DOI: 10.7860/JCDR/2017/28903.10407

Case Report

Obstetrics and Gynaecology Section

Isolated Sixth Cranial Nerve Palsy in a Case of Severe Pre-eclampsia Presenting as Postpartum Diplopia

AARTI YEVALE[1], AKHILA VASUDEVA[2], ANJALI MUNDKUR[3], PRATAP KUMAR[4], ARVIND PRABHU[5]

ABSTRACT

Visual symptoms in pregnancy are ominous. The spectrum of ocular symptoms seen in pre-eclampsia includes blurring of vision, scotoma, hemianopia and even total cortical blindness. Diplopia, though rare has also been reported in cases of pre-eclampsia and occurs due to pathological changes affecting the 6th cranial nerve. Ocular symptoms of pre-eclampsia usually regress after delivery but occasionally may present in the postpartum period. We present a case of a 34-year-old lady with severe pre-eclampsia at 31 weeks of gestation with diplopia presenting seven days post caesarean section. No identifiable pathology was found and the condition recovered spontaneously once blood pressure control was achieved.

Keywords: Abducens nerve palsy, Diplopia, Gestational hypertension

CASE REPORT

A 34-year-old G2Ab1 was referred to our institute at 31 weeks of gestation with blood pressure recordings of 180/100 mmHg with otherwise uneventful antenatal period. High blood pressure recordings were detected 10 days prior and antihypertensive treatment with oral labetalol 100 mg twice daily was initiated for the same. She had no complaints of headache, blurring of vision, diplopia, epigastric pain or any imminent symptom of eclampsia. There were no signs of any cranial nerve palsy, meningeal irritation or increased intra cranial pressure. 24 hr urine protein was 713 mg/l; remaining laboratory investigations were normal. There was no biochemical evidence of Haemolysis, Elevated liver enzymes, Low Platelet (HELLP) syndrome. Fundoscopy was normal. Conservative management was employed and blood pressure control was achieved by an increased dose of oral labetalol 200 mg four times a day along with amlodipine 10 mg twice a day, for six days. On the seventh day, despite adequate antihypertensive therapy, blood pressure recordings dangerously rose to 200/120 mmHg. Therefore, conservative management was abandoned and delivery by caesarean section was done. For the first six postoperative days, her blood pressure could not be adequately controlled despite administration of oral labetalol 200 mg every sixth hourly, amlodipine 10 mg twice daily and clonidine 150 micrograms thrice daily. On the seventh postoperative day, patient complained of diplopia. She was afebrile and did not have any complaints of photophobia, phonophobia, headache or any symptoms of increased intra cerebral pressure. Clinical evaluation did not reveal neck stiffness or neurological deficit and therefore a decision for lumbar puncture and cerebrospinal fluid evaluation was deferred. Repeat fundoscopy was normal. There was restricted abduction of left eye and diplopia charting revealed isolated sixth cranial nerve palsy. MRI brain and Magnetic Resonance Venography (MRV) was normal and did not reveal any evidence of increased intracranial tension. Since the onset of symptoms was acute, a differential diagnosis of Posterior Reversible Encephalopathy Syndrome (PRES) and pre-eclampsia induced sixth cranial nerve palsy was considered. In the absence of any other cause, a diagnosis of pre-eclampsia induced isolated sixth cranial nerve palsy was made. The patient was observed and continued on antihypertensive medication. No specific treatment was given for the sixth cranial nerve palsy. Over a period of 4-5 days, blood pressure was adequately controlled medically. The diplopia regressed by postoperative day 12.

DISCUSSION

Isolated sixth cranial nerve palsy is a rare but documented complication in pre-eclampsia. Yousefi SR and Zandi S reviewed literature and found nine cases of abducens nerve palsy occurring in pregnancy, eight of which had isolated Abducens nerve palsy [1]. It's aetiology is uncertain, only theorized, which include inflammation of nerve fascicles due to inflammatory mediators, downward displacement of the nerve due to increased intracranial pressure or cerebral oedema [2,3] and infarction of nerve fascicle [4]. The sixth cranial nerve originates from the pons, runs a long course through the cavernous sinus, finally entering the orbit through the superior orbital fossa where it pierces the lateral rectus supplying it. Its only action is abduction of the eyeball [5]. Donaldson D and Rosenberg NL reported a case of sixth nerve palsy in an elderly hypertensive patient. CT scan revealed a pontine infarct [4]. It was suspected that infarction of the abducens nerve fascicles at the point of origin from the pons was responsible for the symptoms. With regeneration of the nerve fascicles, the symptoms subsided. In pre-eclampsia, a similar pathophysiological process may be responsible.

Fung TY and Chung TK reported a case of isolated sixth cranial nerve palsy in a case of gestational hypertension at 38 weeks gestation, with blood pressure recordings of 140/90 mmHg and no other neurological deficit [2]. CT brain, renal, liver and haematological evaluation were normal. Ds-DNA antibody, serum complement (C3,C4) levels were also normal. Blood pressure returned to normal post delivery without any anti hypertensives and her symptoms regressed by the sixth post partum day. They concluded that if there is no associated pathology, nerve recovery is almost always complete.

Vallejo-Vaz AJ et al., reported a case of sixth cranial nerve palsy in a 36-year-old pre eclamptic lady at 36 weeks of gestation [6]. They also reviewed seven other cases of sixth cranial nerve palsy in pregnancy, five of them were complicated by hypertensive disorders of pregnancy. The time of recovery varied from six days to six weeks postpartum. In all cases, however, recovery was complete [2,3,7-11].

Serial number	Author	Associated high blood pressure recordings/ Eclampsia/pre-eclampsia	Time of presentation of palsy	Additional comments	Time till recovery of palsy
1	Yousefi SR and Zandi S[1]	Pre-eclampsia	Antepartum at 39 weeks		5 days
2	Fung TY and Chung TK [2]	Gestational HTN	Antepartum at 38 weeks	-	6 days
3	Barry-Kinsella C et al., [3]	Pre-eclampsia	Antepartum at 38 weeks	Associated frontal headache	2 weeks
4	Vallejo-Vaz AJ et al., [6]	Pre-eclampsia	Antepartum at 36 weeks	Associated frontal headache	< 12 weeks
5	Bladé J et al., [7]	Eclampsia	Postpartum,<24 hours	Coma, delivered at 40 weeks gestation	6 months
6	Sternberg I et al., [8]	No	Antepartum at 6 months of gestation	Post febrile disease, recurrent	3 months
7	Thurtell MJ et al., [9]	Pre-eclampsia	Postpartum on day 11	Delivered at 30 weeks gestation	Slowly after months
8	Thamban S et al., [10]	No	Antepartum at 40 weeks	-	6 weeks
9	Park CM et al., [11]	Pre-eclampsia	Postpartum <24 hours	Foetal growth restriction, delivered at 39 weeks gestation	7 days
10	Case	Pre-eclampsia	Postpartum on day 7	Delivered at 31 weeks gestation	12 days

[Table/Fig-1]: Cases of isolated sixth cranial nerve palsy in pregnancy.

Barry-Kinsella C et al., reported a case of isolated sixth cranial nerve palsy in a pre eclamptic lady at 38 weeks gestation [3]. Her blood pressure ranged between 140/90-160/100 mmHg. Evaluation for diplopia was inconclusive. No neurological deficit was found. Postpartum blood pressure was controlled medically for two weeks and symptoms subsided.

This condition is a diagnosis of exclusion. An MRI brain should be taken in order to rule out any pathology such as PRES, a tumour compressing the sixth nerve or cerebral oedema. Anti-Neutrophil Cytoplasmic Antibodies (ANCA), Anti-Nuclear Antibodies (ANA), complement levels and Erythrocyte Sedimentation Rate (ESR) may be evaluated to rule out connective tissue disorders and small vessel diseases like giant cell arteritis. In a patient presenting with fever and signs of meningeal irritation, a lumbar puncture with cerebrospinal fluid evaluation will be helpful in diagnosing meningitis [12]. Once a diagnosis of isolated sixth cranial nerve palsy is made, no specific treatment is needed. The condition resolves spontaneously with full recovery of nerve function. [Table/Fig-1] describes the series of isolated sixth cranial nerve palsy in pregnancy.

CONCLUSION

Isolated sixth cranial nerve palsy is a rare complication of pre-eclampsia and hypertension in pregnancy. Commonly presenting in the antenatal period, it may also present postnatally. The condition does not require any specific treatment and resolves once blood pressure is controlled. It is a diagnosis by exclusion and neuro imaging must be done in all cases.

REFERENCES

[1] Yousefi SR, Zandi S. Abducens nerve palsy in pregnancy: a case report. J Clin Diagn Res. 2016;10(12):QD03–QD04.
[2] Fung TY, Chung TK. Abducens nerve palsy complicating pregnancy: a case report. European Journal of Obstetrics and Gynecology and Reproductive Biology. 1999;83(2):223-24.
[3] Barry-Kinsella C, Milner M, McCarthy N, Walshe J. Sixth nerve palsy is an unusual manifestation of preeclampsia. Obstetrics and Gynaecology. 1994;83(5):849-51.
[4] Donaldson D, Rosenberg NL. Infarction of abducens nerve fascicles as cause of isolated sixth cranial nerve palsy related to hypertension. Neurology. 1988;38:1654.
[5] Azarmina M, Azarmina H. The six syndromes of the sixth cranial nerve. J Ophthalmic Vis Res. 2013;8(2):160-71.
[6] Vallejo-Vaz AJ, Stiefel P, Alfaro V, Miranda ML. Isolated abducens nerve palsy in preeclampsia and hypertension in pregnancy. Hypertension Research. 2013;36(9):834-35.
[7] Blade´ J, Peborde J, Darleguy P. Paralysie du VI au cours d´ une e´ clampsie. Bull Soc Ophtalmol Fr 1968;68:284–87.
[8] Sternberg I, Ronen S, Arnon N. Recurrent, isolated, post-febrile abducens nerve palsy. Journal of Pediatric Ophthalmology and Strabismus. 1980;17(5):323-24.
[9] Thurtell MJ, Sharp KL, Spies JM, Halmagyi GM. Isolated sixth cranial nerve palsy in preeclampsia. J Neuro-Ophthalmol. 2006;26:296–98.
[10] Thamban S, Nama V, Sharma R, Kollipara PJ. Abducens nerve palsy complicating pregnancy. J Obstet Gynaecol. 2006;26:811–12.
[11] Park CM, Kim SY. Abducens nerve palsy in preeclampsia after delivery: an unusual case report. J Obstet Gynaecol Res. 2007;33:543–45.
[12] Weiner CP. The clinical spectrum of pre ecalmpsia. Am J Kidney Dis.1987;9:312-16.

PARTICULARS OF CONTRIBUTORS:
1. Junior Resident, Department of Obstetrics and Gynaecology, Kasturba Medical College and Hospital, Manipal, Karnataka, India.
2. Associate Professor, Department of Obstetrics and Gynaecology, Kasturba Medical College and Hospital, Manipal, Karnataka, India.
3. Associate Professor, Department of Obstetrics and Gynaecology, Kasturba Medical College and Hospital, Manipal, Karnataka, India.
4. Professor, Department of Obstetrics and Gynaecology, Kasturba Medical College and Hospital, Manipal, Karnataka, India.
5. Assistant Professor, Department of Neurology, Kasturba Medical College and Hospital, Manipal, Karnataka, India.

NAME, ADDRESS, E-MAIL ID OF THE CORRESPONDING AUTHOR:
Dr. Akhila Vasudeva,
Kasturba Medical College and Hospital, Manipal-576104, Karnataka, India.
E-mail: akhilavasudeva@gmail.com

FINANCIAL OR OTHER COMPETING INTERESTS: None.

Date of Submission: **Mar 29, 2017**
Date of Peer Review: **Jun 07, 2017**
Date of Acceptance: **Jul 05, 2017**
Date of Publishing: **Aug 01, 2017**