ANENCEPHALY AND ITS ASSOCIATED CONGENITAL ANOMALIES: A CASE REPORT OF A DELIVERY IN A RURAL HOSPITAL, IN EASTERN UGANDA

Abstract

Introduction: Anencephaly also called cranioschisis is part of neural tube defects spectrum which occur when the neural tube fails to close normally during the 3rd and 4th weeks of development leading to fetal loss, still birth or neonatal death. Literature show that causation of anencephaly is multifactorial involving interaction of genetics and the environment though not well characterized. In this case report, we present the features of anencephaly and its different clinical associated malformations.

Methods: A case report study design was employed to explore the prenatal and antenatal events that resulted into an anencephaly delivery. The intra-natal and immediate postnatal fetal observations and outcome were documented. We also documented the care given to the mother throughout ANC, time of delivery and postnatally until discharge.

Results: 29-year G2P1+0 at 29W2D, presented with 2 days' sudden progressive per vaginal bleeding and clear non-foul discharge on 2nd day of admission with no history of abdominal pain, fever or trauma. Two days later she was delivered by Caesarean section to a grossly neural malformed preterm baby boy with APGAR score 6 at 1 minute and 4 at 5 minutes. Birth weight and length were 1.5 kg and 48.6cm respectively. The baby life indicators deteriorated progressively and finally died at 36 minutes after time of delivery.

Conclusion: Although interaction between genomic and environmental factors that play a key role in the causation of anencephaly can not clearly be evaluated, there is an understanding of pre-natal and antenatal factors that predispose to this case such as lack of Folate and or interference with its bioavailability, use of teratogenic drugs taken during pregnancy and antenatal maternal conditions. We therefore recommend routine supply of folate to girls and women intending or risk to conceive 3 months before pregnancy through first trimester and health education about use of native drugs and any other conventional medicines during pregnancy.

Key words: anencephaly, neural tube defects, neuroschisis, congenital malformation, spinal bifida, neurological birth disorders

INTRODUCTION:

Anencephaly is part of neural tube defects (NTDs) spectrum which occur when the neural tube fails to close normally during the 3rd and 4th weeks of development leading to fetal loss, still birth or neonatal deaths.[1] In anencephaly also known as cranioschisis, a major portion of the cranial vault fails to form and the anterior neural pore fails to close. Both the brain and spinal cord may be affected a condition called craniorachischisis. Anencephaly is a condition present at during intrauterine development that affects the embryogenic formation of the brain and skull bones. It results into minimal development of brain tissue. The brain is exposed to amniotic fluid and gradually degenerates. The condition is incompatible with life.[2] The brain lacks part or the entire cerebrum which is an area of the brain responsible for cognitive functions like thinking, vision, hearing, touch, and some movement. There is no bony structure covering over the back of the head and there may also be missing bones around the frontal and lateral parts the head.[3]

According to Best[1] in mediscape and Copp et al,[4] causation of Anencephaly is multifactoral involving interaction of genetics and the environment although neither genes nor environment are well characterized. Some cases may be due to chromosomal abnormality or part of the complex process involving single gene defects or disruption of the amniotic membrane. Although NTDs are multifactoral in modal, rare cases have been found to be autosomal dominant or autosomal recessive in some families. These sources also add that it may be detected prenatally with USS and may be suspected as a result of an elevated maternal serum alpha fetoprotein (MSAFP) considered as a screening test as well.

Folic Acid has been shown to be an efficacious preventive agent that reduces the potential risk of anencephaly and other neural tube defects by approximately two thirds.[5] Reproductive health providers need to have a diverse understanding about causes, presentation and prevention of neurodevelopmental disorders so as to prevent the incidences of such cases. In this case report therefore, we present the features of anencephaly and its different clinical associated malformations.

Pathophysiology.

In a normal human embryo, the neural plate arises approximately 18 days after fertilization a process called neurulation.[5] During the fourth week of development, the embryonic neural plate invaginates along the midline to form a grooved structure called neural groove. The neural groove progresses from midline towards the end directions completing closure at twenty fourth

day for cranial end and 26th day for caudal end (Fig. 1).[6] Disruptions of normal closure gives rise to NTDs. Anencephaly result from failure of the neural tube normal closure at the cranial end of the developing embryo while the absence of the brain and calvaria may be partial or complete[1].

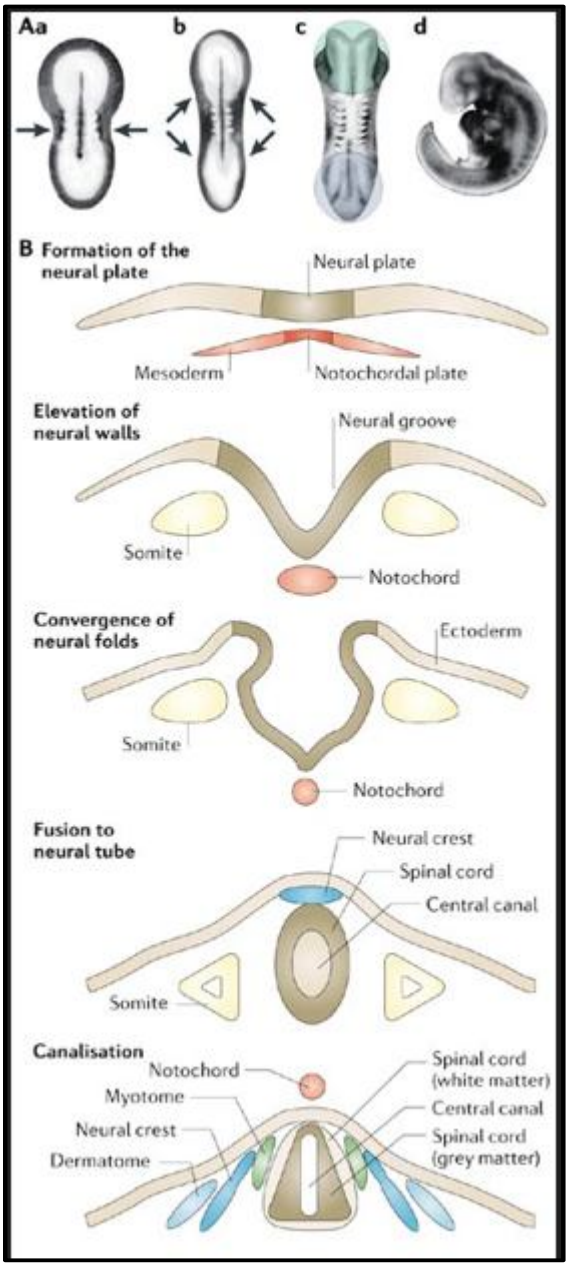

Figure 1: Phases of Neurulation in Neural Tube closure ***(Source: Blom,[6] P.724)***

Though not well understood, genes involved in folate metabolism are implicated in anencephaly (Fig. 2); methylenetetrahydrofolate reductase is associated with neural tube defects and VANGL1, a membrane associated protein is also implicated [1].

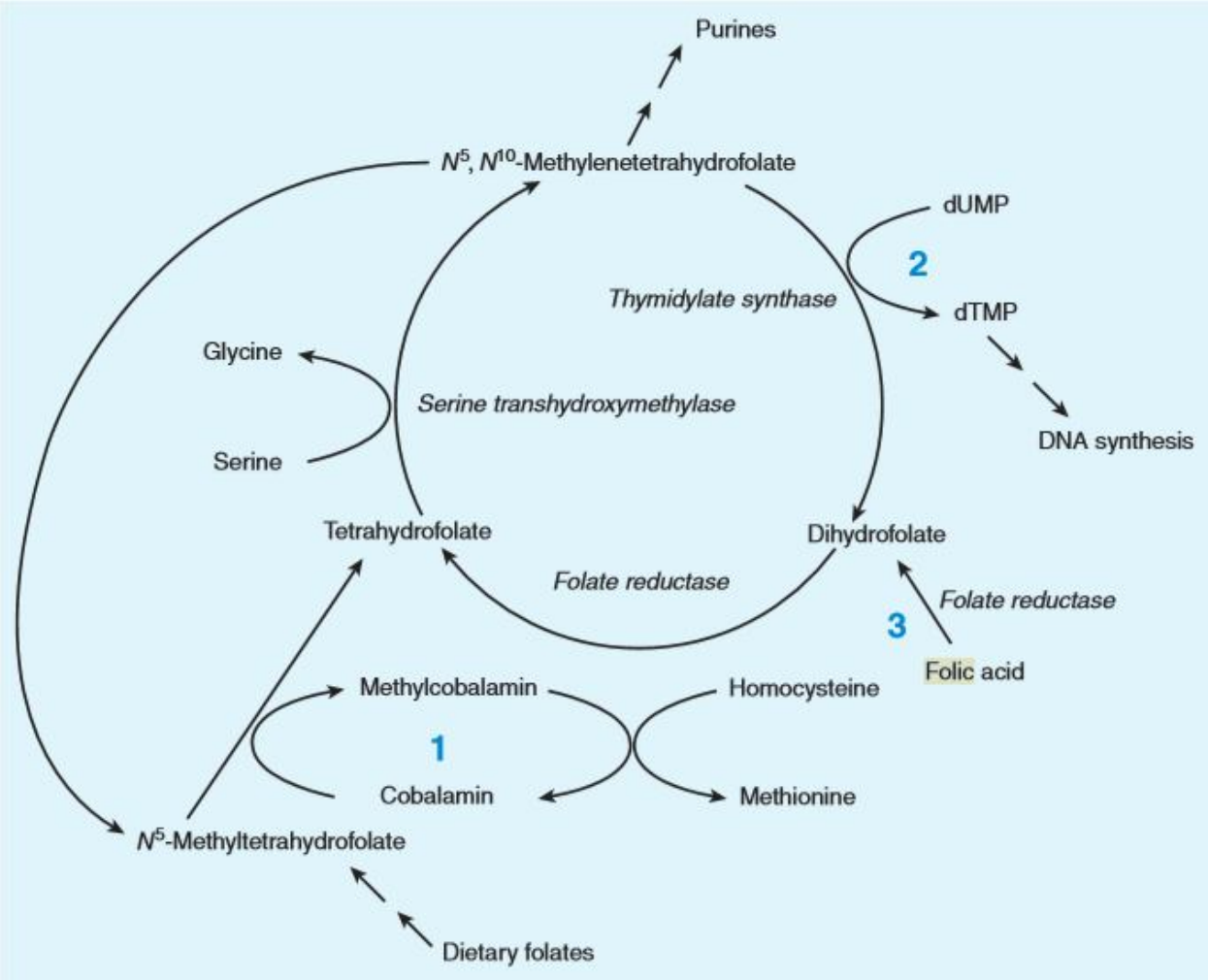

Figure 2: Folate Biometabolism in formation of DNA, (Source: Katzung,[7] p.587)

During pregnancy, human brain and spine begin as a flat plate of cells, which rolls into a tube called neural tube. If all or part of neural tube fails to close, leaving an opening, this is known as an open neural tube defect. This opening may be left exposed or closed with bone or skin. Anencephaly and spina bifida are the two most common neural tube defects.[8,9] An anencephalic infant presents a distinctive classical appearance with a large defect of the calvarium, meninges and scalp associated with an exposed rudimentary brain, which results from failure of closure of the rostral neuropore, which is the opening of the anterior neural tube.

The additional anomalies include; folding of ears, cleft palate, and congenital heart defects in 10% to 20% of the cases. Most anencephalic infants die within a few hours to several days of birth. Approximately 50% of cases of anencephaly have associated polyhydraminous.[10]

Epidemiology of anencephaly.

Geographical variation of NTDs exists with hot spots in Guatemala, Northern china, mexico, parts of UK. Hispanics and non-Hispanic whites are more affected than women of African origin females more affected than males.[1,2]

Anencephaly and spina bifida comprise 95% of neural defects and the remaining 5% is encephalocele. It is common in lower socioeconomic group. Recurrence risk after one affected child is 4% and about 70% of anencephalic fetuses are females mainly born to the very young and old mothers in their 1st birth.[11]

According to Christianson and colleagues in their Dimes Global report on birth defects cited in Mumpe-Mwanja, Uganda didn't have a national birth defects registry and also lacked adequate accurate up-to-date data on prevalence of birth defects. The overall prevalence of birth defects in Kampala, the Capital of Uganda was 66.2/10,000, of which Hypospadias were 23.4/10,000, talipes equinovarus 14/10000, NTDs at 10.3/10000, microcephaly 1.6/10000, microtia and anotia 1.6/10000, and imperforate anus was 2.0/10000.[12]

Linda WXu and colleagues in their study; Neural tube defects in Uganda: A follow up out comes from national referral hospital,[13] revealed that out of a sample size of 230 participants; myelomeningocele accounted to 92%, hydrocephalus and club foot (18%). Anencephaly was not mentioned in the findings.

Frequency during pregnancy is higher than at birth prevalence because such pregnancies end early by spontaneous abortions, fetal death and pregnancy termination [1]. In Uganda cases of neural tube defects are rarely reported, and there is no intentional systems formal recording of such cases most of which go un-noticed.

Frequency of anencephaly during pregnancy is higher than at birth because such pregnancies end early by spontaneous abortions, fetal death and pregnancy termination.[1] The recurrence risk is approximately 4% and increases to 10% if a couple has had 2 previously affected pregnancies. Kliegman,[10] recommends that families with a previously affected child of anencephaly should use folic acid dose 10 times higher than is generally advised for the general population which is 4mg/day compared to 0.4mg mcg/day.

Risks of Neural Tube Defects

Mai,[14] enlist that women with high risk include; women ≥35 years of age, previous pregnancy affected by birth defect, women with chronic diseases (such as; Lupus, high blood pressure, diabetes, epilepsy, and women on certain medications).

Many factors in addition to genetics are implicated as a cause of anencephaly including low socioeconomic status, nutritional and vitamin deficiencies and a large number of environmental and toxic factors.[10]

Investigation of congenital abnormalities prenatally.

Earlier literature denotes that ultra sound scan has been shown to be most commonly used and accurate in detecting neural tube defects as early as first trimester.[10] However, Ultrasound provides little evidence for suspicion of anencephaly in early pregnancy below 10 weeks when Biparietal Diameter cannot be obtained,[15,16] and so MRI is the mainstay imaging study test for neural tube malformation defects.[17]

Ultra Sound Scan done at 12th week of gestation is diagnostic. It also helps evaluate polyhydramnios which is a common feature.[17] This evidence adds that extra fluid behind baby's neck and increased fluid indicates chromosomal disorder or heart defect.

Nancy and mennuti,[18] and Appoline[5] outlined other investigations that could be of benefit though not routinely performed due to limited access that include;

1. Maternal serum alpha fetal protein (MSAFP); effective in 2nd trimester for vast majority of cases with or without history.
2. Amniotic alpha fetoprotein in late 1st and 2nd trimester is diagnostic biochemical marker for anencephaly. False positive is excluded by acetylcholinesterase (AChE) which is positive for open anencephaly. Cytogenic testing excludes trisomy 13. Abnormal levels of alpha fetoprotein may also indicate; multiple fetuses, incorrect fetal dates, chromosomal disorders, brain or spinal cord defects.
3. Chorionic villi sampling, for detecting chromosomal and genetic fetal abnormalities.
4. Amniocentesis for protein levels namely fetal allantoic fluid and acetylcholinesterase
5. Elevated maternal plasma homocysteine.
6. Cell free fetal DNA (cff-DNA) done at 10 weeks of amenorrhea.
7. First trimester screening 11 to 13 weeks of pregnancy,
    a. Looking for defects related to baby's heart or chromosomal disorders like Down syndrome.
    b. Maternal blood screen to measure two proteins; human chorionic gonadotrophin and pregnancy associated plasma protein A (PAPP-A). Abnormally high or low protein levels indicates chromosomal defect or heart defect in the baby.

Some of the possible complications of anencephalic congenital abnormalities;[1]

1. High incidence of polyhydramnios 70%
2. Malpresentation (face or breech).
3. Premature labour.
4. Tendency of post maturity.
5. Shoulder dystocia
6. Obstructed labour if head and shoulders try to engage together because of short neck.

Management

Medscape[1] suggested termination of pregnancy when confirmed in which the couple is counseled.

The uterus is often refractory to oxytocin because of the low levels of estradiol as a result of insufficient production of precursor cortisol from the fetal adrenals. Use of prostaglandin E_2 (PGE_2) vaginal gel has been proved to be effective in resistant cases.

During labor, there is a tendency to delay and shoulder dystocia may be managed by cleidotomy.

Prevention

Pregnancy counseling on causes, prevention, preexisting medical conditions, ongoing medications, drug and substance abuse like smoking and alcohol, and timing of conception. The most important preventive practice is use of folic acid pre- and during early antenatal period.[2,19]

Study Methods

Study design

A case report design was used to retrospectively document the circumstances and conditions of a mother before and during pregnancy, at time of labour and after delivery. Clinical observational findings were recorded and presented according to obstetric case clerkship guidelines.

Study setting

This case report was a mother delivered in Amuria district General Hospital Operation theatre, a rural hospital located in the heart of Amuria Town, Teso sub-region, in far Eastern part of Uganda.

Methods and procedures of data collection

Data was collected through clinical care-based observation and case notes recordings. Retrospective in-depth interviews were conducted with the mother and other care providers to enrich and substantiate delivery findings.

Case presentation

Maternal biodata

A J was a 29-year old Gravida 2 Para 1 + 0 at 29 Weeks 2 Days of amenorrhea was admitted on June 7, 2020. She was an Atesot Catholic married in Komolo village Wera sub-county Amuria district. Her highest level of education was Tertiary institution and working as a secretary at a local police station.

Presenting complaint.

Bleeding per vagina for 2 days.

History of presenting complaint.

AJ was well till 2 days prior to admission when she developed sudden bleeding per vagina which happened while in the garden digging. It was initially mild during the first day occurring in intervals of about every 2 hours and progressively intensified on the second day which prompted her to seek medical attention in nearest health center. The bleed was fresh and bright red with no clots only filling two pads on day 1 and 4 on day 2. This was her index episode of bleeding per vagina since conception, she reported no history of; trauma, abdominal pain, fever and with no history suggestive of significant haemodynamic compromise. While in the hospital she developed per vagina discharge of non-foul-smelling clear fluid on 2nd day of admission. She reported normal bowel and normal micturition habits.

Review of other systems.

Central nervous system

She reported no history of; headache, loss of consciousness, convulsions, impaired vision and hearing.

Cardiovascular and respiratory systems

She reported no history of; cough chest pain, facial and limb oedema, difficulty breathing while lying on flat surface, dizziness, fatigability and breathlessness on exertion.

There was no remarkable history in ear nose and throat and musculoskeletal systems.

Current obstetric history.

AJ was a 29-year G2P1+0, LNMP at 29Weeks and 2Days of amenorrhea who attended first ANC at 20 weeks of gestation in Amuria General Hospital. She had the following tests done; HIV negative, Hb estimation was 10.2g/dl, BP 120/85 mmHg, weight 62 Kg, and height 1500cm. Medications given include; IPT fansidar, iron sulfate and folic acid.

On 5 different occasions, she had a history of lower abdominal pain (LAP), burning pain on micturition, per vagina discharge of white foul-smelling fluid with associated genital itching and abdominal discomfort in suprapubic area. She reported to have been managed for urinary tract infections and experienced temporal relief following completion of dose given treatment. She then used unknown herbal medicines in 2nd trimester for 3 months, and noticed temporary relief after 1 month of use. She also reports being treated with unspecified medication from a local peripheral health facility for genital candidiasis. There was no history of exposure to X-ray and she had never done abdominal Ultrasound scan. She had no history of trauma to the abdomen, febrile illness and no known history of use of neurotoxic agents, folate antimetabolites and no previous history of prior per vagina bleeding during this pregnancy. She reported infrequent episodes of mild vomiting of food eaten in first trimester and resolved without treatment. She fed

on her usual food throughout pregnancy similar to previous pregnancy namely; silver fish, greens vegetables, cabbages, beans, red meat, millet bread and cassava, among others.

There was no history of hypertensive disorders like preeclampsia during previous and present pregnancy.

The father of the current pregnancy was a new sexual partner and had 3 normal children with another sexual Partner.

Past obstetric history

She delivered 1st baby by Spontaneous vaginal delivery at term when she was twenty-four years old, birth weight 2.8kg, male, APGAR score not known but baby didn't cry immediately and was resuscitated. She had four ANC visits, no Abdominal Ultrasound scan was done. She reports no other complications like Post-partum haemorrhage. The child is now 5 years, schooling.

Gynecological history.

She had menarche at 15years, coitarche at 21 years and could not recall exact period of thelarche. She has had 3 sexual partners. No history of sexually transmitted diseases. She had family planning 1 year after delivering her first baby when she received 3 months injections for 4 cycles. She then changed to pills because of excessive bleeding during menses; but she would only take pills when the husband is around. She also reports use of Intra uterine device but didn't specify the timing though she agrees it was after stopping injectable family planning method.

She had never had any gynecological operations and she had never screened for ca cervix.

Past surgical history

She reported no history of surgical operation; no history of trauma / accident and she has never been transfused.

Past medical history

She reports no history chronic illnesses like diabetes, hypertension, asthma, cancer and no H/o drug and food allergies. No history of hospital admissions.

Family and social history

There is no known history of familial illnesses like sickle cell disease, asthma, epilepsy, diabetes, among others. She was the 4th borne to her mother and all siblings were alive with no congenital births among parents and sibling families.

A.J. was in her 2nd marriage to a policeman who had another wife with 3 normal children (no congenital abnormalities). She had history of drinking a ½ glass and or little more of alcohol per day which she had stopped 3 years prior following episodes of vomiting after eating mutton

while taking alcohol. She had never smoked tobacco. The current husband smoked tobacco and took alcohol. She slept in a semi-permanent house under an insecticide treated mosquito net and used borehole water.

Examination Findings

General Examination

Mother was in a fair general condition, not in respiratory distress, afebrile with a temperature of 35.8C, no jaundice, no pallor, no cyanosis, no edema, and no dehydration.

Cardiovascular system

She had a normal BP of 123/85 mmHg, normal pulse of 90 beats per minute and SpO_2 of 95%. Heart sounds 1 and 2 were normal with no added heart sounds and no murmurs, no cardiac rub.

Respiratory system

She was not in distress with respiratory rate of 14 breaths per minute. The chest was of normal shape, symmetrical, moving with respiration and no scars. Non-tender, no palpable masses, normal percussion note in all lung fields. Vesicular breath sounds heard in all lung fields with no added sounds like crepitations.

Urinary system.

There was no renal angle tenderness and the urinary bladder was not palpable, no urethral discharge and lesions in the urethral orifice.

Obstetric Abdomen and Pelvic exam:

The abdomen was grossly distended (Gravid), symmetrical with stria gravidarum and linear Alba. No visible masses, no scars. The abdomen was moving with respiration. Superficially, the abdomen was tensed and non-tender. On deep palpation, the liver, spleen and kidneys were not palpable and non-tender

The obstetric pelvic examination showed:

- Fundal Height = 36/ 40.
- Lie = longitudinal
- Position = Left Occipital Anterior
- Presentation = cephalic
- Absent uterine contractions
- Fetal heart = 120 beats per minute (by fetoscope).

Vaginal Examination

- Vulva and vagina were normal, soiled with blood, vagina was warm and moist.
- Cervix was thick and firm.
- Os was closed.

Other systemic examination findings were unremarkable.

Investigations done

Abdominal Ultra Sound Scan findings revealed a single live fetus with anencephaly at 28 Weeks of amenorrhoea, in longitudinal lie, cephalic presentation. The scan also revealed polyhydramnios with Amniotic Fluid Index of 35 cm and other neuronal fetal anomalies which included; spinal bifida, cleft lip and palate.

The uterine adnexa were normal. The ultra sound scan showed normal findings of; liver, spleen, urinary bladder and kidneys.

Hematological assessments revealed normal CBC parameters, Blood group O Rh D+, negative blood slide for malaria parasites and random blood sugar of 5.8 mmol/L

Urinalysis;

The urine was pale yellow clear in appearance. Negative findings of; proteins, glucose, ketones, bilirubin, urobilinogen, nitrites, blood, and a slightly acidic pH of 6.5.

Microscopic examination of urine revealed 5 epithelial cells per high power field, and no; pus cells, red blood cells, parasites among others.

Peri-natal Diagnosis

A 29-year old G2P1+0 at 29 W2D with multiple congenital fetal abnormalities - anencephaly, spina bifida, cleft lip and palate, under lapping little right toe, short neck and low set ears.

Management plan

Maternal and partner counselling:

1. Causes, management plan and the possible prognosis of this condition. The possible risk factors and prevention were highlighted in the discussion, which included;
    a. minimizing exposure to; x-rays, radiations,
    b. Use of teratogenic drugs especially in first trimester like thalidomide, and there was need to consult a trained health worker before considering use of medications during pregnancy
    c. Infectious agents like viruses.
    d. Missing out ANC early interventions and health education among others.
    e. Inadequate intake of folic acid in first trimester.
    f. Chromosomal abnormalities.

2. Pre-conceptual care and ANC for the next pregnancy to improve the outcome despite documented 4% chance of recurrence.
3. The need to terminate the current pregnancy as the abnormalities were severe and incompatible with life. The termination would be medical induction and cesarean section would be an option in case induction fails.
4. Psychosocial Support. The couple was referred to social worker for psychosocial support. The couple was advised not to blame one partner as cause but consider the occurrence of congenital abnormalities as accidental occurrence and may never occur though there's a possibility of recurrence and can be minimized with medical consultations before and during pregnancy.

Delivery notes

Induction with Misoprostol was attempted according to Uganda MOH clinical guidelines.[20] and there was no progress of labour after two days of monitoring. The decision for caesarean section was then made. The couple was counseled and consented for elective cesarean section. This was done on day 3 of admission under spinal anesthesia by pfannestiel abdominal incision and crescentic lower segment uterine incision. A live syndromic male baby was born, weighing 1.5 Kg, length 48.6 cm and APGAR scores of 6/10 at 1 minute and 4/10 at 5 mutes. Newborn examination was done and revealed multiple physical abnormalities i.e. anencephaly, spine bifida, split face frog-like protruding eyes and cleft pallet (Fig.4), extra digits, plantarflexed under lapping right little toe (Fig. 5), short neck, low set ears and postero-hyperarched back (Fig.6). Excessive clear amniotic fluid was found. No resuscitation was tried due to gross congenital malformations. The baby died 32 minutes after delivery.

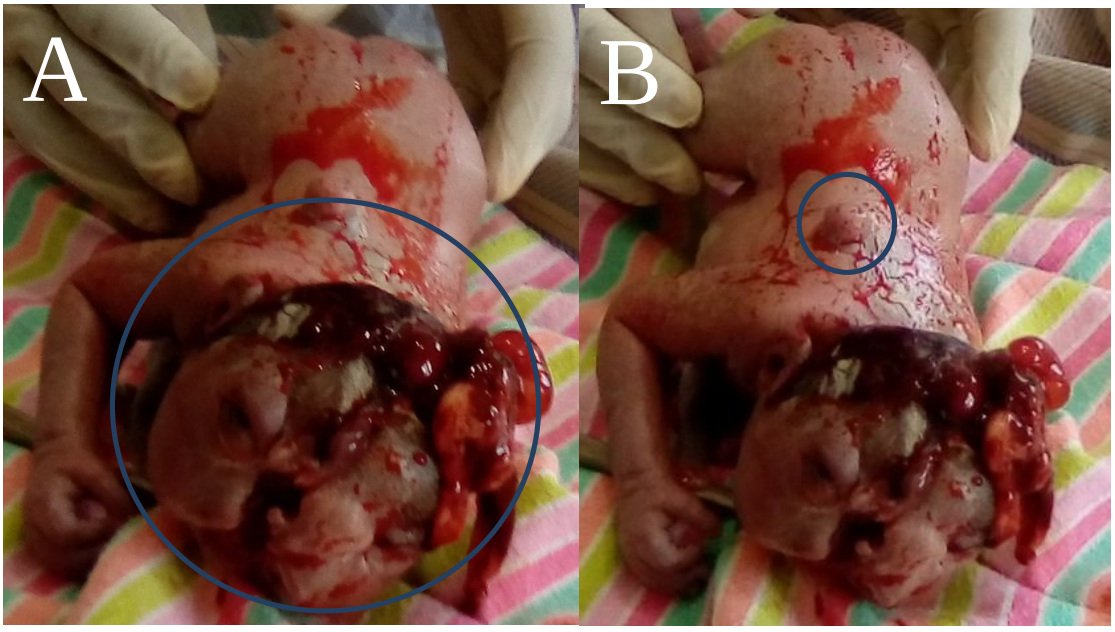

Figure 3: (A) exposed poorly developed brain tissue, absent calvarium and grossly malformed facial structures and (B) back of the baby showing spina bifida (Case photos).

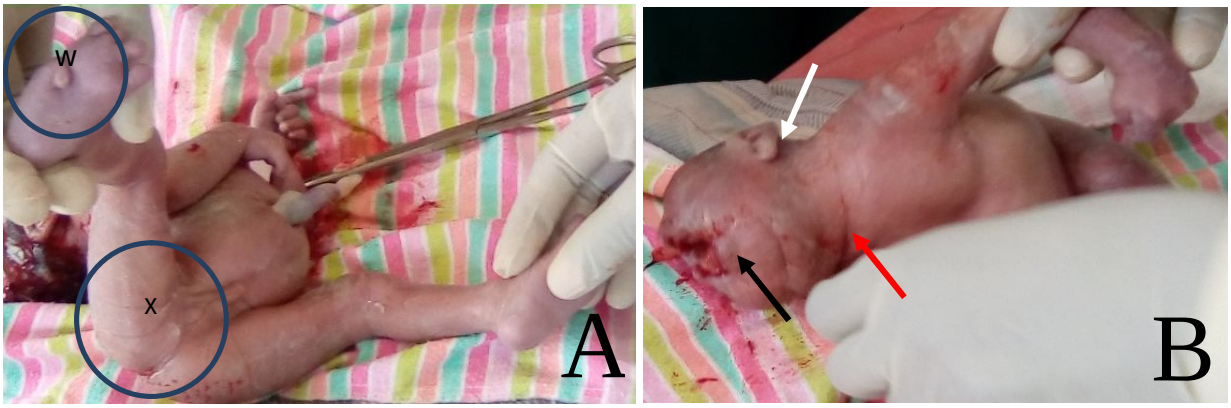

Figure 4: Photo showing (A) right foot with under-lapping poorly formed little toe[w] and normal external ano-genitals[x] (B) short neck and low set ears, cleft lip and palate (Case photos).

Post-operative management and outcomes

The mother was clinical stable after the procedure with;

Mild pain at incision site, no pallor, not in distress, BP 130/85 mmHg, pulse 80beats per minute, RR 14 breath per minute, GCS 15/15 pale yellow urine out put 2500mls in 24 hours.

Treatment given to mother;

Antibiotics; IV ceftriaxone and IV metronidazole; Analgesics; IV paracetamol; IV fluids; normal saline 2 liters, dextrose 5% 1.5 liters in 24 hours; dressing of the incision site with povidone iodine and continuous bladder drainage for 24 hours.

Mother was counseled on family planning at discharge to prevent pregnancy for 2 years to allow scar healing.

Discussion

According to DC Dutta[11] anencephaly is common in lower socioeconomic group. Recurrence risk after one affected child is 4% and about 70% of anencephalic fetuses are females mainly prevalent in 1st birth, young and elderly mothers. The case in question is among the 30% with exceptional a male fetus, in 2nd birth as opposed to 1st birth but within the lower social economic similar to studies elsewhere.

Anencephaly and some other neural tube defects rarely present as an isolation malformation as it was observed for this case. They are always associated with other syndromes and or malformations such as trisomy like down's syndrome, or rarely Meckel Gruber syndrome.[21] and other isolated urogenital, gastrointestinal and or musculoskeletal malformations.[22] In this case, no pathological autopsies were done to extensively evaluate internal malformations.

According to Best In: Medscape[1] points out the risks to anencephaly as; Folate antimetabolites, Maternal diabetes (insulin dependent Diabetes Mellitus), Maternal obesity, Mycotoxins in contaminated corn meal, Arsenic, Hyperthermia in early development. In this scenario, the mother had no history of diabetes, obesity. However, exposure to mycotoxins, arsenic, and folate antimetabolites could not be ruled out.

The mother reported no exposure to x-rays, no history of chronic medication no history of drug abuse during this pregnancy although the husband is a smoker and takes alcohol. She reported no history of syphilis infection. This is parallel to an article; a hospital-based birth defects surveillance system in Kampala, Uganda, which implicated high fertility rate, nutritional deficiencies, recurrent exposures to teratogenic agents, weak regulation of medication, high prevalence of congenital infections to be risks for neural tube defects.[12]

The mother was less than 35-years old and had no previous history of birth defect and chronic illness which are said to be among probable high risks to neural-tubal defects.[14] Conversely, since the mother had not attended ANC, and she had used unknown medicines both conventional from a peripheral clinic and herbal medicines from a alternative and complementary sources, we believe that the possible risk to this anencephaly birth were (1) lack of folic acid in the pre- and antennal period, (2) use of antifolates early pregnancy related infection and (3) poor nutrition.

The mother attended first visit at 20 weeks indicating missing folic acid during first trimester when organogenesis occurs. She would have also benefited from other antenatal services including health education and counselling during the early development. Folic acid has been proved to help prevent neural tube defects.[23] According to CDC,[2] folic acid supplementation beginning 1 month before conception to 12 weeks of pregnancy has shown reduced risk of neural tube defects by 85%. This might have been the main gap in the management of this patient.

Management of neural tube defects with uterotonic agents using oxytocic agents to induce labour is less effective and so Medscape[1] recommends that since the uterus is often refractory to oxytocin because of the low levels of estradiol as a result of insufficient production of precursor cortisol from the fetal adrenals. Use of prostaglandin E_2 vaginal-gel has been proved to be effective in resistant cases. It is not clear as to why Misoprostol given in this case was futile.

Conclusion

Anencephaly and neural tube defects though not very common still do occur and cause a lot of psychological challenge to the parents and caregivers. Although interaction between genetic and environmental factors that play a role in the causation of anencephaly cannot clearly be evaluated, there is a clear understanding of prenatal and antenatal factors that predispose to this case such as lack of Folate and or interference with its bioavailability, use of teratogenic drugs during pregnancy and antenatal maternal conditions.

Recommendations

Since majority of pregnant girls and women begin late to seek ANC we recommend routine supply of folate to girls and women in reproductive age who at any one time miss their expected normal menstruation following a heterosexual penetrative vaginal sexual intercourse until they are proven not pregnant and up-to at least 3 months of amenorrhoea for those pregnant. There is also need for health education about use of native drugs and any other conventional medicines during pregnancy. Amuria Hospital unpublished HMIS reports revealed that the rate of spontaneous abortions in Amuria General Hosptal in the preceding months was 58 in 1st trimester and 6 in 2nd trimester. This is presumably high and yet no studies have been done to rule out neural defect abnormalities which might be the contributing factors. Therefore, there is need to conduct a study to evaluate the magnitude of neural defect abnormalities during early pregnancy because many neural defect pregnancies end up into abortions and premature deliveries.

ACKNOWLEDGEMENTS

We recognize the tremendous effort offered by the entire staff of Amuria general hospital in the management of the patient in question including; Dr. Okwii Nick, Dr. Aisu Emmanuel, Dr. Ojamo Simon, Adonyo Moses the Radiographer FOKA emergency clinic, colleagues on clinical pre-internship placement in Amuria Hospital namely Emaru Job of Busitema university, Oria Tom of Gulu university, as well as the maternity ward midwifery team namely, Apogo Sarah, Apale Esther and Anyiko Christine among others.

Gratitude goes to the mother and the close family relatives for the cooperation, patience and voluntary consent throughout the entire case management and case-write up.

Thanks to Busitema University for the skills and knowledge impacted on me in the 5 years while at school as a medical student.

Competing interests

The principal investigator was a staff at the facility where this case was delivered and managed and at time of management of the case the PI was a 5th year Medical student undertaking his pre-internship clinical placement at Amuria Hospital.

Ethical Approval:

As per international standard or university standard written ethical approval has been collected and preserved by the author(s).

Consent

As per international standard or university standard, patients' written consent has been collected and preserved by the author(s).

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