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Terson's syndrome in a pregnant woman: A fatal presentation

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ABSTRACT

Terson's syndrome is a condition of vitreous haemorrhage occurring in association with subarachnoid haemorrhage (SAH). This condition is uncommon during pregnancy. We report a fatal case of Terson's syndrome in a pregnant lady. She presented with an episode of seizure and complained of blurring of vision in the right eye after the seizure. Examination revealed subhyaloid haemorrhage in the right eye. The left fundus was normal. Subsequent investigations revealed a posterior communicating artery aneurysm. We would like to highlight that any case of non-traumatic vitreous haemorrhage with neurological symptoms and signs must be worked up as neuro-ophthalmic emergency.

1. Introduction

Terson's syndrome is a condition of vitreous haemorrhage occurring in association with subarachnoid hemorrhage (SAH). It was first reported by Albert Terson in 1900. Later, this term was altered to encompass any intraocular haemorrhage associated with intracranial haemorrhage. Intraocular haemorrhage includes preretinal, intraretinal, subretinal, subhyaloid or vitreal blood[1]. In adult, Terson's syndrome most commonly occur secondary to subarachnoid haemorrhage caused by ruptured cerebral aneurysm[1].

Aneurysmal SAH is rare in pregnancy but can be disastrous for both mother and baby. Incidence of SAH due to ruptured cerebral aneurysm ranges between 1/1 100 to 1/25 000 cases during pregnancies[2]. Hemodynamic and hormonal changes during pregnancy are associated with aneurysmal growth[3]. The mortality rate can vary from 13% to 35% for the mother and from 7% to 25% for the fetus when an anuerysmal rupture complicates pregnancy[2]. A prompt diagnosis with timely surgical intervention may reduce maternal mortality and morbidity.

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2. Case report

A 32-year-old Malay lady Gravida 2 Para 1 at 22 weeks of pregnancy, presented with an episode of generalized tonic clonic seizure which lasted about 5 min. It was associated with up rolling of eyeball and urinary incontinence. The fit aborted spontaneously. There was post-ictal drowsiness. She had headache and vomiting prior to the fit. There was no history of fever or trauma. On arrival to casualty, patient had full Glasgow Coma Scale (GCS). Vital signs were stable. Blood pressure was normal. Body temperature was normal. There was no neck stiffness. Examination revealed no neurological deficit. Fetal heart was present.

Patient had been well prior to this incident except for persistent proteinuria which was detected during antenatal visits and was still under investigations. Her blood pressure readings during antenatal visits were within normal range. Her first pregnancy was uneventful. There was no significant past ocular, medical or surgical history.

Patient was referred to the ophthalmology team when she noticed blurring of vision in the right eye a few hours after the seizure. She described that her central vision was obscured. There was no eye pain or redness. There

was no trauma to the eye during the seizure according to her husband who witnessed the fit. On examination, vision in the right eye was counting finger at 3 feets and left eye was 6/6. Anterior segment examination was normal bilaterally. Right fundus examination revealed a large subhyaloid haemorrhage involving the posterior pole. The left fundus was normal with no hemorrhage or neovascularisation (Figure 1). There was no papilloedema. Blood investigation showed that coagulation profile was normal.

Electroencephalography was reported as normal with no evidence of epileptic foci. An urgent magnetic resonance imaging (MRI) brain was done and showed a right frontal cystic lesion with areas of hyperintense signals in the sulci, unable to exclude SAH. Magnetic resonance angiography (MRA) did not reveal any abnormal vessel. The patient was counselled for lumbar puncture for further assessment to detect SAH. However, she refused and took discharge against medical advice.

She presented the next day after discharge with status epilepticus. After stabilization, she was arranged for an urgent computed tomography (CT) scan which showed multiple intracranial haemorrhages. Later, conventional angiography was performed and it showed left narrow neck post communicating artery aneurysm (Figure 2). She was planned for further surgical intervention but unfortunately her condition deteriorated and she succumbed to it.

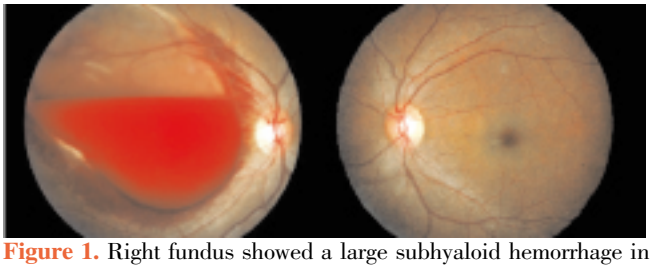

Figure 1. Right fundus showed a large subhyaloid hemorrhage in the posterior pole. The left fundus was normal.

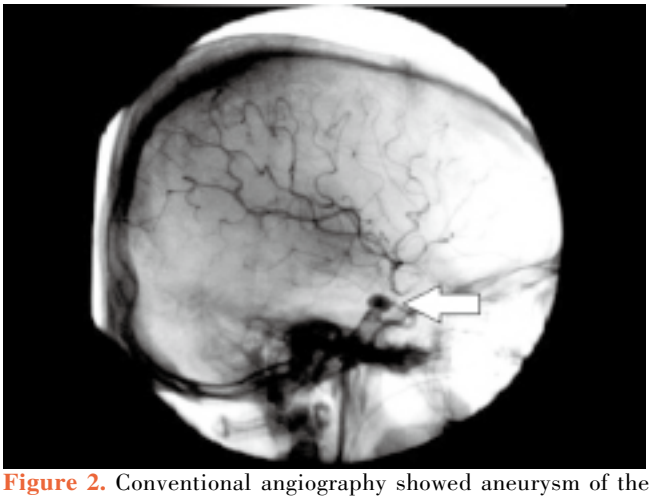

Figure 2. Conventional angiography showed aneurysm of the posterior communicating artery.

3. Discussion

Subarachnooid haemorrhage (SAH) is caused by a ruptured cerebral aneurysm in about 80% of cases[4]. Terson's syndrome is mostly reported with aneurysms of the anterior circulation, commonly arising from the anterior communicating artery[1]. Watanabe Sung et al reported in their study that Terson's syndrome was found to occur in 29% of their patients who had SAH[5]. The presence of Terson's syndrome is associated with higher mortality rate as compared to those without the syndrome[5,6].

Terson's scccyndrome is under diagnosed in the acute setting as patients are usually in critical condition rendering them unable to voice their visual complaint. Ophthalmic consultation is usually done after the acute stage of life threatening condition. Terson's syndrome was found to occur in 13% of patients with SAH[7]. In pregnancy, Terson's syndrome was reported to be present in approximately 25% of patients with aneurismal SAH[3]. Funduscopic examination is essential in patients with SAH to prevent visual morbidity. Ocular haemorrhages in Terson's syndrome tend to be bilateral but can be uniocular[1,6]. Ocular hemorrhages were reported to be bilateral in 57% of cases[5].

The pathogenesis of Terson's syndrome remains controversial. An early theory suggests that the resulting intraocular haemorrhage comes from direct dissection of SAH down the optic nerve sheath which has continuation with the subarachnoid space[1,4]. It is postulated that blood passed through the lamina cribosa to appear intraocularly. However, this theory is less favoured as no connection is found between the vitreous cavity and subarachnoid space. Another theory proposed that the sudden spike in intracranial pressure during the intracranial bleed causes a quick effusion of cerebrospinal fluid into the optic nerve sheath. This causes an increased pressure along the optic nerve. Venous stasis subsequently developed and lead to venous hypertension and eventually causes rupture of the retinal vessels[4,8].

The presentation of SAH due to ruptured aneurysm can be easily confused with eclampsia especially when patient presents with seizure along with blood pressure variation and proteinuria[2]. Other symptoms and signs to suggest eclampsia include weight gain, epigastric pain, progressive headache, thrombocytopenia and deranged liver enzymes. A high index of suspicion is essential for early diagnosis of SAH in pregnancy.

CT scan is the diagnostic study of choice to diagnose SAH[2,3,9]. However, some clinicians prefer MRI to limit

the potential risk of exposure to radiation especially during pregnancy. There is concern that MRI is relatively insensitive in detecting SAH in the acute stage[9]. The radiation effect of CT scan on the fetus is considered relatively small and far outweighed its benefits. Evidence suggests that there is no increase risk in fetal malformation, restriction in growth and abortion in radiation dose less than 50 mGy[3]. Cerebral angiography is the gold standard in diagnosing cerebral aneurysm. MRA may be used to detect cerebral aneurysm but it is still considered inferior to digital subtraction angiography[3].

In our case, patient had blurring of vision noticed after the seizure and examination revealed a large subhyaloid hemorrhage in the posterior pole of the right eye. Fundoscopic finding in this patient has provided the managing clinicians with valuable clues to correlate her signs and symptoms with possible diagnosis of life threatening intracranial conditions. MRI and MRA were performed initially for our patient but there were no clear cut diagnosis.

Thus, patient was advised for lumbar puncture which is sensitive to detect SAH. Unfortunately, patient refused. Eclampsia was a differential diagnosis in our case but history of pre-existing hypertension is lacking, making it less likely, as blood pressure readings during antenatal visits were within normal range.

The principles of management of aneurysmal SAH in pregnancy are similar to non-pregnant patients[3]. A multidisciplinary team approach is essential. Obstetric management would depend on the gestational age of the fetus and is proceeded as best for the mother. Management of intraocular haemorrhage in patients with Terson's syndrome should be individualized. The haemorrhage will usually resolve by itself.

Observation and conservative management is reasonable. However, vitrectomy is indicated to hasten recovery of vision in haemorrhages which are nonclearing or unlikely to clear within a reasonable time frame. Other factors such as the extent of the vitreous haemorrhage, bilaterality, co-existing retinal detachment and visual needs of the patient are to be considered in the plan management. Thus, management of the intraocular haemorrhages can vary from case to case.

We report this case to highlight that any case of non-traumatic vitreous hemorrhage with neurological symptoms and signs must be worked up as neuro-ophthalmic emergency to prevent fatal consequences. A high index of suspicion is needed for early diagnosis of SAH in pregnancy. Ocular hemorrhages in Terson's syndrome are usually bilateral but can be uniocular.

Conflict of interest statement

We declare that we have no conflict of interest.

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