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Case Report

A case of malignant hypertension with multi-organ injury *,**

Ikuo Misumi, MD^a,*, Mikiko Fukushima, MD^b, Kuniyasu Wada, MD^c, Joji Urata, MD^d, Koji Sato, MD^a, Miwa Nagano, MD^a, Kenichi Tsujita, MD^e

- ^a Department of Cardiology, Kumamoto City Hospital, 4-1-60, Higashi-machi, Higashi-ku, Kumamoto City, Kumamoto 862-8505, Japan
- ^b Department of Ophthalmology, Kumamoto City Hospital, 4-1-60, Higashi-machi, Higashi-ku, Kumamoto City, Kumamoto 862-8505, Japan
- ^c Department of Neurology, Kumamoto City Hospital, 4-1-60, Higashi-machi, Higashi-ku, Kumamoto City, Kumamoto 862-8505, Japan
- ^d Department of Radiology, Kumamoto City Hospital, 4-1-60, Higashi-machi, Higashi-ku, Kumamoto City, Kumamoto 862-8505, Japan
- ^e Department of Cardiovascular Medicine, Kumamoto University School of Medicine, 1-1-1 Honjo, Chuo-ku, Kumamoto City, Kumamoto 860-8556, Japan

ARTICLE INFO

Article history: Received 8 November 2021 Accepted 12 November 2021

Keywords:
Malignant hypertension
Optic disc edema
Myocardial edema
Cerebral infarction

ABSTRACT

A 33-year-old woman visited our hospital due to visual loss. Her BP was 280/150 mm Hg and pulse rate was 111 beats per minute. A urinalysis showed protein in urine, suggesting kidney injury. A transthoracic echocardiography showed left ventricular hypertrophy. A Cardiac magnetic resonance imaging suggested left ventricular endocardial edema or inflammation. Ophthalmoscopy showed optic disc edema and hard exudates in both eyes. A brain MRI showed multiple high-intensity areas at the pons and white matter of the cerebrum and cerebellum. Although the patient had malignant hypertension, she was successfully treated by medication.

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Introduction

Malignant hypertension, defined by diastolic blood pressure (BP) above 130 mm Hg, is accompanied by severe retinal le-

sions, and is regarded as the most severe form of hypertension. It also complicates universal multi-organ damage, leading to poor prognosis [1]. We report a case of malignant hypertension with multi-organ injury.

E-mail address: misumi_ikuo@yahoo.co.jp (I. Misumi).

https://doi.org/10.1016/j.radcr.2021.11.034

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Acknowledgments: I would like to thank Mr. Yoshiharu Saito and Ms. Yuko Sakami for recording echocardiographic images.

^{**} Competing Interests: The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

^{*} Corresponding author.

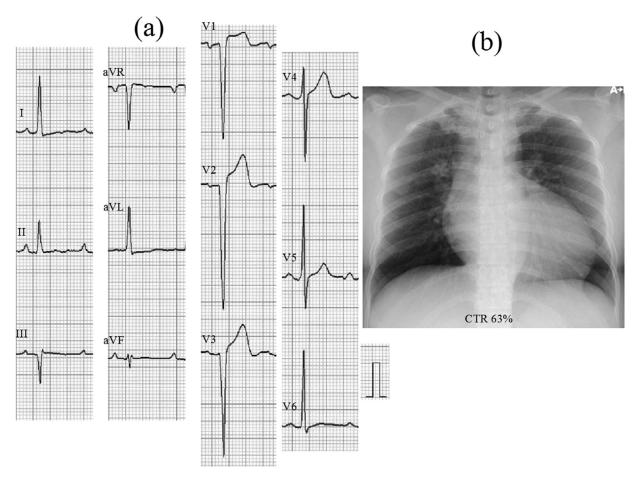


Fig. 1 – A 12-lead electrocardiogram indicated left atrial overload, a QS pattern in leads V1 and V2, and left ventricular hypertrophy (A). A chest radiograph showed a cardiothoracic ratio of 63% with protrusions in the left first and fourth arches (B).

Case report

A 33-year-old woman visited our hospital due to visual loss. She had been diagnosed with hypertension 12 years before but had not visited a hospital thereafter. On physical examination, her BP was 280/150 mm Hg, and her pulse rate was 111 beats per minute. Auscultation revealed a fourth heart sound at the apex. After repetitive intravenous administration of propranolol and nicardipine, she was admitted to our hospital.

Blood sample analysis showed a high white blood cell count (13,800/µL), mild liver injury, and hyperlipidemia. Plasma renin, aldosterone, and cortisol levels were normal. However, plasma catecholamine levels were mildly elevated. Urinary sediment tests showed urine protein 3(+) and occult blood 2(+). A 12-lead electrocardiogram indicated left atrial overload, a QS pattern in leads V1 and V2, and left ventricular hypertrophy (Fig. 1A). A chest radiograph showed a cardiothoracic ratio of 63% with protrusions into the left first and fourth arches (Fig. 1B). Transthoracic apical 4-chamber echocardiography revealed diffuse left ventricular (LV) hypertrophy, as the thickness of the interventricular septum, and LV lateral walls were both 14 mm (Fig. 2). The ejection fraction of the LV was

73%. The global longitudinal strain rate from a 2-dimensional apical 4-chamber view was within normal limits (17.5). There were no signs of significant valvular heart disease from color-flow Doppler echocardiography. Mitral inflow was assessed with pulsed-wave Doppler echocardiography revealing an E wave of 92 cm/s, an A wave of 118 cm/s, and an E wave deceleration time of 152 ms. A tissue Doppler echocardiograph of the septal annuls showed an e' wave of 5.4 cm/s and an E/e' ratio of 17.0, suggesting LV diastolic dysfunction. Myocardial T2 mapping using cardiac magnetic resonance imaging (C-MRI) showed an area of high density from the anterior to the lateral endocardium (Fig. 3). Gadolinium enhancement imaging also showed a late gadolinium enhancement (LGE) in the same region (Fig.4). There was no significant coronary artery stenosis.

Ophthalmoscopy showed optic disc edema and hard exudates in both eyes (Figs. 5A and B). Optic disc edema (Fig. 5, c, d), retinal detachment (Fig. 5D), and cystic changes in the inner retina (Fig. 5D) were confirmed with optical coherence tomography (OCT). A temporal-superior-nasal-inferior-temporal (TSNIT) graph indicated a markedly thickened retinal nerve fiber layer (Fig. 6).

A brain MRI showed multiple high-intensity areas at the pons and white matter of the cerebrum and cerebellum

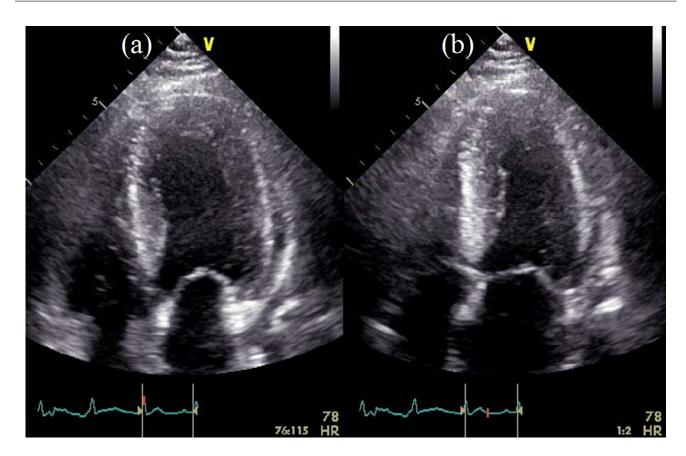


Fig. 2 – A transthoracic apical 4-chamber echocardiography showed diffuse left ventricular (LV) hypertrophy. The thickness of the interventricular septum and LV lateral wall were both 14 mm.

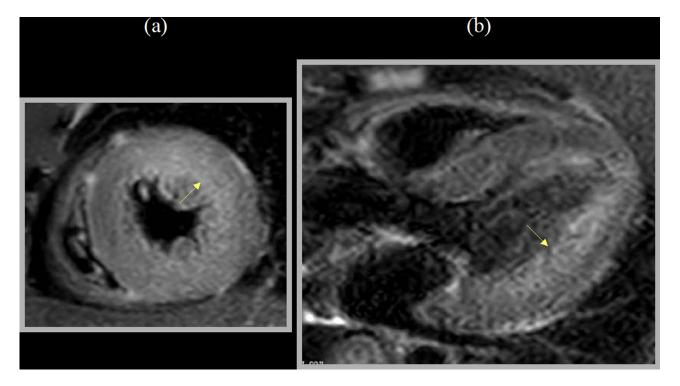


Fig. 3 – Cardiac magnetic resonance imaging (MRI) with T2 mapping revealed that there was a high-density area at the anterior to the lateral endocardium.

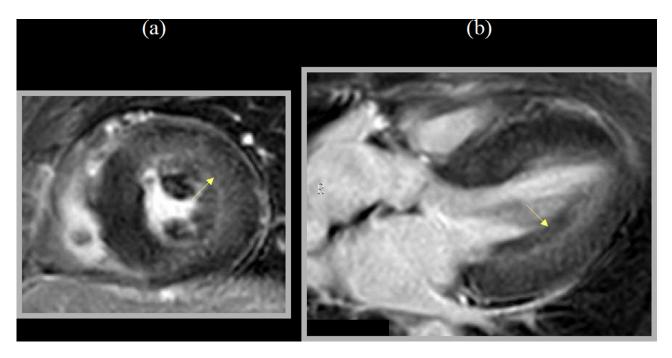


Fig. 4 – Gadolinium enhancement imaging showed a late gadolinium enhancement (LGE) at the anterior to the lateral endocardium, suggesting fibrosis in this region.

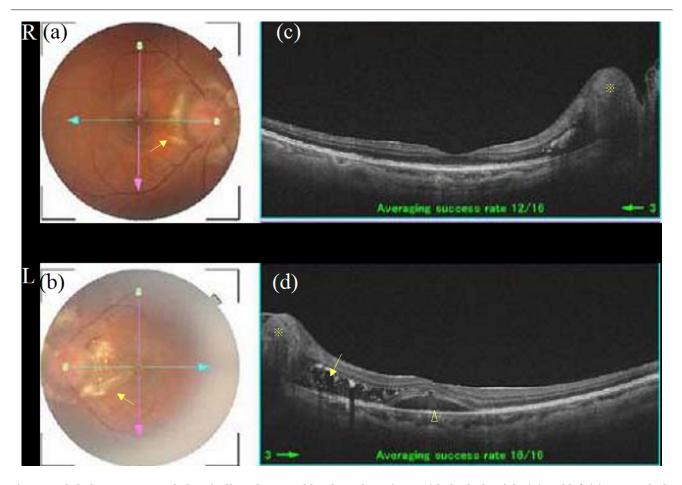


Fig. 5 – Ophthalmoscopy revealed optic disc edema and hard exudates (arrows) in both the right (A) and left (B) eye. Optical coherence tomography showed optic disc edema, retinal detachment involving the fovea, and cystic changes to the inner retina (C and D).

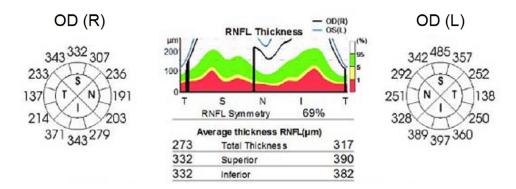


Fig. 6 – Retinal nerve fiber layer (RNFL) thickness was displayed in a temporal-superior-nasal-inferior-temporal (TSNIT) graph. In both eyes, swelling of optic disc RNFL thickness was well above the normal range.

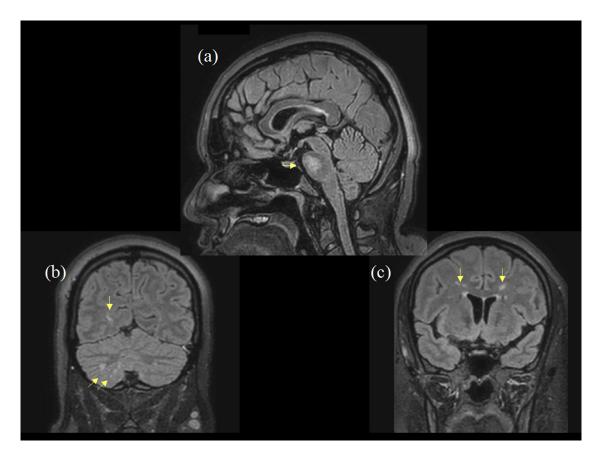


Fig. 7 - A brain MRI showed multiple high-intensity areas at the pons and white matter of the cerebrum and cerebellum.

(Fig. 7). An abdominal MRI, including the adrenal gland, was normal.

The patient was medically treated by Nifedipine 80 mg, Bisoprolol 10 mg, Olmesartan 40 mg, Bizafibrat 400 mg, and Trichomethiazide 2 mg. As her blood pressure still high, Doxazosin 4 mg was added on the sixth day (Fig. 8). The patient complained right weakness, and a follow-up MRI of the brain revealed a new cerebral infarction at the left anterior robe and stenosis at the left anterior communicating artery. The lesions of the white matter and pons had disappeared. After rehabili-

tation, she was discharged from our hospital without obvious complications.

Discussion

Markedly high BP (280/150 mm Hg) and optic disc edema were observed in the present case. Furthermore, multi-organ damage to the brain, heart, and kidneys revealed that the patient

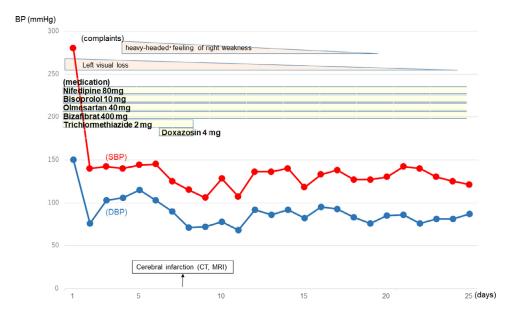


Fig. 8 – Clinical course. The patient was medically treated by Nifedipine 80 mg, Bisoprolol 10 mg, Olmesartan 40 mg, Bizafibrat 400 mg, and Trichomethiazide 2 mg. As the blood pressure was still high, Doxazosin 4 mg was added on the sixth day. The patient complained right weakness, and a follow-up MRI of the brain revealed a new cerebral infarction. After rehabilitation, she was discharged from our hospital without obvious complications.

had malignant hypertension. Malignant hypertension has a prevalence of 1-2 new cases per 100,000 people per year [1].

The patient had LV hypertrophy with impaired diastolic function. However, LV systolic function was normal. The C-MRI demonstrated the impact of malignant hypertension, with T2 mapping, and LGE suggesting myocardial edema, inflammation, or fibrosis of the anterior to lateral endocardium [2].

The vascular changes in the retina were consistent with hypertensive retinopathy. Keith et al. classified hypertensive retinopathy into 4 grades—the present case exhibited the most severe exudates and optic disc edema. Moreover, the patient had marked optic disc edema, retinal detachment, and cystic changes to the inner retina, as well as a thickened retinal nerve fiber layer through the whole circumference. A previous report has described the mechanisms of retinopathy, which involve a breakdown of the blood-retina barrier causing lipid exudation. Furthermore, very severe hypertension may lead to high intracranial pressure, causing swelling of the optic disc [3], and other severe outcomes.

The brain MRI showed that the patient had multiple lesions to the pons and white matter of the cerebrum and cerebellum. White matter lesions are related to the duration of poorly controlled hypertension. A possible explanation of this specific change is that long-standing hypertension results in decreased cerebral blood flow due to the impairment of cerebral autoregulation. Most vulnerable areas are subcortical and include periventricular white matter [4].

Renal function may also be impaired in malignant hypertension. In the present case, only proteinuria, and hematuria were observed.

Without medical treatment, patients with malignant hypertension have a very poor prognosis, with a mortality rate of \sim 80% after 2 years. Developing a more efficient and tol-

erable anti-hypertensive drug therapy has improved patient prognosis. However, cardiovascular complications are still significant. Multivariate analyses have shown a higher risk of malignant hypertension among patients with advanced age, baseline creatinine, and follow-up systolic BP. Indeed, precise BP control at follow-up independently, and significantly improves survival over the long term in patients with malignant hypertension.

In the present case, although cerebral infarction occurred during the course, she discharged our hospital without significant complication.

Conclusion

We experienced a case of malignant hypertension and successfully treated with medication. In a case of malignant hypertension,

Patient consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images.

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