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Ptosis And Paresis of The Nerves III, IV, VI in Patient with Diabetes Mellitus and Hypertension: A Case Report

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ABSTRACT

Ptosis, a condition characterized by drooping of the upper eyelid, can be caused by various factors, including muscle weakness or nerve disorders such as ocular nerve palsy. This case report presents a 54-year-old male patient who experienced ptosis in the right eyelid, along with complaints of blurred vision and double vision. The patient also had a medical history of diabetes mellitus and hypertension, both of which were not well-controlled. Upon examination, the patient had ptosis in the right eye with a vision of 20/400 and normal intraocular pressure. Treatment included methylprednisolone and citicoline, along with other medications, which led to significant improvement after five days. This case highlights the importance of optimal management of diabetes and hypertension to prevent complications such as nerve paresis. The combination of diabetes mellitus, hypertension, and nerve paresis presents a unique clinical challenge, as these conditions can exacerbate one another. Effective management requires a comprehensive and multidisciplinary approach, focusing not only on the immediate symptoms but also on long-term glycemic and blood pressure control to prevent recurrence. This case emphasizes the need for clinicians to consider the interactions between comorbid conditions in the management of ptosis caused by diabetic neuropathy. The conclusions of this study are diabetes mellitus and hypertension caused vascular ischemia which leading to isolated cranial nerve palsy in adults.

KEYWORDS *ptosis; diabetes mellitus; hypertension; oculomotor nerve*

INTRODUCTION

Ptosis is a condition in which there is a decrease or relaxation in a part of the body. *Ptosis* of the upper eyelid (*ptosis of the upper eyelid*, *blepharoptosis*, descending upper eyelid, *drooping eyelid syndrome*) is defined as the position of the upper eyelid margin being abnormally low in primary gaze, causing narrowing of the *palpebra* gap and partially covering the eye. In normal adults, the edge of the upper eyelid is usually positioned 0.5–2 mm below the superior corneal limbus and reaches the highest point slightly in the nasal part of the pupil (the height of the upper eyelid) (Carrie L. Morris & David A Chesnutt, 2025; Shahzad & Siccardi, 2023).

Based on available data, congenital *ptosis* is the most common type, with a higher prevalence in males than females. Among the various forms of congenital *ptosis*, simple congenital *ptosis* is the most dominant form. Meanwhile, in the case of acquired *ptosis*, aponeurotic *ptosis* is the most common type to be found, especially in individuals of advanced adulthood (Keene et al., 2022; Shahzad & Siccardi, 2023). *Ptosis* is classified as congenital or acquired based on the age at the onset of symptoms. *Ptosis* caused by neurogenic factors occurs due to a disorder in the innervation of the levator muscle of the upper eyelid. For example, ocular nerve palsy (third nerve palsy) can cause this condition (Shahzad & Siccardi, 2023).

Ocular nerve palsy is one of the most common cranial neuropathies found in neurological practice. These nerves can be affected along their pathways, from the brainstem

to the orbit. In some cases, there may be isolated involvement of several cranial nerves at the same time. The ocular cranial nerves, namely *oculomotor* (III), *trochlear* (IV), and *abducens* (VI), play an important role in the efferent visual pathway and are responsible for adequate extraocular movement (Dhany et al., 2019; Shree et al., 2022).

The etiology of third cranial nerve palsy can be caused by microvascular ischemia, which includes conditions such as diabetes, hypertension, dyslipidemia, coronary artery disease, and atherosclerosis. This ischemia generally occurs in individuals over the age of ≥50 years, where the prevalence of hypertension, diabetes, coronary artery disease, and dyslipidemia increases significantly, making them an important risk factor. Therefore, the treatment of this condition must focus on managing these risk factors, especially through optimal control of diabetes, hypertension, and dyslipidemia (Shahzad & Siccardi, 2023; Shree et al., 2022).

In this case report, we discuss a male patient who presented with the main complaint of a closed right eyelid, blurred and double eyelids, accompanied by dizziness. Based on the results of clinical examinations and medical history, the patient was diagnosed with *ptosis*, a condition of decreased eyelids due to muscle weakness or nerve disorders. In addition, the patient has a history of comorbidities, including diabetes mellitus and hypertension, which may be predisposing factors or exacerbate the condition.

What makes this case unique is the combination of diabetes mellitus, hypertension, and oculomotor nerve palsy in a single patient. While both diabetes and hypertension are well-known contributors to ocular motor neuropathies, the co-occurrence of these conditions with *ptosis* and the involvement of multiple cranial nerves is a rare phenomenon. This case provides valuable insight into the complex relationship between these conditions, particularly how they can exacerbate one another, especially in older adults. It highlights the importance of understanding the combined effects of these risk factors and contributes to the medical community's knowledge of diabetic and hypertensive neuropathies.

There is a limited number of case reports describing the simultaneous occurrence of diabetes, hypertension, and oculomotor nerve palsy. Most studies tend to focus on either diabetes or hypertension as the primary risk factor for oculomotor nerve palsy, with few exploring the interaction between these two conditions in causing multi-cranial nerve involvement. This gap in the existing literature emphasizes the need for further research to understand how these risk factors converge and how they can be managed effectively in clinical practice.

METHOD

The research method used in this experiment includes analytical methods. It contains the type of method, time, place, and tools of research materials. Picture captions are placed as part of the picture title (*figure caption*), not as part of the picture. The methods used in completing the research are listed in this section.

This study employs a case report methodology, which is a detailed analysis of a single case to explore and understand a particular medical condition and its management. The case was selected based on specific criteria, including the presence of *ptosis* and *oculomotor nerve paresis* in a patient with a history of *diabetes mellitus* and hypertension.

- **Type of Research**: Case Report

- **Research Design**: This is an observational case report, focusing on the medical history, physical examination, diagnosis, and treatment of a 54-year-old male patient.
- **Data Collection Techniques**: Data were gathered through patient history, physical examination, clinical tests, treatment records, as well as follow-up observations to track the patient's progress.
- **Data Analysis Techniques**: The data were analyzed through qualitative methods, emphasizing the assessment of symptoms, clinical findings, treatment responses, and the evaluation of the underlying conditions (*diabetes* and hypertension) that contributed to the patient's condition.

This approach allows for a comprehensive understanding of the interplay between *diabetes*, hypertension, and *oculomotor nerve paresis*, as well as the effectiveness of the treatment regimen.

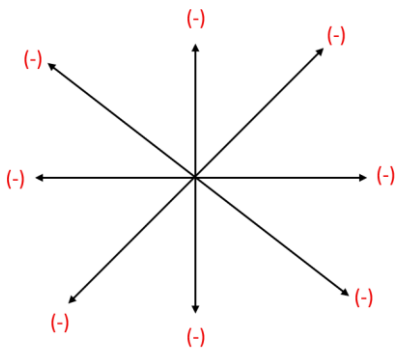
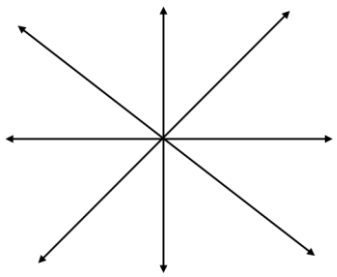
RESULTS AND DISCUSSION

Case

A 54-year-old male patient came to the eye polyclinic with the main complaint of the right eyelid which seemed to close slowly since 2 days ago. This complaint appears progressively, starting from a feeling of heaviness in the right eyelid until finally closing most of the field of view. Patients also complain of blurred vision and double looking objects, especially when looking upwards. Complaints are accompanied by dizziness. In further anamnesis, it was found that the patient had a history of type 2 diabetes mellitus since 10 years ago and hypertension for 7 years, both with suboptimal control.

On physical examination, the patient was found to be aware of composing mentis, pulse 86x/m, breathing frequency 20x/m, axillary temperature 36.8' C and O2 saturation of 97%, with a pain score of 5. The patient's weight is 79 kg.

Table 1. Results of Eye Physical Examination

Examination	OD (Oculus Dextra)	OS (Oculus Sinister)
Visus	20/400	20/50
Intraocular Pressure	11 mmHg	13 mmHg
Eyeball Position	Orthophoria	Orthophoria
Eyeball Movement	Cannot be moved in any direction 	Normal in all directions 
Eyelid	Ptosis	Calm
Conjunctiva Bulbs	Calm	Calm

Cornea	Clear	Clear
Front Eye Room	Inside, cell -, flare -	Inside, cell -, flare -
Iris/Pupil	Bulat, central, RCL +, RCTL +, RAPD -	Bulat, central, RCL +, RCTL +, RAPD -
Lens	Turbid -, shadow test +	Turbid -, shadow test +
Glass Body & Fundoscopy	Strand -, spherical papillae, firm border, pink, CDR dbn, AVR 2:3, neovascularization -, arterial spasm -, retinal bleeding dot -, blot -, exudate -, macula fovea reflex dbn	Strand - spherical papillae, firm border, pink, AVR 2:3, CDR dbn, neovascularization - , arterial spasm - , retinal bleeding dot - , blot -, exudate - , macular reflex fovea dbn
Confrontation Test	Same as checker	Same as checker

Source : Primary Data by Researcher

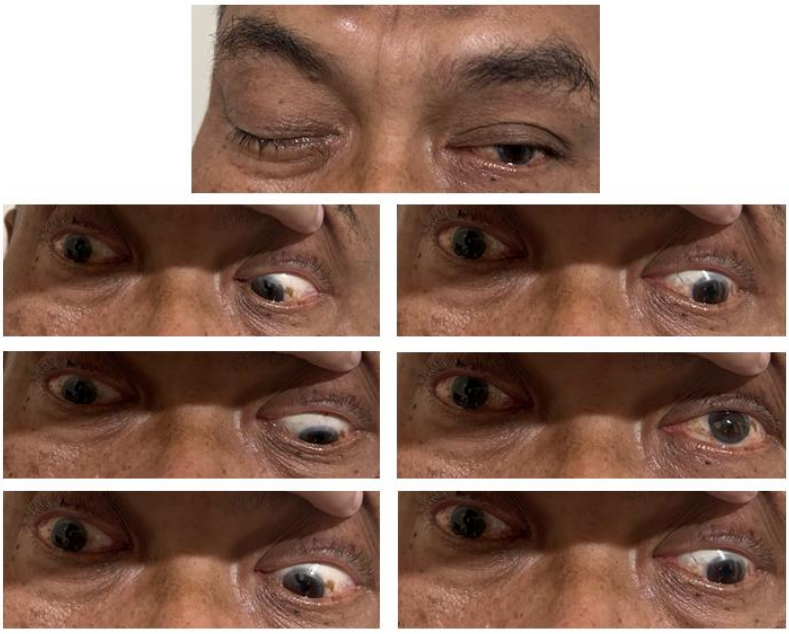


Figure 1. Clinical photo of the patient upon admission to the hospital. It can be seen that the patient's right eye cannot glance.

Source : Data Taken by Researchers

Patients were given methylprednisolone 125 mg injection four times a day for three days, accompanied by citicoline 500 mg injection twice a day. In addition, patients also receive ranitidine injections twice a day and ketorolac injections twice a day. Topical eye therapy is in the form of Protagenta eye drops four times a day in the right eye (OD) and Timol eye drops twice a day in the right eye (OD).

Table 2. Medical Management

Date	Information	Procedure
13/5/25 (Day 1)	The right eyelid closes HR: 89x/m TD: 147/89mmhg RR : 20x/m SpO2 : 99% T: 36.8o C	• Inf RL 12 tpm • Inj. Methylprednisolone 4×125 mg for 3 days • Inj. Citicoline 2×500 mg • Inj. Ranitidine 2×1 • Inj. Ketorolac 2×1 • Protagenta ed 4×1 ODS • Timol ed 2×1 OD • Lab: electrolyte, GDS, uric acid,
14/5/25 (Day II)	The patient said dizziness (+) the right eye closed (+) nausea(-) optimal eating. BAK (+) Vital signs TD: 154/97 HR: 61x/m RR : 20x/m SpO2 : 97% T: 36.8o C GDS 09:00→ 213 mg/dl GDS 13:00 → 279 mg/dl	• Inj RL 12 tpm • Inj. Methylprednisolone 4×125 mg for 3 days • Inj. Citicoline 2×500 mg • Inj. Ranitidine 2×1 • Inj. Ketorolac 2×1 • Protagenta ed 4×1 ODS • Timol ed 2×1 OD Patients are treated with an internal medicine specialist and receive therapy: • Clonidine 1x0.15mg PO • Candesartan 1x8mg PO • Gabapentin 1x300mg PO • Clopidogrel 1x75mg PO • Phenofibrate 1x1 PO • Sliding scale/ 4 jam sansulin rapid 50 unit in 50cc NS
15/5/25 (Day III)	The patient said dizziness (-) The right eye closed (+) the right eye could already blink, the right eyelid felt thick Vital signs TD: 123/76 mmhg HR: 74x/m RR : 20x/m SpO2 : 99% T: 36.5o C	• Inj RL 12 tpm • Inj. Methylprednisolone 4×125 mg for 3 days • Inj. Citicoline 2×500 mg • Inj. Ranitidine 2×1 • Inj. Ketorolac 2×1 • Protagenta ed / 2jam ODS • Bralitex ed/2-jam OD • Lanosan ed 2×1 OD (7 am and 7 pm) Patients are treated with an internal medicine specialist and receive therapy: • Clonidine 1x0.15mg PO • Candesartan 1x16mg PO • Gabapentin 1x300mg PO • Clopidogrel 1x75mg PO • Phenofibrate 1x1 PO

- Sliding scale/ 4 jam sansulin rapid 50 unit in 50cc NS

Source : By Researcher

The patient went home on the fifth day of treatment with a good state of consciousness and vital signs, no complaints of dizziness, the right eye could be slightly opened and the last GDS examination was 267mg/dl. Patients were given drugs to go home in the form of protagenta *eye ointment* 8x1, bralifex *eye drop* 8x1 (OD), Lanosan *eye drop* at 07.00 and 19.00, p.o prednisone 3x30mg Clonidine 1×0.15 mg, Candesartan 1×16 mg, Metformin 3×500 mg, Sansulin 2×22 units, Gabapentin 1×300 mg, CPG 1×75 mg, Fenofibrate 1×1, OMZ 2×1, Prednisolone 3×30 mg, Citicoline 1×1, Proneuronal 1×1, Pamol 3×1.

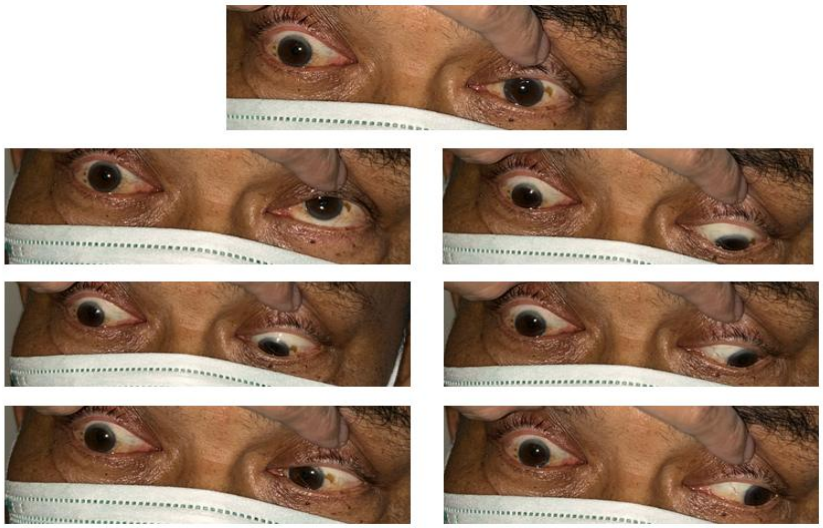


Figure 2. Day I Treatment
Source : Data Taken by Researchers

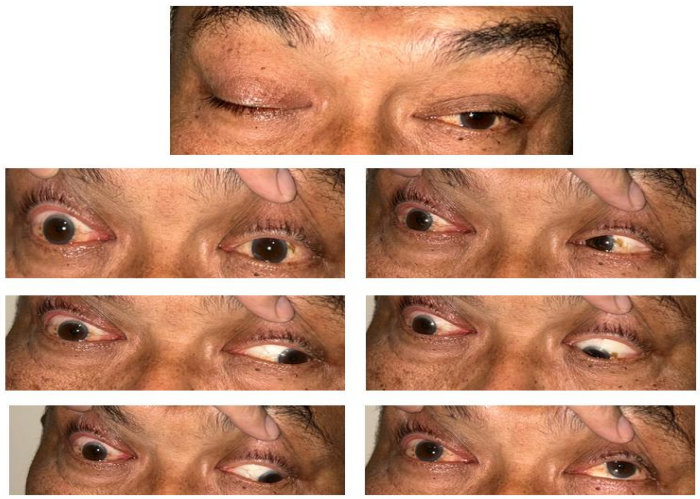


Figure 3. Day II Treatment
Source : Data Taken by Researchers

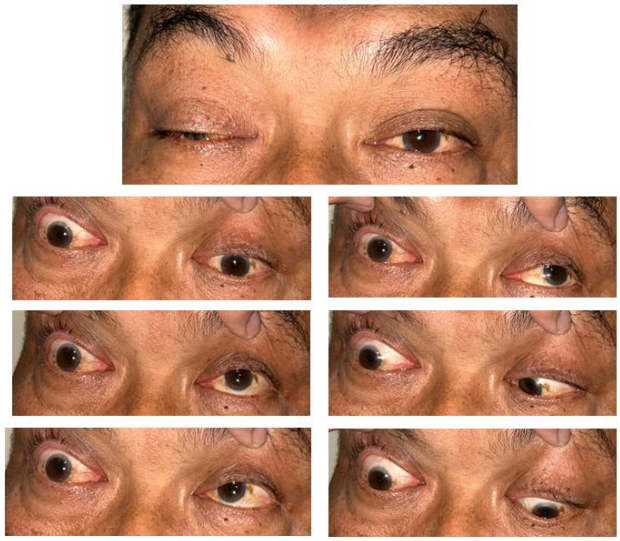


Figure 4. Day III Treatment
Source : Data Taken by Researchers

The patient was checked into the eye polyclinic on May 31, 2025 with improvements. Complaints of eyeball movement have improved, but when the patient sees with two eyes, the patient's vision becomes blurred.

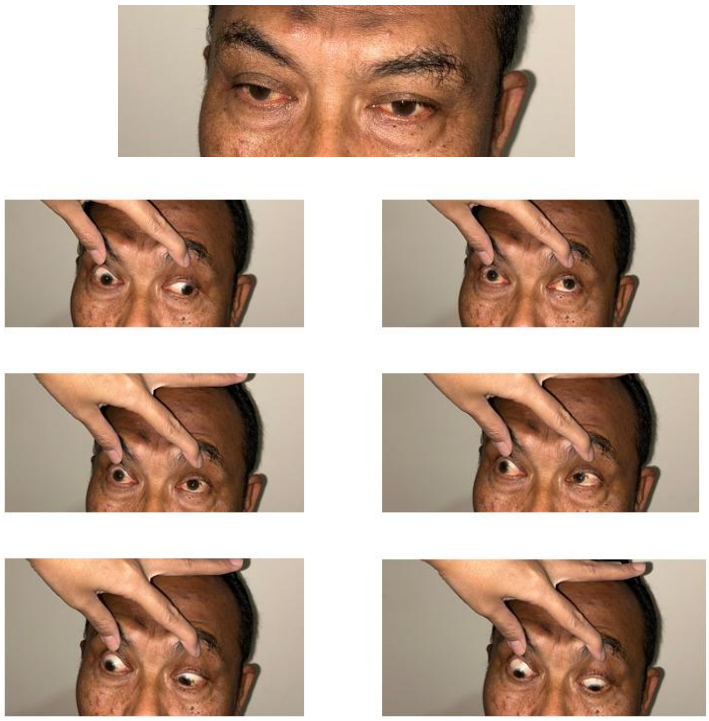


Figure 5. Clinical photo of an outpatient after hospitalization.
Source: Data Taken by Researchers

Deteriorating glycemic status can theoretically lead to total paralysis of the oculomotor nerve, but trigeminal, glossopharyngeal, and vagus nerve involvement is very rare as a manifestation of diabetic neuropathy. Oculomotor paresis in diabetes is thought to be caused by microvascular ischemia that mainly affects the central fibers; Pupillomotor fibers located in

the periphery are generally, but not always, spared from this impact. Pupil involvement is more commonly observed in compressive or infiltrative lesions of the oculomotor nerve, such as aneurysms, neoplasms, or granulomas (Chou et al., 2017 H. J. Kim et al., 2024; Mathew et al., 2019). In this patient, the presence of paresis nervus III, IV, VI was obtained. The patient also had a history of uncontrolled diabetes mellitus.

Diabetes mellitus is one of the most common causes of isolated cranial nerve paralysis in adults, where the third cranial nerve paralysis becomes the second most common diabetic ocular motor neuropathy, after the more common abducens nerve palsy. The pathophysiology of DM involves microvascular ischemia of the nerve vasa, which leads to acute demyelinating injury to the somatic fibers, while superficial parasympathetic pupillomotor fibers are often retained. The oculomotor nerve obtains its vascular supply primarily through the vasa nervorum which originates from the posterior cerebral artery and the superior cerebellar artery. In patients over 50 years of age, isolated third cranial nerve paralysis with pupils remaining responsive is most often caused by microvascular ischemia due to DM or hypertension. Patients with this condition usually show symptoms of sudden onset diplopia and ptosis, while the pupils remain reactive, thus distinguishing this condition from ophthalmoplegia with mydriasis seen in compressive lesions. (Chou et al., 2017; Flindris et al., 2025; Lajmi et al., 2018)

Neuropathy due to diabetes mellitus is mainly caused by microvascular ischemia. However, there are other mechanisms that also contribute, such as the accumulation of sorbitol and fructose due to the hyperactivity of the polyol pathway that causes myoinositol depletion, the activation of the protein kinase Cβ isoform that results in microvascular damage, and the formation of John Roarty (2025) *advanced glycation end products* derived from nonenzymatic glycosylation of neural structural proteins such as laminin, and the accumulation of toxic free radicals. In addition, neurotrophins deficiencies such as Chou et al., (2017) *nerve growth factor*, *ciliary neurotrophic factor*, as well as *insulin-like growth factor* (IGF-1 and IGF-2), along with increased concentrations of tumor necrosis factor-alpha and interleukin, play an important role in the pathogenesis of diabetic neuropathy (Mathew et al., 2019; Shree et al., 2022).

Ptosis and paresis of the oculomotor nerve are not conditions directly related to hypertension. However, there is a significant association between diabetes mellitus and hypertension, especially in the case of the coexistence of the two conditions. The combination of diabetes mellitus and hypertension often exacerbates the clinical complications that occur, given that these two diseases have pathophysiological mechanisms that affect each other. On the other hand, stand-alone hypertension in the absence of diabetes mellitus does not show a direct link with the manifestations of ptosis or paresis of the oculomotor nerve.(Carrie L. Morris & David A Chesnutt, 2025; Jacobs et al., 2018; John Roarty, 2025; Shahzad & Siccardi, 2023; Shree et al., 2022)

Optimization of glycemic control in patients can be achieved through the use of insulin or appropriate oral hypoglycemic agents, which aim to keep blood glucose levels within normal limits. In addition, blood pressure management with the use of antihypertensives is also necessary to maintain blood pressure in a safe range. Clinical observation for 6 to 12 weeks is an important step in assessing the overall improvement of the patient's condition. In the case of ptosis, management depends on the underlying etiology, the severity of ptosis, and the function of the levator muscle. In mechanical ptosis, removal of abnormal structures such as chalazions can be a sufficient solution. However, surgery remains the primary method in the treatment of ptosis, although certain non-surgical options are also available for specific conditions. This combination of medical and surgical approaches demonstrates the importance

of tailoring therapy based on the individual needs of the patient to achieve optimal outcomes. (Mathew et al., 2019; Pauly & Sruthi, 2019; Shahzad & Siccardi, 2023; Shree et al., 2022)

The prognosis in this condition is excellent, with a high chance of full recovery after about three months. During the onset of palsy, supportive measures such as the use of a blindfold in one eye or prismatic glasses can help reduce diplopia. In addition, aggressive control of blood glucose levels and vascular risk factors is highly recommended, as recurrent microvascular cranial neuropathy can occur if the underlying risk factors are not well controlled.(K. Kim et al., 2018; Schroeder et al., 2022)

CONCLUSION

In this case, it was found that there were patients with *diabetes mellitus* and hypertension presenting with *ptosis* of the right eye and paresis of the *nerves III, IV, and VI*. Physical examination of the eyes found ocular *dextra* 20/400; 20/50 *Sinistra* ocular eyes; ocular *palpebra* with extra *ptosis*. Intraocular pressure examination at ocular *dextra* 11 mmHg, ocular *sinistra* 13 mmHg. Digital examination of intraocular pressure of the right and left eyes was within normal limits. The blood sugar test showed 213 mg/dL, with high blood pressure of 147/90 mmHg. After treatment for 5 days, there was an improvement in the condition of the patient's *oculomotor nerve paresis*. Treatment was continued with outpatient care, accompanied by observation of vital signs. Optimization of glycemic control in patients can be achieved through the use of insulin or appropriate oral hypoglycemic agents, which aim to keep blood glucose levels within normal limits. In addition, blood pressure management with the use of antihypertensives is also necessary to maintain blood pressure in a safe range. Clinical observation for 6 to 12 weeks is an important step in assessing the overall improvement of the patient's condition. In the case of *ptosis*, management depends on the underlying etiology, the severity of *ptosis*, and the function of the levator muscle.

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