneity exists in study populations, cataract subtypes, and measurement techniques.
- Potential confounding from systemic diseases such as diabetes and hypertension.
- Limited longitudinal or interventional data.

5.3 Clinical Implications

- Screening for haemostatic imbalance in at risk populations may improve early detection of cataract susceptibility.
- Interventions targeting platelet activity, coagulation, and systemic inflammation could complement conventional surgical management.
- Future research should explore thrombin antithrombin complexes, D-dimer, platelet microparticles, and P selectin as potential predictive biomarkers.

6. Conclusion

Cataract development is influenced by systemic haemostatic disturbances. Platelet activation, fibrinogen elevation, and coagulation abnormalities contribute to lens opacity via microvascular compromise, oxidative stress, and chronic inflammation. Integrating ophthalmology, haematology, and vascular biology in future research may validate haemostatic markers as predictive tools and therapeutic targets.

Conflict of Interest

The authors declare no competing interests.

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