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A Case Report of Post Partum Uterine Inversion with Sheehan's Syndrome

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Introduction

Puerperal uterine inversion is a rare event but a life threatening complication of 3rd stage of labor in which uterus is turned inside out following delivery. It can produce severe postpartum hemorrhage (PPH) & maternal collapse.

Immediate reposition & resuscitation will save the patient & rarely laparotomy or hysterectomy may be needed. Sheehan's Syndrome (SS) may follow severe PPH with hypotension which is due to ischemia of the arteries feeding the anterior pituitary. Prevalence of SS in India is 2.7 -3.9 %. Clinical signs & symptoms are related to the deficiency of the anterior pituitary hormones of which most common is amenorrhea & lactation failure. Management is by appropriate hormone replacement.

Case Summary

A28 year old lady from a poor family belonging to a state outside Kerala, had her 3rd full term delivery at home developed acute inversion of uterus with severe bleeding. Taken to a nearby hospital immediately. Laparotomy was done there, could not correct inversion. Six units of blood transfused & referred to a higher center. Did not turn to any hospital due to shortage of money. As her parents are working in Kerala, she came to Kerala about two months after delivery. Brought to our institution after referring from a local hospital as chronic inversion of uterus. Previous two were home deliveries without any complications. Antenatal period uneventful according to the patient. No breast milk secretion after delivery.

Clinical Status

She was emaciated, pale, otherwise general condition good .Vitals were stable. Both breast normal without milk secretion. Uterus not felt abdominally. A reddish globular mass of size 5x 5 cm seen protruding through the vagina which bleed on palpation. Cervix not felt on pelvic examination. Mass was irreducible. Diagnosed as third degree uterine inversion with lactation failure. USS done, showed uterine fundus noted in the vagina. Uterus appears to be mirror image of normally situated uterus. No other pelvic

pathology. Prepared for surgery. All blood investigations were normal. Hemoglobin was 8.9gm%. Anemia corrected by blood transfusion.

Reposition of inverted uterus tried under anesthesia but failed. Proceeded to Total Abdominal Hysterectomy. At laparotomy uterine fundus was not seen, a dimpled area was noted in the region of fundus below which there was a tough constriction ring. Both tubes and ovaries were normal and was drawn inwards through the dimpled area, Right ovary was adherent to the tube, same could not be retained. Uterus was normal in size, gangrenous and was protruding outside the vagina which was delivered up after cutting the constriction ring from above. One unit blood transfused during surgery .Otherwise surgery was uneventful. Postoperative period uneventful. Was not willing to do further evaluation due to lack of money. Discharged from the hospital at request on 8th postoperative day.

On 12th postoperative day readmitted with c/o diarrhea, dizziness, nausea, headache, lack of interest for baby, self & surroundings. O/E patient was drowsy. Breast milk secretion nil. Not anemic. Bradycardia present. BP normal. Afebrile except for one or two fever spikes. Supportive measures given to improve general health. Investigated with a provisional diagnosis of SS with sepsis. Important investigations done were Blood sugar, Sepsis markers, serum Cortisol, Thyroid, Renal & Liver function tests, X-Ray chest, ECG, USS & MRI .Started on parenteral antibiotics, InjectionHydrocortisone100mg I/V BD & Tab Thyroxin 50 mg OD. Other hormonal evaluation not done as she cannot afford the cost and not willing also. Hemoglobin, Blood sugar, Liver & Renal function test values normal. Fever markers negative. CRP was positive .TSH value was normal (1.2mcg/dl), FT3 & FT4 were reduced 0.42mcg & 3.19mcg/dl respectively. Serum cortisol reduced 1.23mcg/dl. Sodium value low 122meq/l. X-ray chest &Ultrasound abdomen did not reveal any abnormality.ECG showed bradycardia, not significant as per the Cardiologist's opinion

MRI Brain –Atrophic pituitary gland with heterogeneous T2 signal intensity seen with T1 hyperintense non enhancing foci (bleed) showing peripheral enhancement and internal non enhancing areas suggestive of anterior pituitary necrosis .

Patient symptomatically improved. Serum cortisol, T3 & T4 f were found to be reduced on repeat test also.TSH was again normal. Sodium level became normal. Bradycardia persisted. Discharged on 10thday of readmission, advised to continue Prednisolone 100 mg tablet BD & Thyroxin 50mg OD. Patient did not turn for follow up as she belongs to a state outside Kerala.

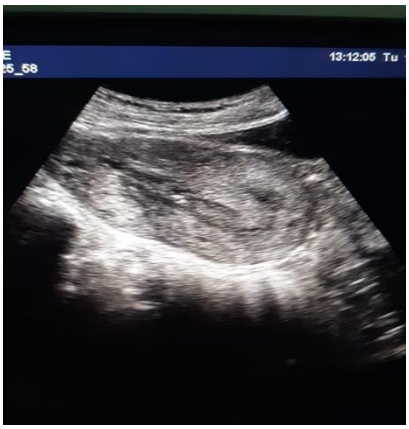

Fig 1: Ultrasound of the patient showing uterine inversion.

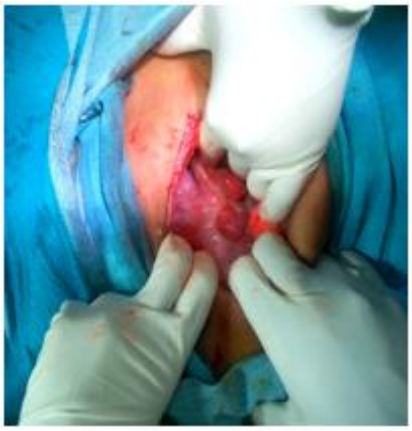

Fig 2: Laparotomy findings. Fallopian tubes drawn into dimpled area.

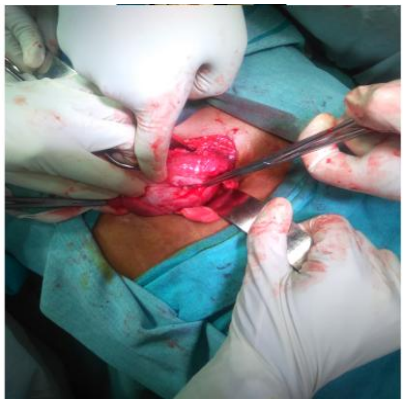

Fig 3: Arrow showing ovaries drawn into dimpled area

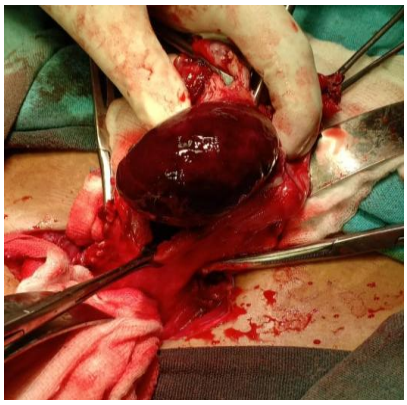

Fig 4: Gangrenous uterus delivered into the pelvis after incising constriction ring.

Discussion

Inversion of uterus refers to the descent of the uterine fundus to or through the cervix so that uterus is turned inside out partially or completely. Incidence is about 1:17000 to1:20000 deliveries. Depending on the time of onset it can be puerperal or non-puerperal. On the basis of duration of onset it can be classified as acute inversion is one occurring within 24 hours of delivery, sub-acute after 24 hours & within 4 weeks of delivery, chronic is one that happens beyond 4 weeks postpartum[1]. Different types of inversion are1stdegree, where depressed fundus of inverted uterus remains in the uterine cavity, bulges into the uterine cavity but not passing through cervix, 2nddegree means fundus protrudes through the external os and enter the vaginal canal and in 3rd degree fundus protrudes through vagina & appears outside vulva[2].

Postpartum shock is a constant feature which is due to stretching of the nerves in the infundibulopelvic ligament, peritoneal irritation or pressure on the dragged ovaries .Other complications include severe hemorrhage, DIC & pulmonary embolism[2]. Diagnosis is made by P/V examination where globular mass which is the uterine fundus, protruding through dilated cervix & appear outside vaginal orifice in 3rd degree inversion. Diagnosis can be confirmed by USS. USS & MRI is mostly useful in 1stdegree inversion. Management of a/c inversion mainly depends on circumstance it developed and condition of the patient. Immediate management is resuscitation, correction of shock, manipulative reposition of fundus by firm constant pressure on the inverted uterus till replacement is effective[3]. Reposition may be done under anesthesia if attempt fails without anesthesia which in addition to pain relief, promote uterine relaxation which will help in reposition also.

Hydrostatic replacement by O Sullivan's method is the best method where replacement is done under hydrostatic pressure[3]. Last resort is laparotomy. At laparotomy inverted fundus can be pulled back in to pelvis with simultaneous push from vagina without damaging the organ. Sometimes the constriction ring has to be cut for reposition. May end up in hysterectomy occasionally. If not corrected, will become chronic one, uterus become Infected &gangrenous & Sloughing of uterus will occur[4]. Repositioning is not easy in delayed cases. Laparotomy & correction may be needed. Huntington's or Haultane's methods are the commonly used procedures. Silastic vacuum cup can be used from above to deliver the fundus at laparotomy. Hysterectomy is indicated in case of chronic inversion when conservative procedures are not feasible. Prognosis is poor in unfavorable situations like low socioeconomic, non - availability of good healthcare system & home delivery.

SS is pan hypopituitarism that develop after severe PPH complicated by prolonged hypotension. Pathogenesis is supposed to be as a result of spasm blood vessels of anterior pituitary

resulting in ischemia & edema, finally necrosis & thrombosis of the portal sinuses and capillaries[5]. Pituitary necrosis secondary to arterial vasospasm & ischemia may occur within hours after severe PPH. Rarely SS can develop following shock due to other causes & severe preeclampsia. In cases of DIC fibrin deposition in capillaries occurs leading to vasospasm. Varying degree of endocrine deficiency syndrome occurs due to loss of anterior pituitary hormones. The pituitary cell types are differentially sensitive to damage[6].Prolactin secretion deficiency is the commonest one occurring in about 90-100% of cases where as Gonadotropins, TSH and ACTH range from 50-100%. Hyponatremia is present in 30% of cases[4].

75% of the pituitary gland has to be destroyed for clinical manifestations to appear. Signs and symptoms may take months or years to appear. Earliest presentation is absence of lactation and breast engorgement. Thereafter features of ovarian failure like lethargy, amenorrhea, loss of pubic & axillary hair, atrophy of breast& external genitalia develops[4].Later adrenal and thyroid failure appears. Acute crisis can occur due to hypothermia, hypoglycemia, hypothyroidism or adrenal failure. In severe variety a/c maternal shock & pituitary apoplexy with features of severe hyponatremia, Diabetes Insipidus, Hypothyroidism, Congestive cardiac failure or psychosismay occur. Obesity due to myxedema is seen in cases of severe thyroid deficiency but most common presentation is with cachexia.

Usual course is progressive deterioration. Incomplete variety will have clinical presentation depending on hormones involved. If the signs are very much delayed diagnosis may be missed. Secondary adrenal insufficiency is difficult to diagnose as the secretion is pulsatile, diurnal rhythm and stress response.

Typical history, signs & symptoms usually help in diagnosis. Estimation of blood levels of trophic hormones, Estrogen, prolactin T3 &T4levels will show low values. TSH will be low or normal usually. Morning serum cortisol level < 3 micro gram/ dl indicate adrenal insufficiency. Various Dynamic stimulation tests can be used for confirmation which include insulin tolerance test, Corticotrophin stimulation test, clomiphene citrate challenge test, trophic hormones such as LHRH to test the target organs. More specific dynamic testing of hypothalamo pituitary adrenal axis is insulin tolerance test. MRI of brain will confirm the diagnosis by presence of evidence of pituitary necrosis

Treatment of SS is replacement of the hormone which is deficient. Lifelong Growth hormone replacement is needed in case of deficiency. Adrenal insufficiency is treated with glucocorticoids like hydrocortisone or prednisolone, Thyroid hormone replacement in case of hypothyroidism and Hormone replacement in hypogonadism. Infertile patient may require ovulation inducing agents. Lifelong follow up with hormonal evaluation and special attention required during stress situations.

Conclusion

Uterine inversion and SS are very rare life threatening situations following delivery. Timely diagnosis and appropriate management can save the life of the patients

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Declaration

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