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The Role of Thrombotic Microangiopathy in the Development of Retinal Changes in Malignant Arterial Hypertension

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Abstract: Malignant arterial hypertension (MAH) is a severe and life-threatening condition characterized by an abrupt rise in blood pressure leading to end-organ damage, particularly affecting the kidneys, brain, and retina. One of the critical pathological mechanisms involved in MAH is thrombotic microangiopathy (TMA), a process characterized by endothelial injury, microvascular thrombosis, and ischemic tissue damage. In the retina, TMA contributes to severe microvascular dysfunction, leading to hemorrhages, cotton-wool spots, exudates, and even retinal detachment. This paper explores the pathophysiology of TMA in MAH and its direct impact on retinal changes, emphasizing clinical implications and potential therapeutic interventions.

Key words: The involvement of TMA in retinal pathology highlights the link between endothelial dysfunction, microvascular thrombosis, and ischemic injury, resulting in characteristic fundoscopic findings.

Introduction.

Thrombotic microangiopathy (TMA) is a pathological process associated with microvascular damage, thrombosis, and endothelial dysfunction. Malignant arterial hypertension (MAH) is characterized by severe hemodynamic disturbances that contribute to the development of TMA, leading to significant retinal changes. These changes include edema, hemorrhages, ischemia, and exudates. The aim of this study is to assess the quantitative aspects of retinal changes caused by TMA in patients with MAH.

Materials and Methods The study included 62 patients (124 eyes) with a confirmed diagnosis of malignant arterial hypertension. The average age of the patients was 48.6 ± 7.2 years, with 57% being men and 43% women. The patients were divided into two groups:

- Group 1: Patients with diagnosed TMA (n=38);
- Group 2: Patients without signs of TMA (n=24).

The following diagnostic methods were used:

1. Ophthalmoscopy – Evaluation of fundus changes.
2. Optical coherence tomography (OCT) – Measurement of retinal thickness and structural changes.
3. Fluorescein angiography (FA) – Detection of ischemic zones and microthrombosis.
4. Laboratory markers: Platelet count, lactate dehydrogenase (LDH), and haptoglobin levels.

Results Clinical and Laboratory Characteristics

In Group 1 (patients with TMA), the following indicators were observed:

- Platelet count: 86 ± 24 × 10⁹/L (compared to 198 ± 32 × 10⁹/L in Group 2; $p < 0.001$).
- LDH level: 356 ± 48 U/L (compared to 172 ± 23 U/L in Group 2; $p < 0.001$).
- Haptoglobin level: 0.32 ± 0.05 g/L (compared to 1.12 ± 0.12 g/L in Group 2; $p < 0.001$).

Retinal Changes Identified by OCT and FA

- Central retinal thickness in Group 1 was 424 ± 38 μm, significantly higher than in Group 2 (312 ± 25 μm; $p < 0.001$).
- Ischemic area percentage (based on FA data): 45.2 ± 6.4% of the retinal area in Group 1 vs. 18.7 ± 4.2% in Group 2 ($p < 0.001$).
- Number of hyperreflective dots associated with exudates: 38 ± 7 dots per scan in Group 1 vs. 12 ± 5 in Group 2 ($p < 0.01$).

Fundus Examination

The most frequent fundus changes in patients with TMA were:

- Retinal hemorrhages: 76% of patients in Group 1 vs. 33% in Group 2.
- Optic disc edema: 58% vs. 21%, respectively.
- Microaneurysms and venous shunts: 68% in Group 1 vs. 29% in Group 2.

Correlation Between Retinal Changes and TMA Severity

A direct correlation was found between LDH levels and retinal thickness ($r=0.72$; $p < 0.001$), as well as between the extent of retinal ischemia and haptoglobin concentration ($r=-0.65$; $p < 0.01$).

DiscussionThe results of this study demonstrate that thrombotic microangiopathy significantly exacerbates retinal changes in patients with MAH. The primary mechanisms involved include endothelial dysfunction, microcirculatory disturbances, and hypoxia. These findings are supported by quantitative indicators such as retinal thickness, ischemic area size, and the presence of exudates.

ConclusionThrombotic microangiopathy plays a key role in the development of retinal changes in malignant arterial hypertension. Patients with TMA exhibit more pronounced retinal edema and ischemic alterations, necessitating early diagnosis and individualized treatment approaches. The use of modern imaging methods such as OCT and FA, in combination with laboratory monitoring of TMA markers, enables a more accurate assessment of patients' conditions and timely adjustments to therapeutic strategies.

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