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Bilateral choroidal detachment: A novel feature of hypertensive chorioretinopathy in a 26-year-old man with end-stage renal disease

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1. Case report

We describe a case of a 26-year-old man with multiple bilateral choroid detachments due to hypertensive end-stage renal failure. The patient, with congenital left kidney agenesis and chronic kidney disease (CKD) in hemodialysis treatment, was referred to the Ophthalmic Emergency Room of Policlinic of Bari complaining of bilateral vision loss. The patient's mean blood pressure was 165/95 with ramipril, amlodipine, clonidine, nebivolol, and aspirin treatment. According to the patient's daily medication, none of the drugs he was taking could induce choroid detachment or macular edema. There was no family history of any vascular retinal disease. At hospital admission, the patient's best-corrected visual acuity (BCVA) was bilaterally 20/200 (Snellen charts). Intraocular pressure (IOP) measured with Perkins applanation tonometer was 8 mmHg in both eyes. Bilateral extrinsic ocular motility and slit-lamp examination were unremarkable.

After dilation with tropicamide, fundus examination showed bilateral mild optic nerve-head pallor, vascular tortuosity, retinal arteriolar narrowing, and arteriovenous nicking, microaneurysms, hemorrhages, cotton-wool spots, multiple choroidal detachments, and macular edema in the left eye.

Color fundus photos and ocular ultrasonography confirmed these findings.

The blood pressure was found to be 210/110 mmHg at hospitalization. The nephrological evaluation was made, doxazosin was administered una tantum, and a hemodialysis session was planned. On the next day, the corticosteroid therapy was postponed until a cardiological evaluation was made. The consultation reported severe hypertension with hypertrophic, dilated and hypocontractile left ventricle. Nitroglycerin was administered and corticosteroid was not recommended, according to the cardiologist. Besides, cardiologists have doubled the ramipril dose.

During the next days, blood pressure stabilized at around 150/85 mmHg, and nephrological and cardiological consultations were repeated. After concluding that the patient's issues could be referred to a systemic condition, he was discharged and referred to a cardiologist to find the target pressure.

One month after discharge, the patient presented at the follow-up visit: He had changed his antihypertensive therapy, and his blood pressure was under control (135/70 mmHg); no corticosteroid therapy was done. A complete ophthalmological examination was due, with bilateral BCVA 20/20, bilateral IOP 12 mmHg, and a full resolution of choroidal detachments and macular edema.

The patient was also monitored by the cardiologist and nephrologist, who confirmed systemic antihypertensive therapy. The patient is rechecked every month by ophthalmologist, cardiologist, and nephrologist to plan any adjustments to antihypertensive therapy and dialysis treatment.

2. Discussion

Hypertensive choroidopathy is due to acute hypertension. It occurs in relatively young subjects due to their high vessel elasticity.[1] Hypertensive choroidal changes may occur in patients with essential

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hypertension, and renal disease, causing choroidal vasculature dysfunction, with progressive ischemic damage to the retinal pigment epithelium (RPE).[2] Hypotony is the primary etiological factor in fluid accumulation, with choroidal effusion serving as a secondary complication that further compromises aqueous humor production.[3] Bansal et al. described patients with short-term bilateral visual loss due to vascular compromission due to hypotony after hemodialysis, specifying the close collaboration between physicists and ophthalmologists.[4]

Classic features include arteriolar narrowing, arterio-venous nicking, flame hemorrhages, cotton-wool spots, macular edema or macular detachment, disk swelling, optic nerve ischemia caused by hypoperfusion and ischemia of the peripapillary and sub-macular choroid.[5]

We chose repeated and close follow-ups because if hypertension were to become chronic, it would lead to non-reversible vascular retinal complications such as occlusions and macroaneurysms; at the fundoscopic examination, widening of the arteriole reflex, arteriovenous crossing signs, wire arterioles, and copper or silver-colored arterioles.[6]

We suggest an important clinical correlation between systemic hypertensive condition and hypertensive choroidopathy, with significant visual loss for the patient.

3. Conclusion

Systemic hypertensive status and end-stage renal failure can severely impair the patient's ocular condition anatomically and functionally. These systemic conditions and choroidal failure are clinically closely related. The hypertensive chorioretinopathy can be transient and allow total anatomical and functional resolution if the blood hypertension is promptly lowered with medication.

CRediT authorship contribution statement

Valeria Albano: Writing – review & editing, Writing – original draft, Visualization, Validation, Supervision, Software, Resources, Project administration, Investigation, Funding acquisition, Formal analysis, Conceptualization. **Maria Grazia Pignataro:** Writing – review & editing, Writing – original draft, Visualization, Supervision, Software, Resources, Investigation, Formal analysis, Data curation, Conceptualization. **Rosanna Dammacco:** Visualization, Validation, Supervision, Resources, Methodology, Data curation, Conceptualization. **Giovanni Alessio:** Visualization, Validation, Supervision, Software, Resources, Conceptualization.

Patient consent

Consent to publish this case report has been obtained from the patient in writing form.

Authorship

All authors attest that they meet the current ICMJE criteria for Authorship.

Color fundus photos (Fig. 1A–B) display peripheral temporal choroidal detachment in the right eye (A) and near-kissing choroidal detachments and macular edema in the left eye (B), and in both eyes diffuse intraretinal hemorrhages, mild optic nerve-head pallor, vascular tortuosity, retinal arteriolar narrowing, and arteriovenous nicking, cotton-wool spots.

Images C-D (Fig. 1) show multiple bullous choroidal detachments in both eyes: the majority involving the temporal sector in the right eye (C), and near-kissing choroidal detachments in the left eye (D) through standardized B-scan ultrasound.

After the 1-month, under controlled blood pressure, some choroidal folds, complete resolution of macular edema, and total reabsorption of retinal hemorrhages were shown in the follow-up color fundus photos (Fig. 2E–F).

The B-scan ultrasonography displayed a total resolution of choroidal detachments, with a mild choroidal thickening in both eyes, especially in the left eye (Fig. 2G–H).

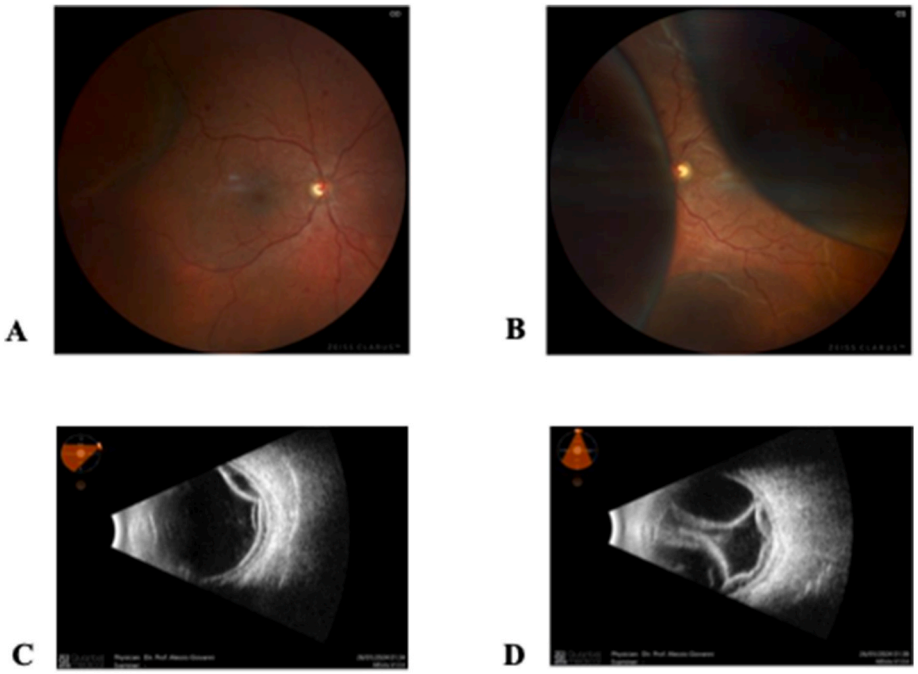

Fig. 1. The figure describes in detail the ocular findings at the onset of symptoms. Color fundus photos (A-B) exhibit peripheral temporal choroidal detachment in the right eye (A) and near-kissing choroidal detachments and macular edema in the left eye (B), and in both eyes (A-B) the diffuse intraretinal hemorrhages, mild optic nerve-head pallor, vascular tortuosity, retinal arteriolar narrowing, and arteriovenous nicking, cotton-wool spots. In C-D images, multiple bullous choroidal detachments in both eyes are shown: the majority involving the temporal sector in the right eye (C) and near-kissing choroidal detachments in the left eye (D) through standardized B-scan ultrasound.

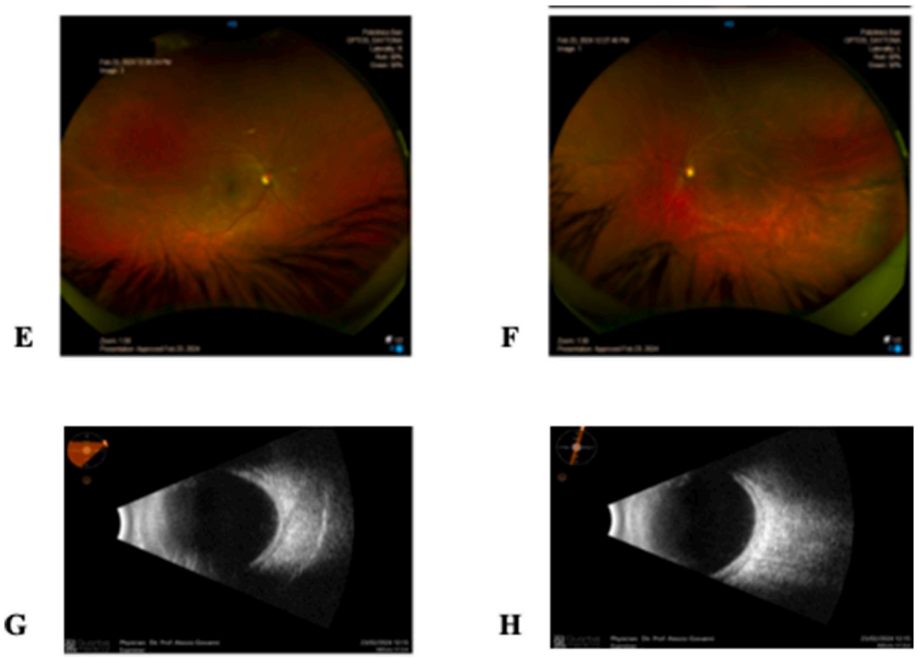

Fig. 2. The figure displays the ocular findings after one month of controlled blood pressure therapy: some choroidal folds, complete resolution of macular edema, and total reabsorption of retinal hemorrhages were shown in the follow-up color fundus photos (E-F). The B-scan ultrasonography revealed a total resolution of choroidal detachments, with a mild choroidal thickening in both eyes, especially in the left eye (G-H).

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Declaration of competing interest

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