

Google Chrome isn't your default browser [Set as default](#)

Gmail international journal of case reports and medical reviews Active

Submission Confirmation External Inbox x

International Journal of Medical Reviews and Case Reports <ijmrcr@ejmanager.com> to me Jan 9, 2020, 12:55 PM

Dear Lothar Matheus Manson Vanende Silalahi,

Your submission entitled **Rare Case of Simultaneous Diabetic Oculomotor and Trochlear Nerve Neuropathy: Case Report** (Manuscript Number: IJMRCR-2020-01-06) has been received by **International Journal of Medical Reviews and Case Reports**.

You could follow status of your manuscript by login to your author account at www.ejmanager.com.

Thank you for submitting your work to our journal.

Best regards,

Editor
International Journal of Medical Reviews and Case Reports
<http://mdpub.net>

Windows taskbar: 99+ 32°C Sebagian cerah 11:38 27/09/2024 20

**Rare Case of Simultaneous Diabetic Oculomotor and Trochlear Nerve Neuropathy:
Case Report**

Lothar Matheus Manson Vanenede Silalahi, MD, Neurologist
Medical Faculty, Duta Wacana Christian University, Yogyakarta, Indonesia
Siloam Hospitals Yogyakarta, Indonesia

ABSTRACT

INTRODUCTION. Diabetic cranial neuropathy is one spectrum of diabetic neuropathy. Isolated cranial nerve involvement is common as the presentation of diabetic cranial neuropathy. The most frequently involved are abducens and oculomotor nerve with trochlear nerve the least involved. Simultaneous cranial nerve involvement is rare and need further investigation for other possible etiology.

CASE REPORT. We reported a 52-year-old, diabetic and hypertensive male, presented with acute onset, first ever, severe temporal headache with left eye ptosis, diplopia and ophthalmoplegia. Neurologic examination revealed left oculomotor and trochlear nerve palsy with sparing of pupillary function. Examination of blood D-dimer and C-reactive normal. Head contrast MRI and MR Angiography revealed no lesion found. Final diagnosis is diabetic cranial neuropathy supported with improvement of the ptosis 5 days after onset

CONCLUSION. Simultaneous involvement of two or more cranial nerve neuropathy in diabetic patient is uncommon and need further investigation to exclude the other possibilities. Exclusion of the other possible diagnosis is the mainstay to diagnosis diabetic cranial neuropathy.

Keyword: diabetes, neuropathy, cranial nerve

Corresponding author:

Lothar Matheus Manson Vanende Silalahi, MD, Neurologist
Jl. Melati no 24A, RT 03 RW 52, Poh Ruboh, Condong Catur, Depok, Sleman
Yogyakarta, Indonesia
Postal Code: 55283
Phone: +6281288173000
Email: lothar@staff.ukdw.ac.id

INTRODUCTION

Diabetes and neuropathy has been recognized and the different subtypes of diabetic neuropathy existed. Many types of nerve can be affected and one of which is cranial nerve[1]. Diabetic cranial neuropathy bring forward less interest than diabetic retinopathy so that few literature review the clinical features that may present in diabetic cranial neuropathy[2]. Many literature found that Isolated cranial nerve palsy is the most common clinical presentation in diabetic cranial neuropathy, with the common cranial nerve affected is abducen and oculomotor nerve, the least affected is trochlear nerve[1],[3]. Simultaneous cranial nerve palsies in diabetic patient need further investigation to exclude the other possible etiology before the establishment diagnosis of diabetic cranial neuropathy. In this article we presented rare case of simultaneous involvement of oculomotor and trochlear nerve involvement of diabetic cranial neuropathy.

CASE REPORT

A 52-year-old Indonesian Male presented to emergency department with 2-days onset of moderate-severe left temporal headache and left eye ptosis. The patient felt the progression of the ptosis and denied diurnal pattern of ptosis. He also felt nausea and vomit occurred with his headache. He reported double vision while seeing object with his two eyes (binocular diplopia). The double vision improved while looking with one eye either the right eye or the ptosis side. That was the first headache he felt.

The patient denied any head trauma, prodromal fever, seizure, decreased of consciousness, unilateral face and body paresthesia or weakness, perioral numbness, face asymmetric, dysarthria, dysphagia and cognitive impairment. He had a history of uncontrolled hypertension and diabetes mellitus.

On physical examination the patient in good condition and fully conscious. His body weight 69 kg, height 169 cm, body mass index 24,5 (overweight). His blood pressure 150/90 mmHg, VAS Score 8 with normal body temperature, respiration and heart rate. On neurologic examination we found ptosis on left eye, impaired adduction, elevation, depression of the left eye. Movement of the right eye normal to all directions. We found both pupil isocor, size 4 mm in both eyes, with normal direct and indirect light reflex. The other cranial nerves were normal. There was no vessel swelling and pain on palpation on the left temporal. Motor and sensory examination on extremities were normal. No pathological reflex and clonus were found. Cognitive screening were normal. No sign of meningeal irritation found. We conclude that the patient had a left third and fourth cranial nerve palsy with sparing of pupillary function.

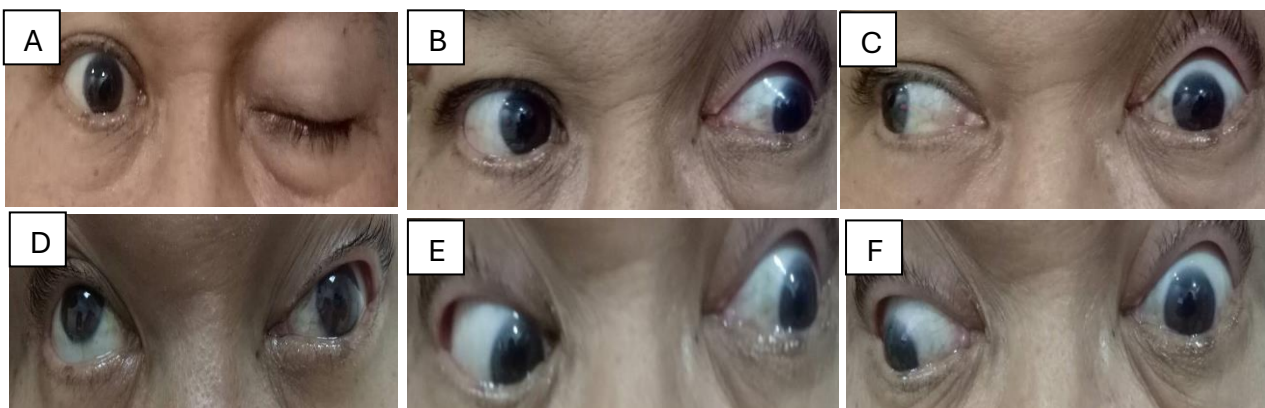


Figure 1. A.Ptosis on the left eye on normal eye position B. Normal left horizontal gaze both eyes C. On horizontal right gaze, the left eye kept in mid position D. On upper vertical gaze, the left eye keep in mid position E. Normal left inferior gaze of both eyes F. On right inferior gaze, the left eye keep in mid position

Because of the acute onset of the symptoms, first ever headache and stroke risk factor we still consider subarachnoid haemorrhage with vascular aneurysm. The other differential diagnosis is sinus cavernous lesion (sinus cavernous thrombosis) although the trigeminal nerve, abducen nerve and pupillary reflex are normal. We also think the possibility of temporal arteritis and the last differential diagnosis is cranial diabetic neuropathy. To confirm the diagnosis we sent the patient to have a contrast head MRI with MR angiography. To confirm the thrombosis and temporal arteritis we measure blood D-dimer and C-reactive protein. We also measure random and fasting glucose profile, HbA1c, lipid profile and renal function because of history of diabetes mellitus and hypertension.

Contrast head MRI examination revealed no lesion on sinus cavernous. Neither blood nor ischemic lesion found on brain parenchyma and subarachnoid space. On MR angiography brain artery and vein were completely normal. Neither aneurysm nor stenosis found on carotid and intracranial vessels. Quantitative measurement of D-dimer and C-reactive protein were normal. We found normal fast blood glucose (93 mg/dl) but high result of 2-hours post prandial blood glucose (247 mg/dL) and HbA1c (10,5 %). High result of HbA1c reflects the poor control of diabetes mellitus. High blood ureum (39,8 mg/dL) and creatinine (1,42 mg/dL) and low estimated glomerular filtration rate (56 mL/min) maybe the result of uncontrolled blood glucose. From all the results we diagnosed patient with diabetic cranial neuropathy.

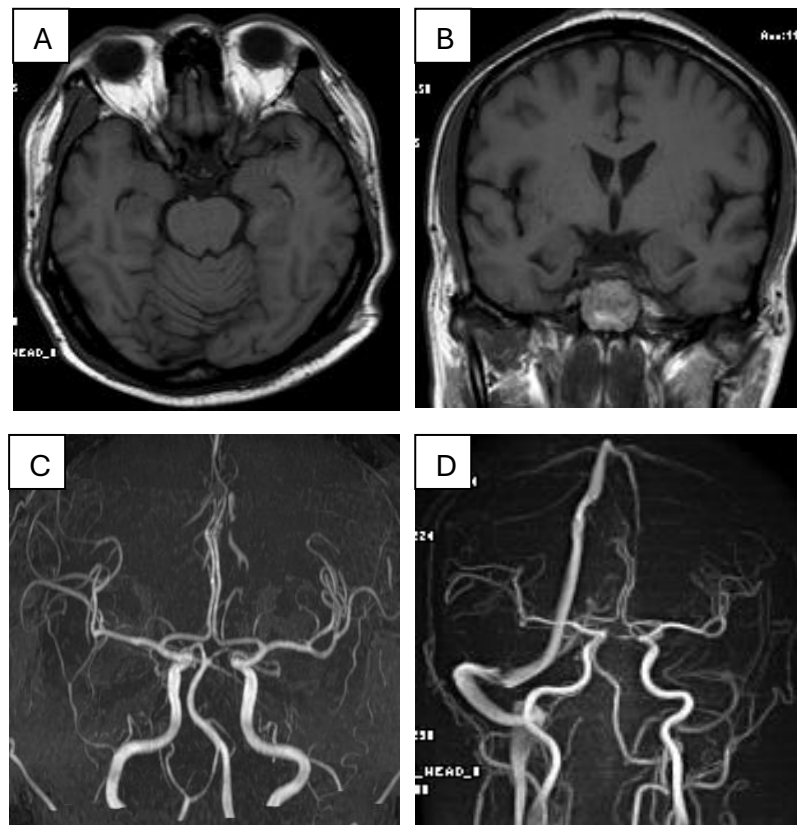


Figure 2. A and B. Axial and coronal section of head MRI showing no lesion in cavernous sinus. C and D. Angiography of carotid, intracranial artery and venous system showing no aneurysm and stenosis. Note the right dominance of transverse sinus.

We treated the patient with high dose mecobalamine to promote the regeneration of the neuropathy. We treated the patient with tramadol and non-steroid anti-inflammatory for the headache. To control the blood glucose we treated patient with acarbose and combination of amlodipine and candesartan for the hypertension. On the fifth days we observed the improvement of the left eye ptosis and that finding make the diagnosis of diabetic cranial neuropathy more certain. The patient then discharge from hospital and we educate the patient

to control his blood glucose for the optimal treatment for the ptosis and left eye movement dysfunction.

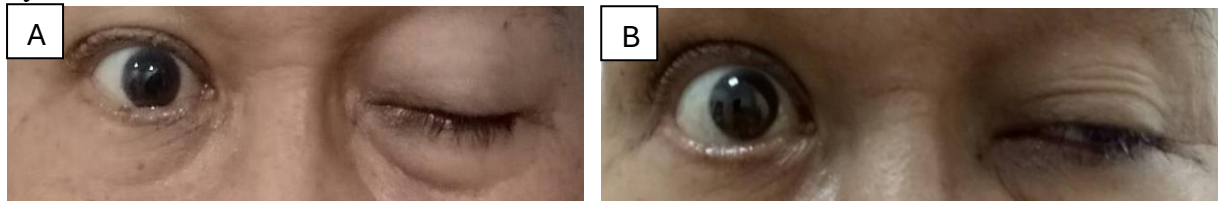


Figure 3. A. Ptosis on the first day admission. B. Ptosis on the fifth day of care. Note the left upperlid improvement on the fifth day.

DISCUSSION

We summarize that the patient presented to the emergency department with unilateral painful ophthalmoplegia. Syndrome of painful ophthalmoplegia involves diverse causes and need comprehensive evaluation. The diagnosis of aneurysms subarachnoid haemorrhage must be considered based in the acute on onset headache with first ever severe headache[4]. The other differential diagnosis that must be excluded are sinus cavernous thrombosis, Tolosa-Hunt Syndrome, ophthalmoplegic migraine and diabetic cranial neuropathy[5]. Temporal location and throbbing nature of the headache make us consider temporal arteritis as the other differential diagnosis[6]. Ophthalmoplegic migraine can be excluded because of the acute onset and didn't meet the criteria for migraine[7].

To confirm the diagnosis we conducted contrast head MRI with MR angiography to the patient. MRI can be used to detect acute and subacute subarachnoid specifically the gradient echo T2[8]. Vascular aneurysm, granulomatous tissue in Tolosa-Hunt Syndrome and sinus cavernous thrombosis may be detected with MRI and MRA examination[5]. If in brain and vascular imaging detected no abnormality the diagnosis will be diabetic cranial neuropathy[4]. We also measure blood D-dimer and C-reactive protein to aid the diagnosis of thrombosis and temporal arteritis respectively[9],[10].

The MRI and MRA result revealed no lesion in cavernous sinus. On MR angiography we found no aneurysm especially on superior cerebellar artery and posterior communicating artery. Blood D-dimer and C-reactive protein also normal. Exclusion of the other possible differential diagnosis, we conclude that the etiology of patient's symptoms is diabetic cranial neuropathy. Diagnosis of diabetic cranial neuropathy supported with the result of abnormally high HbA1c that reflected the poor glycemic control. One study found that 70% patient with diabetic cranial neuropathy had poorly controlled diabetes[2].

Cranial neuropathy is one of the spectrum in diabetic neuropathy[10]. Oculomotor nerve palsy tend to be seen in patient over the 50 years as be seen in the patient[11]. The common nerve that affected are abducen nerve (50%), oculomotor nerve (43,3%) and trochlear nerve (6,7%)[1]. Isolated palsy of the nerve is a well known manifestation of diabetic cranial neuropathy[3]. Our patient presented with combined oculomotor and trochlear nerve palsy which is very uncommon. The trochlear involvement of diabetic cranial neuropathy is uncommon and combination of oculomotor and trochlear nerve make this case very uncommon.

We find the other report that presented the patient with simultaneous oculomotor and trochlear nerve lesion. That reports found the ischemic lesion in midbrain as the etiology of the lesion[3]. The clinical difference that the patient in the report presented with painless ophthalmoplegia while our patient suffer the painful ophthalmoplegia. Meanwhile if the ischemic lesion is still considered in our patient, the MRI result didn't find any ischemic lesion in the midbrain.

The other key features of diabetic cranial neuropathy, especially with oculomotor involvement is pupillary sparring pattern[12]. Basic mechanism of diabetic cranial neuropathy is neuronal ischemic change. The anatomical arrangement of oculomotor nerve is periphery located fibres nerve controlling pupillary reflex and centrally located fibres controlling the somatic motor movement[13]. The ischemic change nature of the disease and centrally located vasculature in muscle is the reason of sparring of autonomic pupil function in diabetic oculomotor neuropathy[1].

Severe acute pain also a typical presentation of diabetic cranial neuropathy[1]. The mechanism of painful diabetic neuropathy is not fully understood and also in diabetic cranial neuropathy. Hypothetically, chronic hyperglycemia promote damage to the nerve and cause regeneration of nerve sprouts called neuromas. This neuromas expands and hyperexcitable, generates pain impulse[14].

Prognosis of diabetic neuropathy varies, some of them progressive and the other as monophasic illness[1]. Diabetic cranial neuropathy is a monophasic illness. No specific requirements is required other than prismatic help of the diplopia as the ptosis improve[11]. Diabetic cranial neuropathy regresses spontaneously after three month in average. After an acute episode, 70 % of patients will cure within 6 months[2]. We observed an improvement of the left eye ptosis on the fifth day after onset without specific treatment. This clinical improvement supports the diagnosis of diabetic cranial neuropathy. Spontaneous resolution nature of the disease make the clinical monitoring and equilibration of diabetes is the core of management[2]. Strict glucose control from the time of diagnosis of DM is the most important aspect of the treatment[11].

CONCLUSION

Diabetic cranial neuropathy is one spectrum of diabetic neuropathy. The common clinical presentation is acute painful severe headache with ophthalmoplegia. The key features if there is involvement of oculomotor nerve is sparring of pupillary function. Simultaneous involvement of two or more cranial nerve in diabetic cranial nerve patient is uncommon and need further investigation to exclude the other possibilities. Exclusion of the other possible diagnosis is the mainstay to diagnosis diabetic cranial neuropathy. Diabetic cranial neuropathy is a monophasic illness, recover spontaneously and need strict control of the diabetes as the main management.

REFERENCES

- [1] J. Tracy and J. Dyck, "The Spectrum of Diabetic Neuropathies," vol. 19, no. 1, pp. 1–26, 2009.
- [2] H. Lajmi *et al.*, "Oculomotor palsy in diabetics," *J. Gynecol. Obstet. Biol. la Reprod.*, 2017.
- [3] N. P. I. Heliopoulos and H. P. E. Maltezos, "Simultaneous , painless , homolateral oculomotor and trochlear nerve palsies in a patient with type 2 diabetes mellitus . Neuropathy or brainstem infarction ?," pp. 19–21, 2006.
- [4] T. Shiode, S. Oya, and T. Matsui, "A Case of the Internal Carotid Artery – Posterior Communicating Artery Aneurysm Mimicking Tolosa – Hunt Syndrome," pp. 1–3, 2015.
- [5] C. Hung *et al.*, "Painful ophthalmoplegia with normal cranial imaging," 2014.
- [6] T. Author and B. Society, "Ophthalmoplegia in an Elderly Woman with Giant Cell Arteritis Running Title: Ophthalmoplegia in an elderly woman Author," no. June, 2018.
- [7] Kelompok Studi Nyeri Kepala Perhimpunan Dokter Spesialis Saraf Indonesia, *Konsensus Nasional V Diagnosis dan Penatalaksanaan Nyeri Kepala*. 2018.
- [8] P. Mitchell *et al.*, "Detection of subarachnoid haemorrhage with magnetic resonance imaging," pp. 205–211, 2001.

- [9] J. Y. Al-hashel, "The Value of D-dimer Test for Diagnosis of Cerebral Venous Thrombosis in Emergency Medicine : Open Access," vol. 5, no. 4, pp. 4–6, 2015.
- [10] T. A. Kermani *et al.*, "Utility of Erythrocyte Sedimentation Rate and C-Reactive Protein for the Diagnosis of Giant Cell Arteritis Tanaz," vol. 41, no. 6, pp. 866–871, 2013.
- [11] J. G. Llewelyn, "The Diabetic Neuropathies: Types, Diagnosis and Management," 2003.
- [12] P. E. Venkatesan, G. Gnanashanmugam, N. Parimalam, and M. B. Pranesh, "Diabetes plus third nerve palsy not always diabetic third nerve palsy," no. 1, pp. 1–2, 2019.
- [13] P. Chou, K. Wu, and P. Huang, "Ptosis as the only manifestation of diabetic superior division oculomotor nerve palsy," vol. 46, no. October, 2017.
- [14] A. Aslam, J. Singh, and S. Rajbhandari, "Pathogenesis of Painful Diabetic Neuropathy," vol. 2014, 2014.

Gmail international journal of case reports and medical reviews Active

Article Revision Letter for Authors - (IJMRCR-2020-01-06) Inbox

International Journal of Medical Reviews and Case Reports <ijmrcr@ejmanager.com> Thu, Jan 16, 2020, 3:27 AM

Dear Lothar Matheus Manson Vanende Silalahi,

Your manuscript entitled "Rare **Case** of Simultaneous Diabetic Oculomotor and Trochlear Nerve Neuropathy: **Case** Report

" (Ms.Nr. IJMRCR-2020-01-06) was reviewed by editorial board members of the **International Journal of Medical Reviews and Case Reports**. As initial decision, your manuscript was found interesting but some revisions have to be made before it can reach a publishable value.

Please answer all the comments below point-by-point in an accompanying response letter to your revised submission.

You should send your revised manuscript via the online system of eJManager on <http://my.ejmanager.com>.

Sincerely yours,

Ivan Inkov, Dr.
Editor
International Journal of Medical Reviews and Case Reports
inkov@journalmedica.com
<http://mv.ejmanager.com/ijmrcr>

82 Mail | 17 of 20

Ivan Inkov, Dr.
Editor
International Journal of Medical Reviews and Case Reports
inkov@journalmedica.com
<http://my.ejmanager.com/ijmrcr>
<http://www.mdpub.net>

COMMENTS for Authors:

=> Reviewer # 1

dear author
Thank you for your study
- there were many english issues (many spelling and grammar mistakes)
-the case report: He reported double vision while seeing object with his two eyes (binocular diplopia). The double vision improved while looking with one eye either the right eye or the ptosis side\ means simply binocular diplopia
-history of uncontrolled hypertension and diabetes mellitus must be mentionned in the beginning of the case report
five days of evolution are insufficient
-discussion : you repeated many informations mentionned in the case report
-references must be revised, reference 2 is false

**Rare Case of Simultaneous Diabetic Oculomotor and Trochlear Nerve Neuropathy:
Case Report**

Lothar Matheus Manson Vanenede Silalahi, MD, Neurologist
Medical Faculty, Duta Wacana Christian University, Yogyakarta, Indonesia
Siloam Hospitals Yogyakarta, Indonesia

ABSTRACT

INTRODUCTION. Diabetic cranial neuropathy is a spectrum of diabetic neuropathy. Isolated cranial nerve involvement is common as the manifestation of diabetic cranial neuropathy. The cranial nerve that frequently involved are abducens and oculomotor nerve, trochlear nerve is the least involved. Simultaneous cranial nerve involvement is not common and need further investigation for other possible etiology.

CASE REPORT. We reported a 52-year-old, diabetic and hypertensive male, presented with acute onset, first ever, severe temporal headache with left eye ptosis, diplopia and ophthalmoplegia. Neurologic examination revealed left oculomotor and trochlear nerve palsy with sparing of pupillary function. Examination of blood D-dimer and C-reactive were normal. Head contrast MRI and MR Angiography revealed no lesion. He was diagnosed with diabetic cranial neuropathy after exclusion of other possible etiology.

CONCLUSION. Simultaneous involvement of two or more cranial nerve neuropathy in diabetic patient is not common and need further investigation. Exclusion of the other possible diagnosis is important to diagnose diabetic cranial neuropathy.

Keyword: diabetes, neuropathy, cranial nerve

Corresponding author:

Lothar Matheus Manson Vanende Silalahi, MD, Neurologist
Jl. Melati no 24A, RT 03 RW 52, Poh Ruboh, Condong Catur, Depok, Sleman
Yogyakarta, Indonesia
Postal Code: 55283
Phone: +6281288173000
Email: lothar@staff.ukdw.ac.id

INTRODUCTION

Relationship between diabetes and neuropathy had been recognized and the different sub types of diabetic neuropathy existed. Many types of nerve can be affected and one of which is cranial nerve[1]. Diabetic cranial neuropathy bring forward less interest than diabetic retinopathy so that not much literature review the clinical features that may present in diabetic cranial neuropathy[2]. Isolated cranial nerve palsy is the most common clinical presentation in diabetic cranial neuropathy, with the common cranial nerve affected is abducens and oculomotor nerve, the least affected is trochlear nerve[1,3]. Simultaneous cranial nerve palsies in diabetic patient need further investigation to exclude the other possible etiology before the establishment diagnosis of diabetic cranial neuropathy. In this article we presented rare case of simultaneous involvement of oculomotor and trochlear nerve involvement of diabetic cranial neuropathy.

CASE REPORT

A 52-year-old Indonesian Male, with history of uncontrolled hypertension and diabetes mellitus, presented to emergency department with 2-days onset of moderate-severe left temporal headache and left eye ptosis. The patient felt the progression of the ptosis and denied diurnal pattern of ptosis. He also came with binocular diplopia, nausea and vomit occurred with his headache. That was the first headache he felt.

The patient denied any head trauma, prodromal fever, seizure, decreased of consciousness, unilateral face and body paresthesia or weakness, perioral numbness, face asymmetric, dysarthria, dysphagia and cognitive impairment.

On physical examination, he was fully conscious. His body weight 69 kg, height 169 cm, body mass index 24,5 (overweight). Vital sign measurement revealed hypertension and 8 point for Visual Analog Scale. On neurologic examination, he had ptosis on left eye, impaired adduction, elevation, depression of the left eye. Movement of the right eye was normal to all directions. Both pupils were isochoric, 4 mm-size on both eyes, and normal direct and indirect light reflex. There was no vessel swelling and pain on palpation on the left temple. Motor and sensory examination on extremities were normal. No pathological reflex and clonus were found. Cognitive screening was normal. No sign of meningeal irritation. In conclusion, the patient had left oculomotor and abducens cranial nerve palsies with sparing of pupillary function.

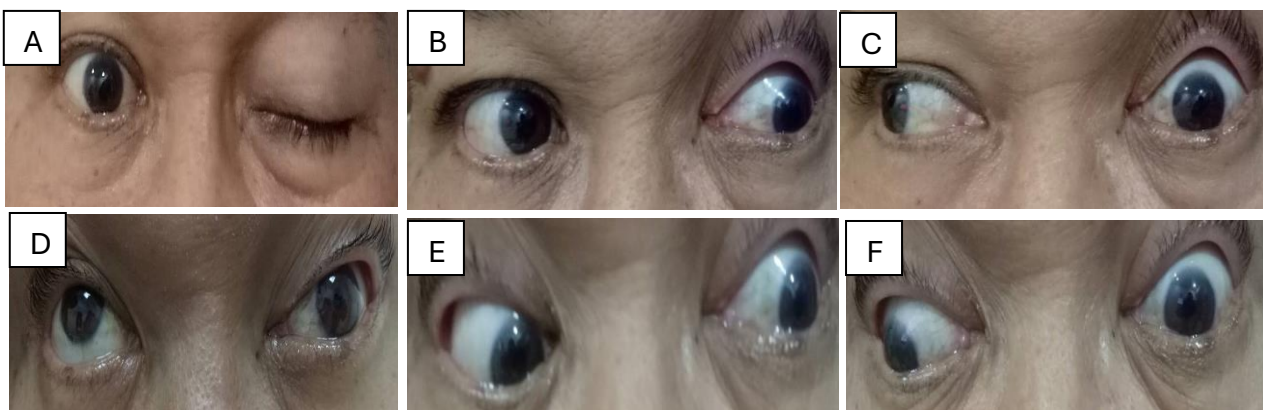


Figure 1. A.Ptosis on the left eye on normal eye position B. Normal left horizontal gaze on both eyes C. On right horizontal gaze, the left eye kept in mid position D. On upper vertical gaze, the left eye keep in mid position E. Normal left inferior gaze of both eyes F. On right inferior gaze, the left eye keep in mid position

There was a suspicion of subarachnoid haemorrhage with vascular aneurysm because of the acute onset of the symptoms, first ever headache and stroke risk factor. The differential diagnosis were sinus cavernous lesion (although the trigeminal nerve, abducens nerve and

pupillary reflex were normal), left temporal arteritis and cranial diabetic neuropathy. To confirm the diagnosis, the patient underwent a contrast head MRI with MR angiography. Blood D-dimer and C-reactive protein were measured to confirm the possibility of thrombosis and arteritis. Random and fasting blood glucose, HbA1c, lipid profile and renal function were also measured.

Contrast head MRI examination revealed no lesion on sinus cavernous. Neither blood nor ischemic lesion found on brain parenchyma and subarachnoid space. On brain MR angiography, artery and vein were all normal. Neither aneurysm nor stenosis found on carotid and intracranial vessels. Quantitative measurement of D-dimer and C-reactive protein were normal. We found normal fasting blood glucose (93 mg/dL) but high result of 2-hours post prandial blood glucose (247 mg/dL) and HbA1c (10,5 %). High result of HbA1c reflected the poor control of diabetes mellitus. From all the examinations, the diagnosis of diabetic cranial neuropathy was made.

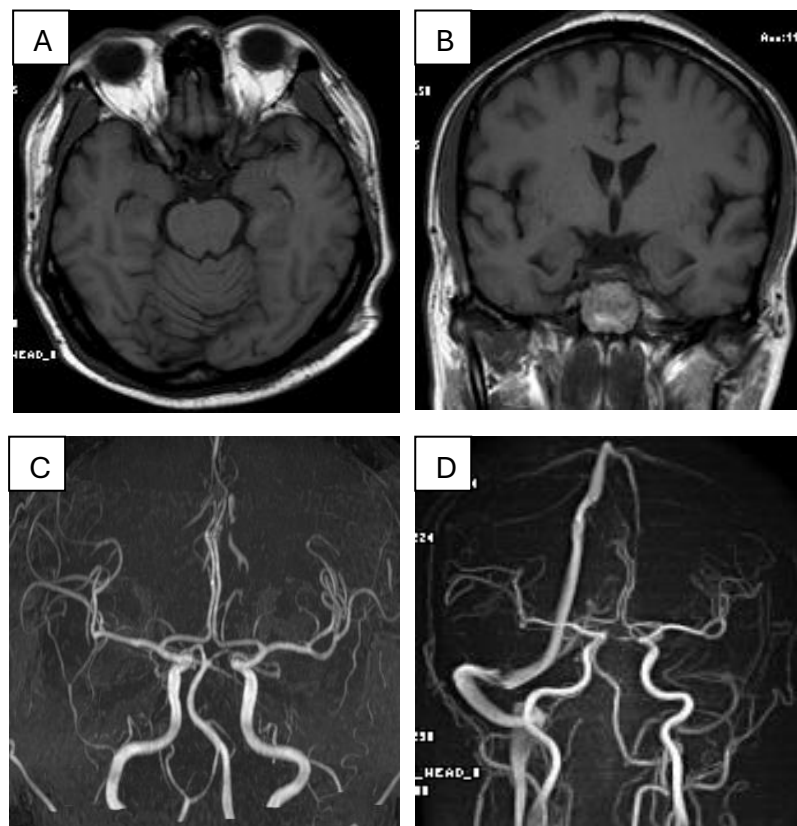


Figure 2. A and B. Axial and coronal section of head MRI showed no lesion in cavernous sinus. C and D. Angiography of carotid, intracranial artery and venous system showed no aneurysm and stenosis. Note the right dominance of transverse sinus.

The patient was treated with neurotrophic for the neuropathy and weak opioid combined with non-steroid anti-inflammatory for the headache. The patient then discharged from hospital and education to control his blood glucose for the optimal treatment were given.

DISCUSSION

We summarized the patient presented to the emergency department with unilateral painful ophthalmoplegia. Syndrome of painful ophthalmoplegia involves diverse causes and need comprehensive evaluation. The diagnosis of aneurysmal subarachnoid haemorrhage must be considered[4]. The other differential diagnosis that must be excluded were sinus cavernous thrombosis, Tolosa-Hunt Syndrome, migraine with ophthalmoplegia, temporal arteritis and diabetic cranial neuropathy[5-6].

Migraine with ophthalmoplegia can be excluded because of the acute onset and didn't meet the criteria for migraine[7]. MRI can be used to detect acute and subacute subarachnoid specifically the gradient echo T2[8]. Vascular aneurysm, granulomatous tissue in Tolosa-Hunt Syndrome and sinus cavernous thrombosis may be detected with MRI and MRA examination[5]. The diagnosis of diabetic cranial neuropathy will be made if the findings on head MRI, MRA, blood D-dimer and C-reactive protein were normal [4, 9-10]. The diagnosis of diabetic cranial neuropathy in this patient was supported with the result of abnormally high HbA1c. One study found that 70% patient with diabetic cranial neuropathy had poorly controlled diabetes[2].

Cranial neuropathy is one of the spectrum in diabetic neuropathy[10]. The common nerve affected were abducens nerve (50%), oculomotor nerve (43,3%) and trochlear nerve (6,7%)[1]. Oculomotor nerve palsy tend to be seen in patient over the 50 years in diabetic neuropathy [11]. Isolated palsy of the nerve is a well-known manifestation of diabetic cranial neuropathy[3]. Our patient presented with combined oculomotor and trochlear nerve palsy which is very uncommon. The trochlear involvement of diabetic cranial neuropathy was uncommon and combination of oculomotor and trochlear nerve make this case more uncommon.

We found the other report that presented the patient with simultaneous oculomotor and trochlear nerve lesion. That reports found the ischemic lesion in midbrain as the etiology of the lesion[3]. The clinical difference was the painless ophthalmoplegia while our patient presented with painful ophthalmoplegia. Meanwhile, if the ischemic lesion is still considered in our patient, the MRI result didn't find any ischemic lesion in the midbrain.

The other key features of diabetic cranial neuropathy, especially with oculomotor involvement is pupillary sparing pattern[12]. Basic mechanism of diabetic cranial neuropathy is neuronal ischemic change. The anatomical arrangement of oculomotor nerve is periphery-located fibres nerve controlling pupillary reflex and centrally-located fibers controlling the somatic motor movement[13]. The ischemic change nature of the disease and centrally located vasculature in muscle are the reason behind the sparing of autonomic pupil function in diabetic oculomotor neuropathy[1].

Severe acute pain also a typical presentation of diabetic cranial neuropathy[1]. The mechanism of painful diabetic neuropathy is not fully understood. Hypothetically, chronic hyperglycemia promote damage to the nerve and cause regeneration of nerve sprouts called neuromas. These neuromas expand, hyperexcitable and generates pain impulse[14].

Diabetic cranial neuropathy is a monophasic illness. No specific requirements is required other than prismatic help of the diplopia as the ptosis improve[11]. Diabetic cranial neuropathy regresses spontaneously after three months in average. After an acute episode, 70 % of patients will cure within 6 months[2]. Spontaneous resolution nature of the disease make the clinical monitoring and equilibration of diabetes is the core of management[2]. Strict glucose control from the time of diagnosis of diabetes mellitus is the most important aspect of the treatment[11].

CONCLUSION

Diabetic cranial neuropathy is one spectrum of diabetic neuropathy. The common clinical presentation is acute painful severe headache with ophthalmoplegia. The key features is oculomotor nerve palsy with sparing of pupillary reflex. Simultaneous involvement of two or more cranial nerve in diabetic cranial nerve patient are uncommon and need further investigation to exclude the other diagnosis. Exclusion of the other possible diagnosis is important to diagnose diabetic cranial neuropathy. Diabetic cranial neuropathy is a monophasic illness, recover spontaneously and need strict control of the diabetes as the main management.

REFERENCES

- [1] Tracy J, Dyck J. The Spectrum of Diabetic Neuropathies. *Phys Med Rehabil Clin N Am* 2008;19:1.-2008;19:26
- [2] Lajmi H, Hmaied W, Jalel WB, Chelly Z, Yakhlef AB, Zineb FB *et al.* Oculomotor palsy in diabetics. *J Fr Ophtalmol* 2017;1836:1.-2017;1836:5
- [3] Papanas N, Heliopoulos I, Piperidou H, Maltezos, E. Simultaneous, painless, homolateral oculomotor and trochlear nerve palsies in a patient with type 2 diabetes mellitus. Neuropathy or brainstem infarction?. *Acta Diabetol* 2006;43:19.2006;43:21
- [4] Shiode T, Oya S, Matsui T. A Case of the Internal Carotid Artery–Posterior Communicating Artery Aneurysm Mimicking Tolosa – Hunt Syndrome. *NMC Case Report Journal* 2015;1:1.-2015;1:3
- [5] Hung CH, Chang KH, Chu CC, Liao MF, Chang HS, Lyu RK *et al.* Painful ophthalmoplegia with normal cranial imaging. *BMC Neurology* 2014;14:1.-2014;14:7
- [6] Quinn R, Hawkes C, Lodhi A, Tang S, Beattie KA, Adel BV *et al.* Ophthalmoplegia in an Elderly Woman with Giant Cell Arteritis. *Rheumatol Adv Pract* 2018;2:1.-2018;2:3
- [7] Weatherall, MW. The diagnosis and treatment of chronic migraine. *Ther Adv Chronic Dis* 2015;6(3):115.-2015;6(3):123
- [8] Mitchell P, Wilkinson ID, Hoggard N, Paley NMJ, Jellinek DA, Powell T *et al.* Detection of subarachnoid haemorrhage with magnetic resonance imaging. *J Neurol Neurosurg Psychiatry* 2001;70:205.-2001;70:211
- [9] Al-hashel JY, Ahmed SF, Youssry D, Alroughani RA, Ismail II, Vembu P. The Value of D-dimer Test for Diagnosis of Cerebral Venous Thrombosis in Emergency Medicine. *Emerg Med* 2015;5:4.-2015;5:6
- [10] Kermani TA, Schmidt J, Crowson CS, Ytterberg SR, Hunder GG, Matteson EL *et al.* Utility of Erythrocyte Sedimentation Rate and C-Reactive Protein for the Diagnosis of Giant Cell Arteritis Tanaz. *Semin Arthritis Rheum* 2013;41:866.-2013;41:871
- [11] Llewelyn JG. The Diabetic Neuropathies: Types, Diagnosis and Management. *J Neurol Neurosurg Psychiatry* 2003;74:15.-2003;74:19
- [12] Venkatesan PE, Gnanashanmugam G, Parimalam N, Pranesh MB. Diabetes plus third nerve palsy not always diabetic third nerve palsy. *J Post Grad M* 2015;61:50.-2015;61:52
- [13] Chou PY, Wu KH, Huang P. Ptosis as the only manifestation of diabetic superior division oculomotor nerve palsy. *Medicine* 2017;96:45.-2017;96:46
- [14] Aslam A, Singh J, Rajbhandari S. Pathogenesis of Painful Diabetic Neuropathy. *Pain Research and Treatment* 2014;2014:1.-2014;2014:7

International Journal of Medical Reviews and Case Reports <ijmrcr@ejmanager.com>
to me

Dear Lothar Matheus Manson Vanende Silalahi,

Your REVISED ARTICLE entitled Rare **Case** of Simultaneous Diabetic Oculomotor and Trochlear Nerve Neuropathy: **Case Report** (Mns No:IJMRCR-2020-01-06) has been received by **International Journal of Medical Reviews and Case Reports**.

You could follow status of your manuscript by login to your author account at www.ejmanager.com.

Thank you for submitting your REVISED version of your article.

Best regards,

Editor
International Journal of Medical Reviews and Case Reports
<http://www.mdpub.net>

Decision Letter to Authors - Acceptance - (IJMRCR-2020-01-06) Inbox x

noreply@ejmanager.com <noreply@ejmanager.com> Wed, Jan 29, 2020, 9:04 PM
to me

Dear Lothar Matheus Manson Vanende Silalahi,

I am pleased to inform you that your manuscript titled as "Rare **Case** of Simultaneous Diabetic Oculomotor and Trochlear Nerve Neuropathy: **Case** Report

" (Manuscript Number: IJMRCR-2020-01-06 was accepted for publication in the **International Journal of Medical Reviews and Case Reports**. You could check your possible publication date at your author page.

You may login to your author account page, and visit accepted articles section in order to get official/formal acceptance letter as PDF.

I would like to remind that you could send your future manuscripts to **International Journal of Medical Reviews and Case Reports**.

Sincerely yours,

#imagesignature#
Ivan Inkov, Dr.
Editor
International Journal of Medical Reviews and Case Reports
inkov@journalmedica.com
<http://mv.ejmanager.com/iimrcr>