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Case report

Delayed identification of massive pituitary apoplexy in pregnancy: A case report

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ABSTRACT

Introduction and importance: Pituitary apoplexy is a very rare cause of sudden and severe headache with a neuro-ophthalmic deficit during pregnancy due to hemorrhage or infarction in the pituitary gland. Delayed identification can be life-threatening to both mother and baby.

Case presentation: A 40-year-old nulliparous female without any prior comorbidities in her 21 weeks of gestation presented with complaints of severe headache, vomiting, decreased vision, and altered sensorium for five days. On a low index of suspicion of pituitary apoplexy, she was managed in the line of impending eclampsia at a local center. On presentation to our center; the neurological deficit had progressed. CT head showed massive pituitary apoplexy with sellar and suprasellar extension. She underwent emergency right pterional craniotomy and resection of the tumor with hematoma evacuation but lost her life on her 2nd postoperative day.

Discussion: In the context of pregnancy, the diagnosis of pituitary apoplexy can get tricky and overlap with other common conditions such as preeclampsia or eclampsia. Magnetic resonance imaging (MRI) is the most sensitive investigation to confirm the diagnosis. Corticotropic deficiency with adrenal insufficiency is a potentially life-threatening disorder for both mother and the fetus if left untreated. The choice between conservative management and surgical approach depends on the neuro-ophthalmic signs, MRI findings, and gestational week.

Conclusion: Pituitary apoplexy should be a differential diagnosis of acute severe headache in pregnancy which when suspected should be investigated promptly. Early identification and multi-disciplinary team management are imperative for better outcomes.

1. Introduction

Pituitary apoplexy (PA) refers to abrupt destruction of pituitary tissue and is a very rare but potentially life-threatening reason for a pregnant woman to present with a sudden onset of headache, ophthalmoplegia, visual and pupillary disturbances, and depressed sensorium [1–3]. It usually results from infarction or hemorrhage into the pituitary gland in an underlying pituitary tumor/adenoma [4].

This clinical syndrome consists of signs and symptoms that occur with the rapid expansion of the contents of the sella turcica [4]. Early diagnosis is important because it can result in a neuroendocrine emergency with acute central hypoadrenalism, hyponatremia, hypotension, and neuro-ophthalmological deficits [2,5–7]. The diagnosis of PA in pregnancy is challenging and delayed as it is rarely a differential diagnosis, which is associated with a significant risk of death for both mother and child [8].

Herein, we report a case of a 40-year nulliparous pregnant woman at 21 weeks of gestation without any prior comorbidities who presented to our tertiary center after 5 days of symptom onset and progression of neurological deficit. She was managed conservatively, misinterpreting as impending eclampsia in a local center. In our center; she was operated on but succumbed to her illness on her 2nd postoperative day. This case has been reported in line with SCARE criteria [9].

Abbreviations: ICU, Intensive care unit; MRI, Magnetic resonance imaging; PA, Pituitary apoplexy.

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2. Presentation of case

A 40-year-old $G_2P_0A_1L_0$ at 21-week period of gestation without any prior co-morbidities presented to our emergency with the complaint of sudden, severe frontoparietal headache for 5 days, projectile non-bilious vomiting, dropping of right eyelid, and bilateral decreased vision along with altered sensorium for 3 days. There was no history of fever, neck stiffness, altered bowel/bladder habit, trauma, pain abdomen, per-vaginal bleed, or any rashes in her body. There was no prior history of chronic headache or history of intake of medications like bromocriptine or cabergoline. Also, her current ante-natal history was uneventful and was perceiving fetal movements normally.

On arrival, she was ill-looking. Her vitals were stable and her Glasgow Coma Scale was E3V5M6 (14/15). During her neuro-ophthalmological evaluation, she could just count fingers with the right eye and vision was 1/60 in her left eye. Right lid ptosis was present. (Fig. 1) The right pupil was 4 mm in size with a sluggish reaction to light with Grade I relative afferent pupillary defect; with a normal left pupil. On ophthalmoscopy, the right eye disc and macular edema were seen. The rest of the cranial nerves were grossly intact. On motor system examination; the power of groups of muscles of bilateral upper and lower limbs was 4+/5. All the reflexes and tone were normal however plantar reflexes were upgoing bilaterally. The rest of the neurological and systemic examinations were unremarkable. On per-abdominal examination, the uterus was 20 weeks in size with fetal parts palpable.

With the provisional diagnosis of impending eclampsia with CN III palsy, she was managed with the injection of $MgSO_4$ in a local center. On hematological investigation, she had hypernatremia, hypokalemia, and elevated uric acid, ALT, and AST. On hormonal profile; prolactin was highly elevated with a blood level of >200 ng/ml (Table 1). On obstetrics scan; a single live intrauterine normal pregnancy of 19^{+2} weeks of gestation with placenta previa was seen.

The patient's party was counseled on the need for an MRI brain. However, due to financial constraints, a non-contrast CT scan of the head with necessary precautions was performed which showed sellar and suprasellar hyperdensity with a cystic hypodensity lesion suggestive of pituitary apoplexy on a pituitary adenoma. (Fig. 2) Due to the deteriorating general condition of the patient and the drop in GCS to 13/15 (E3V4M6); the patient underwent emergency right pterional craniotomy and resection of the tumor with hematoma evacuation. Intra-operatively; sellar/suprasellar mass (soft to firm), with gray and white matter altogether measuring 1.0 cm × 0.4 cm × 0.2 cm, mildly vascular,

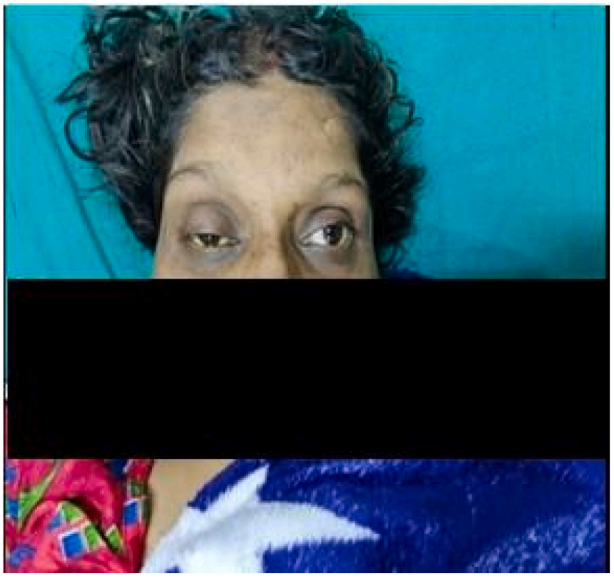

Fig. 1. Right eye-lid ptosis at the time of presentation.

Table 1
Table showing the hematological and biochemistry parameters at the time of presentation and post-operatively.

	Result	Unit	Reference range
Hematology			
TLC	12,000	/cmm	4000–11,000
Hb	11.2	Gm%	12.5–15
Platelets	267,000	/cmm	150,000–400,000
Biochemistry			
RBS	5.8	Mmol/l	3.8–7.8
Urea	2.4	Mmol/l	2.8–7.0
Creatinine	57	umol/l	40–110
Sodium	147.0	Mmol/l	135–146
Potassium	2.6	Mmol/l	3.5–5.2
Uric Acid	455	umol/l	150–340
LDH	210.0	U/L	225–450
Total bilirubin	15	umol/l	3–21
Direct bilirubin	6	uMol/l	4
AST/SGOT	142.0	Units/L	0–35
ALT/SGPT	237.0	Units/L	0–35
Total protein	60.0	Gm/l	60–80
Albumin	34.0	Gm/l	35–52
Lipase	27	U/L	60
Hormonal profile			
fT3	3.06	pmol/L	2.4–6.0
fT4	12.84	pmol/L	9.0–19.0
TSH	0.357	uIU/ml	0.35–4.94
Cortisol	24.0	ug/dl	Morning: 3.7–19.4 Evening: 2.9–17.3
LH	0.05	uIU/ml	Female: 2.58–12.1 Follicular phase: 0.83–15.5 Luteal phase: 27.3–96.9 Mid-cycle: 13.1–86.5
FSH	<0.05	uIU/ml	Female: 1.98–11.6 Follicular phase: 1.38–11.6 Luteal phase: 5.14–23.4 Mid-cycle: 1.5–11.5
Prolactin	>200.0	ng/ml	5.1–26.5
Coagulation parameter and serology			
PT/INR	12/1.0	*sec*	10–12/
HIV Ab/HBsAg/HCV Ab spot/quick	Non-reactive		
Post-operative investigations			
Sodium	161.0	Mmol/l	135–146
Potassium	2.6	Mmol/l	3.5–5.2
pH	7.51		7.35–7.45
pO2	125.9	mm Hg	80–100
pCO2	20	mm Hg	35–45
HCO3	20.2	mmol/dl	22–28

mildly suckable with hemorrhagic component compressing the optic nerve was found. After surgery, she was shifted to ICU, continued on endotracheal intubation, and was under injection hydrocortisone, levetiracetam, fentanyl, mannitol, and labetalol. On MRI (post-op); there was approximately 50 mm × 40 mm mass encasing the right internal carotid artery and cavernous sinus with sellar and suprasellar extension with a central necrotic component with hyperintensity on T2 imaging was seen (Fig. 3). On the 2nd post-operative day, the general condition of the patient became weak and GCS further deteriorated from E1M5VT to E1M1VT. Bilateral pupils were dilated (5 mm), fixed, and non-reactive to light with deranged blood parameters. The patient lost her life on the 2nd postoperative day due to cardiorespiratory failure. The biopsy report revealed multiple fragments of round to oval tumor cells with a moderate amount of eosinophilic to clear cytoplasm composed of uniform round cells and separated by incomplete fibrovascular septa

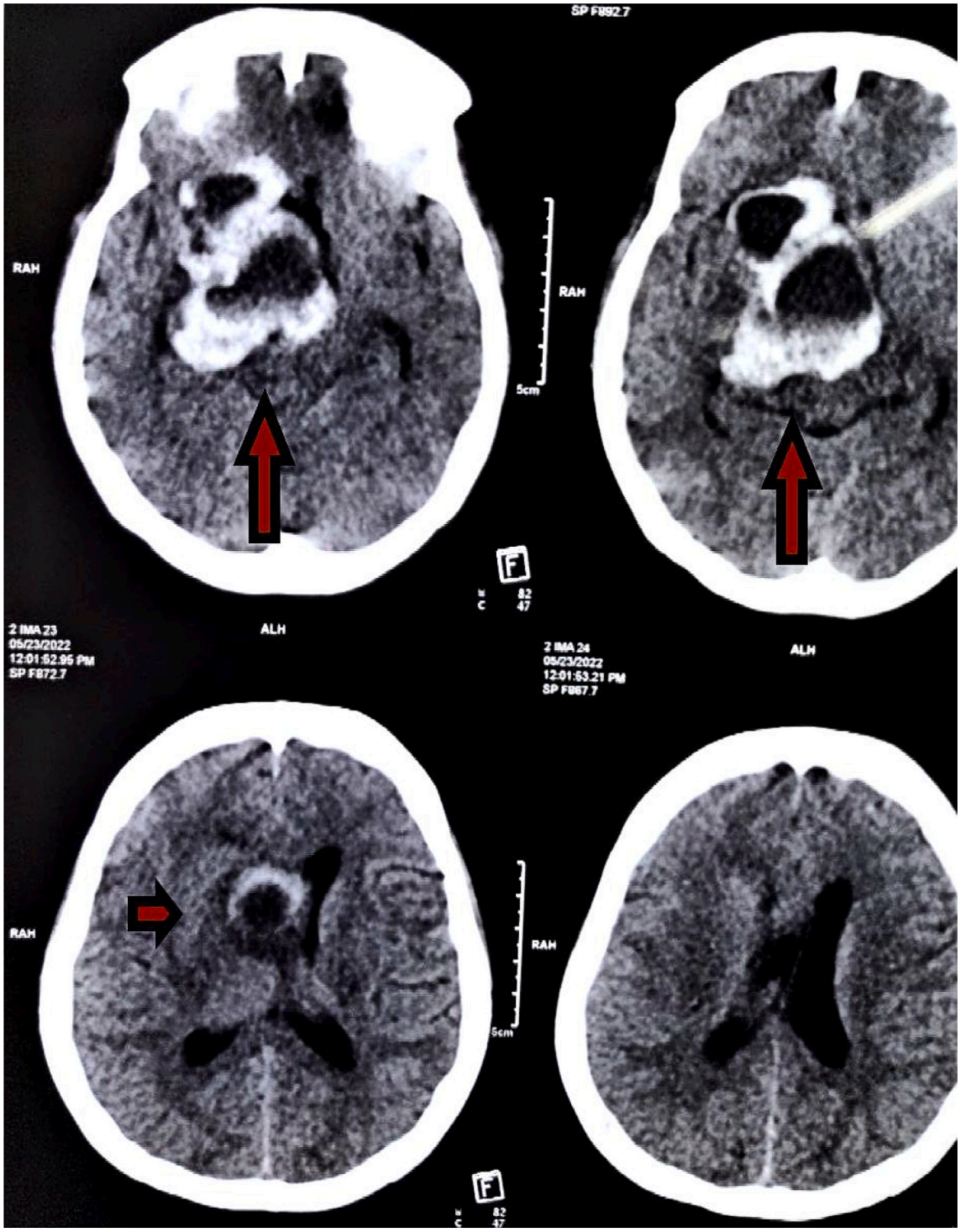

Fig. 2. Non-contrast CT head showing sellar and suprasellar hyperdensity with a cystic hypodensity lesion suggestive of pituitary apoplexy on a pituitary adenoma (arrows).

with the area of hemorrhage; features suggestive of pituitary adenoma (Fig. 4).

3. Discussion

A recent search in the PubMed database for the Medical Subject Headings (MeSH) terms pituitary apoplexy, pituitary disease, and pregnancy showed 98 results, including 35 case reports and series [10]. Overall data of pituitary apoplexy associated with pregnancy is limited to case reports and small case series. As with this case, the majority of incidents appeared in the second or third trimester [11].

Though the exact pathogenesis for this entity remains unknown, an increase in the size of the pituitary gland in pregnancy owing to hormonal change is the proposed mechanism for PA in pregnancy [12]. The majority of cases have underlying pituitary tumors and the risk is much higher with macroadenomas especially macroprolactinomas than with microadenomas [4,6]. Other factors that increase the risk of pituitary tumor apoplexy include head trauma, hypotension, medications such as

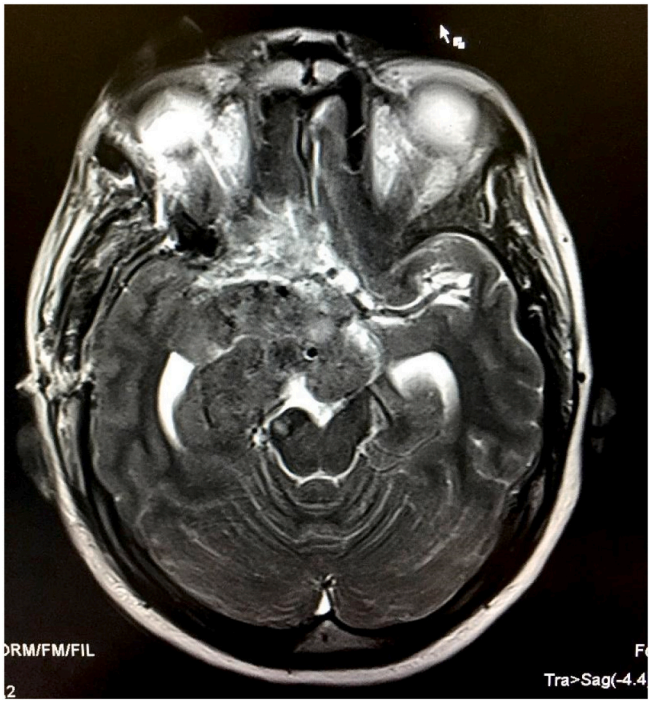

Fig. 3. T2 weighted post-operative MRI showing incomplete evacuation of the mass with central necrotic components.

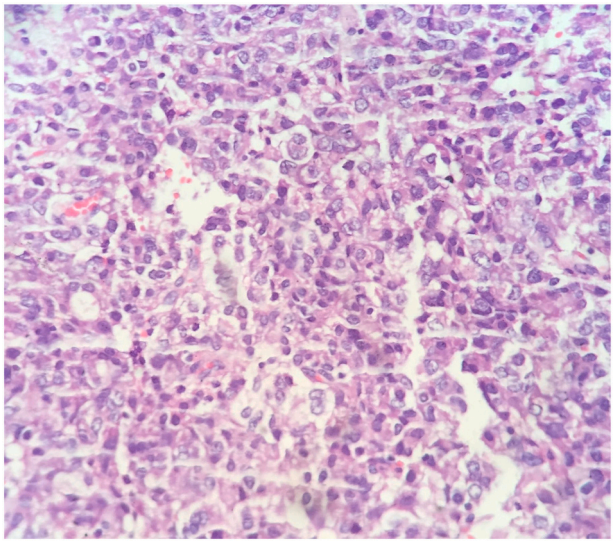

Fig. 4. Histological section of the resected specimen showing multiple fragments of round to oval tumor cells with eosinophilic cytoplasm, separated by incomplete fibrovascular septa; suggestive of pituitary adenoma.

anticoagulants, surgery, pituitary dynamic testing, and history of irradiation and hypertension [4,13].

Pituitary apoplexy usually presents with sudden onset of severe headache, ophthalmoplegia, visual and pupillary disturbances, and depressed sensorium [1–3]. Approximately 80 % of patients will develop a deficiency of one or more anterior pituitary hormones at presentation and can have symptoms of adrenal insufficiency accompanied with circulatory shock [14]. The symptoms in PA are caused by an increase in pressure within the sella turcica produced by hemorrhage into the pituitary gland and its expansion with compression of surrounding brain structure and pituitary tissue itself [5,8]. The clinical picture can mimic multiple neurological conditions including preeclampsia and eclampsia, meningitis, cerebral venous thrombosis, stroke, subarachnoid hemorrhage, and pituitary adenoma, and this is why a high index of suspicion should prompt investigation for pituitary apoplexy and necessary neuroimaging investigation [15,16].

As pregnancy is a condition presenting with hormonal imbalance, interpretation of endocrine and dynamic tests is more difficult. Adrenocorticotropic hormone (ACTH) deficiency is commonly present in pituitary apoplexy, but thyroid-stimulating hormone (TSH), growth hormone (GH), and gonadotropin deficiency have also been reported. Adrenal insufficiency is the most serious complication as it is life-threatening [17]. Hyponatremia complicates pituitary apoplexy as it is a sign of adrenal insufficiency or of the syndrome of inappropriate antidiuretic hormone (ADH) secretion [18]. Therefore, whenever pituitary apoplexy is part of a working diagnosis, a full endocrine (cortisol, ACTH, prolactin, follicle-stimulating hormone, luteinizing hormone, insulin-like growth factor 1, free T4, TSH) and blood assessment (full blood count, glycemia, electrolytes (serum sodium and potassium), and renal and liver function) should be performed urgently [11]. Patients with pituitary apoplexy and low prolactin levels are the most affected and it is unlikely that they will have a successful post-surgical recovery [13]. All the hormonal profiles were within normal limits in our case.

The gold standard for pituitary apoplexy diagnosis is MRI as it confirms the diagnosis in over 90 % of cases, is safe, and also without any damaging fetal effects [19,20]. Anticipation of this clinical entity and prompt recognition of symptoms with the help of a multidisciplinary team may prevent disastrous consequences. However, a real challenge is the diagnosis of pituitary apoplexy in patients with unknown adenomas. Precious time can be lost by interpreting a headache as a migraine type or other secondary headaches. There are several cases, including ours, where pituitary apoplexy was the main cause [11,12,17,21–23].

Once the pituitary apoplectic event is identified in a pregnant woman, care must be directed to both mother and fetus in a manner designed to optimize the physiologic stability of both. A “team” approach is required, which includes a neurosurgeon, ICU personnel, and an obstetrician [24].

Whenever pituitary apoplexy occurs in pregnancy, initial treatment consists of fluid, electrolyte, and hormonal replacement. Meanwhile, it should be emphasized that adrenal insufficiency is a life-threatening condition. Therefore, glucocorticoid input is vital and should be started as soon as pituitary apoplexy is suspected. UK guidelines for pituitary apoplexy recommend 100–200 mg hydrocortisone as an intravenous bolus, followed by 2–4 mg/h intravenous continuous administration or by 50–100 mg every 6 h by intramuscular injection. Once the acute episode is overcome, the steroid regimen should be reduced to a standard maintenance dose of 20–30 mg. It is practical to carry on with medical treatment, and if there is no improvement or a deterioration in clinical condition then surgery must be performed. However, medical management vs surgical decompression is still a matter of debate. In seriously ill patients, the current literature and expert opinion favor surgical decompression [11,25]. Indications for surgical intervention are significant neuro-ophthalmic signs or reduced level of consciousness [25]. Prompt ICU care and subsequent neurosurgical intervention can lead to improvements in neurological deficits. Traditionally, an open transseptal approach may have been used for resection of the pituitary adenoma, but more recently some neurosurgeons may choose to utilize the endoscopic endonasal approach to the sella turcica. Endoscopic, endonasal, and transsphenoidal surgery have comparable surgical outcomes to conventional microscopic transsphenoidal surgery [26]. Our patient has managed accordingly with necessary corticosteroids, and hormones in the emergency department followed by emergency evacuation of apoplexy.

Bromocriptine is a dopamine agonist used as the primary therapy for prolactinomas. Cabergoline is a new dopamine agonist that is proven more effective than bromocriptine in treating pituitary-secreting adenomas while having fewer side effects [27]. Most of the reports of apoplexy in prolactinomas are related to treatment with bromocriptine,

and there are few case reports of cabergoline-induced apoplexy [27–29].

Most data on obstetrical and fetal outcomes and for following pregnancies of affected patients are missing in the reported literature. The available data show that both, conservative as well as operative treatment, have a small impact on delivery and fetal well-being [8]. Though long-term consequences are not reported, full recovery of pituitary function is less common, and therefore, long-term hormone replacement therapy can be required in some patients [4,5]. Also, follow-up with endocrine assessments and cranial MRI scans should be considered to detect possible tumor regrowth and recurrent apoplexy [25].

4. Conclusion

Sudden and severe headaches during pregnancy can be challenging for clinicians, especially in low-resource settings, and require a high index of suspicion for the diagnosis of pituitary apoplexy. Misinterpretation and delayed identification can be life-threatening to both mother and baby. A multi-disciplinary facility is imperative for the proper management of this condition.

Consent

Written informed consent was obtained from the patient's husband for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

Provenance and peer review

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Ethical approval

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Author contribution

Hari Sedai, Suraj Shrestha, Pratima Sharma, Amit Pradhanang, Prajwal Khatiwada = Study concept, Data collection, and medical therapy for the patient.

Hari Sedai, Suraj Shrestha, Elisha Poddar, and Dipendra Dahal = Writing- original draft preparation.

Hari Sedai, Suraj Shrestha, and Elisha Poddar = Editing, and writing.

Amit Pradhanang = senior author and manuscript reviewer.

All the authors read and approved the final manuscript.

Guarantor

Suraj Shrestha.

Registration of research studies

Not applicable.

Declaration of competing interest

None.

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