INTRODUCTION:

Neural tube defects (NTDs) are a group of conditions in which an opening in the spinal cord or brain remains from early in human development.

In the 3rd week of pregnancy called gastrulation, specialized cells on the dorsal side of the embryo begin to change shape and form the neural tube. When the neural tube does not close completely, an NTD develops. The two most common neural tube defects are spina bifida and anencephaly. In anencephaly, CHIARI II malformation, causes the brain tissue to extend into the spinal canal.

Anencephaly occurs when the neural tube fails to close properly at the head. The result is the lack of development of a large portion of the brain and skull. In spina bifida, the fetal spinal column doesn't close completely. There is usually nerve damage that causes at least some paralysis of the legs. Spina bifida is generally classified as either "closed," where the skin covers the defect, or as "open," where the skin is not intact.

Myelomeningocele is the most severe type of spina bifida. It is a open neural tube defect .It develops when the cerebrospinal fluid, meninges, and the spinal cord protrude through the opening in the spine. It leaves the spinal cord vulnerable to damage and can cause paralysis in those parts of the body below the opening. Those affected with a myelomeningocele frequently have bowel and urinary dysfunction. Some will require assistance to walk and others will require a wheelchair. Newborns with this condition are at an increased risk of developing meningitis.

Approximately 70-90% of those affected by myelomeningocele will develop hydrocephalus. This complication, Hydrocephalus occurs when the flow of cerebrospinal fluid is obstructed and accumulates in the brain. The child's head becomes larger and the fluid applies pressure on the brain. Left untreated, this condition can cause mental retardation and learning disabilities and can, in some cases, be fatal.

CASE REPORT:

A female patient of 29 years was admitted in OBG ward with 21 weeks and 5 days of gestation. In trimester 1 she had no history of vomiting, fever, bleeding or leaking per vagina. Bilateral pedal edema was present. Inj T.T 1\textsuperscript{st} dose was taken at this period along with iron & calcium tablets.

In trimester 2 she took 2\textsuperscript{nd} dose of Inj.T.T .Iron and calcium tablets were also taken. There was no history of twinning, abortion, still birth or congenital abnormality in the family .She was married at age of 29 and conceived 6 months after marriage.

She didn’t use any contraceptives methods or any treatment for infertility. Patient doesn’t have a history of other diseases and no addictions were present.
Her USG showed open lumbar neural tube defect with fetal hydrocephalus suggestive of CHIARI 2 malformation (lemon sign). Fetus showed breech position. Patient came for MTP in hospital.

On first day T.Mifeprostone 3 Tab 200mg was given. T.Misoprostol 150mcg was given on third day. Since labour was not induced patient was given additional dose of T.Misoprostol 100mcg, 400mcg, on fourth and fifth day respectively. Patient was then given T.Mifeprostone 200 mg along with T.Misoprostol 200mcg for induction of labour on the sixth day.

On the next day T.Misoprostol 200mcg was given along with Pitocin drops (40 drops at an interval of 15 min was given for 1.5 hours). She expelled dead fetus (female) spontaneously by breech along with entire placenta and intact membrane.

**DISCUSSION:**

Many genetic & environmental factors have been implicated in the causing NTDs. A slight female predominance, higher incidence in certain ethnic groups and in the offspring of consanguineous marriages, have suggested a genetic basis for NTDs. Chromosomal abnormalities also have been associated with NTDs. Marked seasonal trends in the birth incidence of NTDs have also been reported.

The survival rate of fetuses with an NTD has increased due to the multidisciplinary team approach with delivery at a tertiary care center. However, due to the significant sequelae of these lesions, the mean longevity of these patients is reduced to less than 40 years of age, with significant compromises to their quality of life. Approximately 14% of the newborns do not survive beyond age 5 years. At least 45% of these children suffer complications from the ventriculoperitoneal shunt placements within the first year of repair.

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**CONFLICT OF INTEREST:** The author declares there is no conflict of interest.

**REFERENCE:**


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