SOCIETY OF FETAL MEDICINE

Society of Fetal Medicine

NEWSLETTER OF THE DELHI STATE CHAPTER

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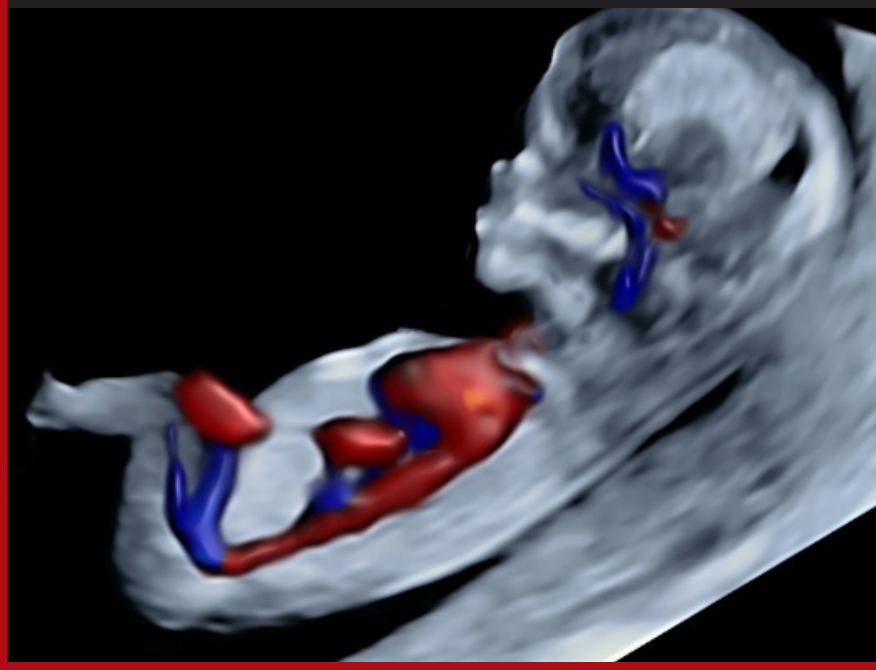

SFM DELHI CHAPTER EXECUTIVE COMMITTEE

Foreword

Dear friends, warm greetings!

It is a great privilege for me to be a part of the revolution created by the Society of Fetal Medicine. As envisaged by the founders, SFM has brought many specialists, clinicians, and researchers from disparate fields to a common platform where the objective is to gather, discuss and dispense knowledge about the rapidly evolving field of fetal care. SFM has transformed the educational landscape of our country with a series of high-quality educational activities, including national conferences, regional webinars, and myriad teaching programs. One of the keys to the success of the Society has been a decentralised approach where regional chapters have been encouraged and nurtured so that knowledge can spread to the far corners of our diverse country. However, despite a gamut of educational activities being hosted in Delhi, the national capital area lacked a formal chapter of its own. Thus came into being the Delhi state chapter.

In the few months since its inception, the chapter has been actively organising continuing professional education programs and collaborating with other professional societies; we have a calendar of events planned for the next two years. We aim to reach out to young students, postgraduates, and practitioners working in the trenches to create awareness about fetal needs and improve practice standards. This newsletter is a continuation of our outreach program to inform the members about the chapter's activities and encourage them to participate in the SFM movement. I hope the readers find the kaleidoscopic array of articles interesting and educative.

I take this opportunity to thank all the executive members of the Delhi state chapter who have collaborated seamlessly in planning and executing the chapter activities. Our indefatigable secretary Dr Sumitra Bachani has been a true driving force and deserves hearty applause. I thank our mentors, Dr Ashok Khurana and Dr Ratna Dua Puri, for encouraging and guiding our fledgling efforts.

Thank you, dear readers, for your precious time.

Dr Kanwal Gujral
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President, Delhi State Chapter, Society of Fetal Medicine

Message from the National President, SFM

Dear friends,

In a modern world, every day brings new advancements. A plethora of academic activities, scientific publications, and new-age communication channels result in near-instantaneous dissemination of information. However, today the challenge for a practitioner is how to integrate the new with the old and how to inculcate the best practices in daily life. A scientific newsletter is an excellent way that ensures the members of a dynamic academic society like SFM get regular scientific updates. It also promotes a wider involvement of the members who wish to share their knowledge and experience to contribute high-quality content.

I am glad that the Delhi state chapter of SFM is publishing a quarterly newsletter. My best wishes to the executive body and the editorial team of the Newsletter, who have done a great job in conceptualizing the journal and collating a wide variety of articles. I hope they will inspire the other chapters of the society to bring out their own publications, thereby establishing a wider communication and broadcasting of information.

I urge the members of the Society of Fetal Medicine to participate wholeheartedly in the activities promoted by the organization. It is the strength of the members which makes the organization a strong, dynamic and cohesive force.

Dr Bimal Sahni
President, Society of Fetal Medicine

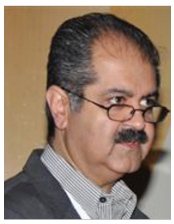
THE NEWSLETTER & THE NEUROENDOCRINOLOGY OF HAPPINESS

The e newsletter of the Delhi Chapter of Society of Fetal Medicine fulfills a long-felt need: a platform for peer communication in a geographically close population. In simple words, we are near and yet so far, we do great work and it goes unrecognized and unacknowledged, and, we need to be in touch with cutting edge practices in our pursuit of offering every fetus an optimal outcome.

I am glad all these needs will now be addressed, and I hope, very substantially. Congratulations to Chanchal, Alok and the entire Delhi Team for this.

Newsletters, and indeed, all forms of communication, lead to a great deal of happiness and well being because the process of writing enhances two of the four Happiness Hormones: Dopamine and Serotonin. Writing gives a sense of personal achievement and a sense of doing something for others, and this leads to higher levels of these neuroendocrine molecules. All we need to achieve healthy levels of all four hormones is exercise for Endorphins and on-site meetings for hugs and Oxytocin!

Keep writing. Keep reading. Keep up relationships. Best wishes.



Ashok Khurana
Mentor Emeritus, Society of Fetal Medicine

From the Patron's Pen

Greetings and warm wishes to all readers

The Society of Fetal medicine has grown in leaps and bounds over the last years. Most of you have witnessed this amazing pace of including many thousands within its fold. There was a felt need for specialists in the multidisciplinary field of fetal medicine to connect and share experiences within the landscape of their workspace. This brought forward the concept of a local chapter – a platform for sharing information, experiences, and striving for excellence. Members engaged in bringing forth academia as a newsletter, and therein is the first newsletter of the ***Delhi Chapter*** of the Society of Fetal Medicine.

I feel honored to be connected as a part of this change in the care of the unborn fetus. The advances in imaging, fetal diagnosis and management have been rapid, and with novel improved technologies, we will witness further changes in the years ahead. With this focus, through the newsletter, we envision creating a forum enunciating best practices and enhancing education through the quantum of excellence in work performance. A quarterly theme for discussion can be a vision that will include uniform standards for diagnosis, management, and counselling. We hope that in times ahead, we can plan multicenter collaborative projects to collect the wealth of data available, which is always the most significant source of learning.

This is the inaugural issue of the newsletter, and I urge the readers to stay engaged; we will be privileged with your attention and participation. As a geneticist, I realize the rapid pace of changing technologies and enhanced versions of published guidelines. This is the space where we will learn advances through sharing our learnings and experiences to benefit each other to stay updated.

Dr Ratna Dua Puri
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From the Secretary's desk

Dear Friends,

"The magic in new beginnings is truly the most powerful of them all."

~ Josiyah Martin

Many congratulations and three cheers for our very own Delhi chapter of SFM!

It is my proud privilege and honour to be associated with the first executive committee of the Delhi chapter of the Society of Fetal Medicine. This vibrant chapter includes many luminaries and passionate members encompassing varied specialities. We hope to bring you many new ventures, insightful discussions and debates to keep us all abreast with the times. We seek blessings from our Patrons, Dr. Ashok Khurana and Dr Ratna Dua Puri, in our academic endeavours.

As one of the first steps to reach our members, we have brought out this Newsletter with varied features, painstakingly put together by Dr Alok Varshney and Dr Chanchal. I am sure you will enjoy the unique articles and crossword.

I am looking forward to your valuable suggestions, comments and feedback. Do write to us at delhichaptersfm@gmail.com.

Happy reading and Quizzing. Stay Safe.

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ULTRASOUND IN RECURRENT PREGNANCY LOSS

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Recurrent pregnancy loss (RPL) is defined as two or more consecutive failed pregnancies documented by ultrasound or histopathology.[1] Most studies report that RPL affects about 1–2% of women. About 50 percent of women with RPL have no clearly defined aetiology.[2] According to ESHRE guidelines, age is a key factor, and RPL is more common in women above 40 years of age.[3] The aetiology of recurrent pregnancy loss (RPL) is classified into:[1]

1. Genetic
2. Anatomic
3. Endocrine
4. Antiphospholipid antibody syndrome
5. Immunological
6. Environmental factors

ULTRASOUND PARAMETERS TO DEFINE EARLY PREGNANCY FAILURE [4]

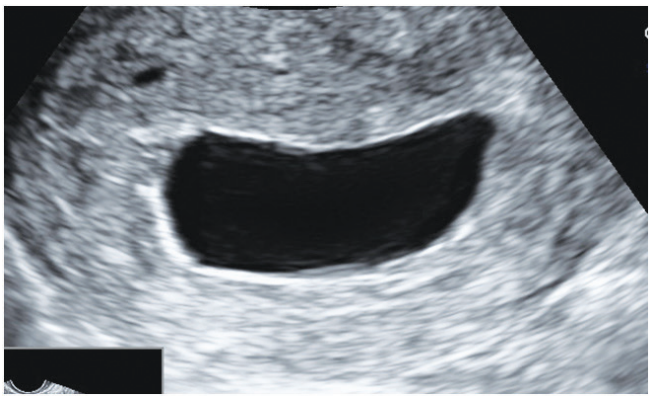

Figure 1: Transvaginal scan (TVS) image showing irregular gestation sac of 20mm& no yolk sac or embryo in a woman with eight weeks of amenorrhea.

1. Mean Gestation sac diameter (MGSD) of 20 mm & no yolk sac
2. MGSD of >25 mm & no embryo
3. Irregular gestational sac
4. Distended yolk sac > 7mm
5. Collapsed & echogenic yolk sac
6. Crown-rump length of 7 mm or more & no cardiac pulsations
7. Loss of a previously seen cardiac activity. Cardiac activity less than 85 beats per minute indicates poor prognosis
8. The MGSD-CRL ratio less than 5 mm are prone to first trimester miscarriage

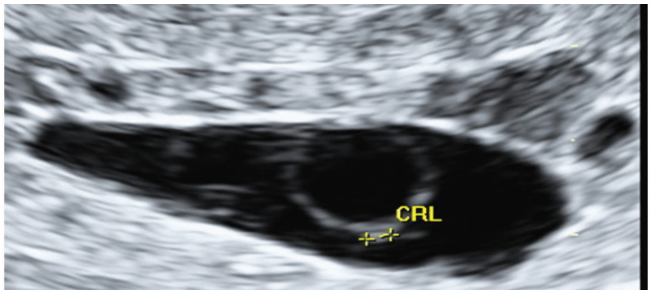

Figure 2: TVS image showing distended yolk sac of 5mm, embryo of 1.1mm CRL & no cardiac activity in a 39-year-old woman with 10 weeks amenorrhea

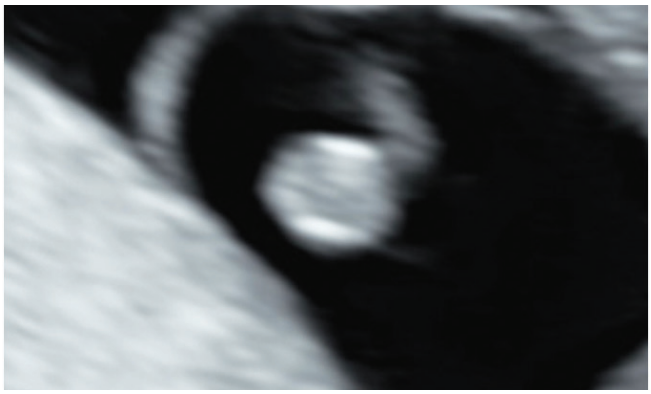

Figure 3: 2D USG image showing echogenic yolk sac& embryo with no cardiac activity

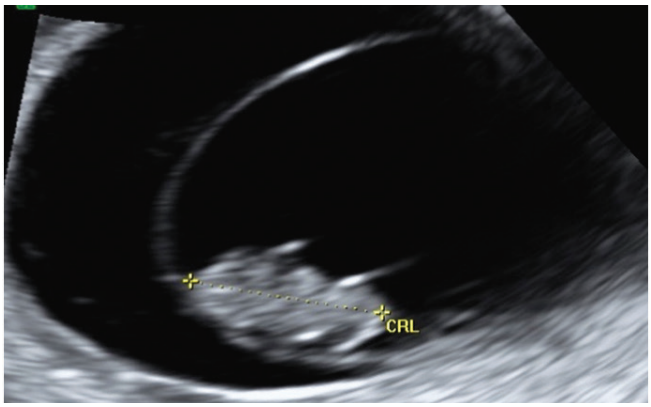

Figure 4: 2D USG image showing embryo of 13mm CRL correlating to 7 weeks 4 days with no cardiac activity in a 42-year-old with 11 week amenorrhea

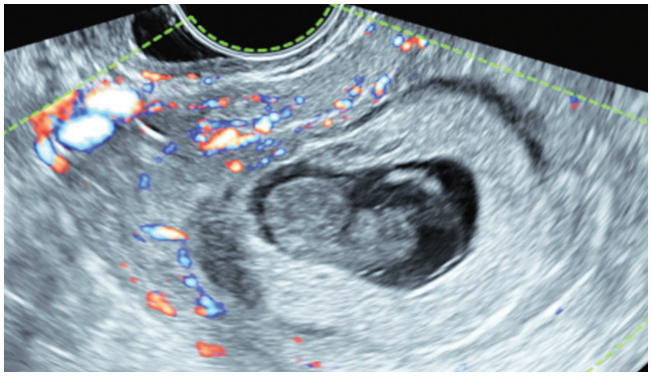

Figure 5: TVS showing sac volume smaller than the embryo at 8 weeks amenorrhea

ANATOMIC/STRUCTURAL ANOMALIES

Anatomic/ Structural anomalies increase women's risk and may account for 10–50% of RPL.[1] These include:

1. Congenital Mullerian Duct Anomalies
2. Acquired abnormalities like fibroids, polyps and synechiae (Asherman syndrome)

Müllerian Duct Anomalies (MDA)

Prevalence of MDA in RPL is about 7% to 28%.[5] Accurate MDA recognition & their classification according to European Society of Human Reproduction and Embryology (ESHRE)[6], The American Society of Reproductive Medicine (ASRM)[7] or Congenital Uterine Malformation by Experts (CUME)[8] is essential for effective & efficient therapeutic planning to improve reproductive outcome.

Septate uterus is most commonly associated with first trimester spontaneous abortion.[1] This may be from abnormal endometrium & septal vascularity. Septum is the tissue resulting in an internal indentation at the fundal midline exceeding 50% of the uterine wall thickness.[6]

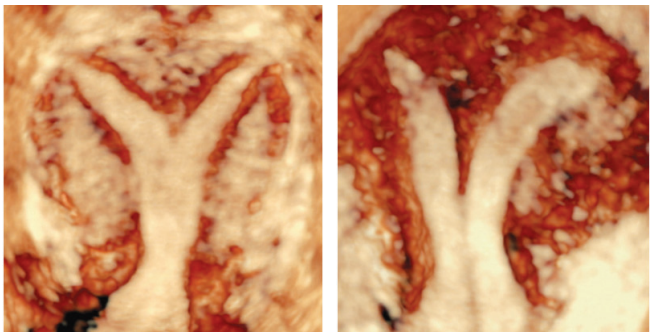

Figure 6: 3DCoronal rendered images of uterus showing normal fundal contour but internal indentation dividing endometrial cavity into two. A.Partial septate: septum above the internal osB.Complete septate: Septum extends into cervix

Bicorporeal/bicornuate uterus has a higher prevalence of cervical incompetence.[9] There is an abnormal external fundal contour; internal mid line indentation divides the cavity into two endometrial cavities and cervices.

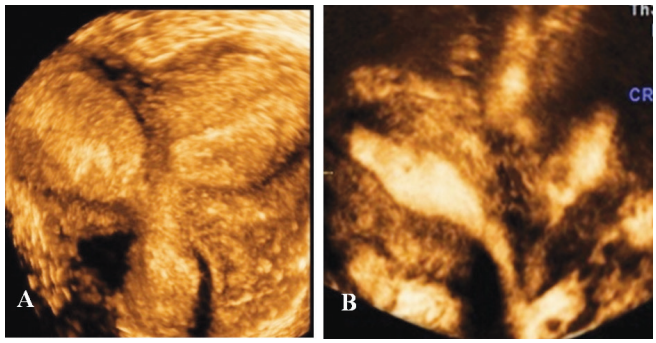

Figure 7: Coronal images of Bi-corporeal uterus (ESHRE classification) with abnormal external fundal contour &internal indentation.
The American Society of Reproductive Medicine (ASRM) classifies these as A: Bicornuate & B: Uterine didelphys

Hemi-uterus/ Unicornuate uterus has an abnormal configuration. It is seen as a small, curved uterus which is located off-midline with a single endometrial cavity & cervix.[7]

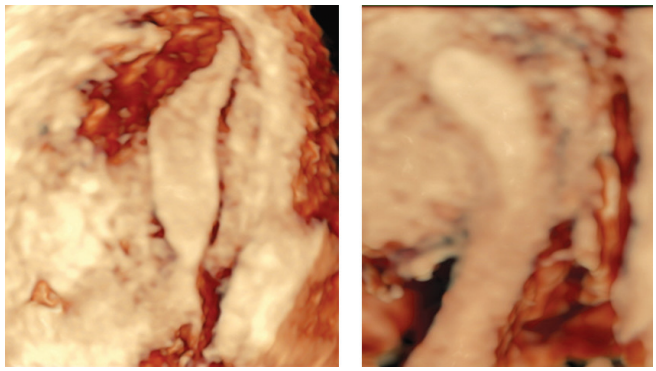

Figure 8 : Coronal images of right & left hemi-uterus

Acquired anatomical factors:

These include Uterine fibroids, endometrial polyps and uterine synechiae, which may be associated with RPL.[1]

Uterine fibroids are reported in 8.2% of women with RPL.[10] Ultrasound evaluation of the uterine cavity and classifying the fibroids according to their growth into the endometrium (Fibroid mapping) are important in the diagnosis & management of RPL.[11] Submucosal fibroids deform the endometrial cavity and may affect implantation & embryonic development.

Removal of submucosal fibroids reduces the chance of miscarriage. Intra mural fibroids do not distort the uterine cavity & there is no evidence

that myomectomy may reduce the chances of an abortion.[10]

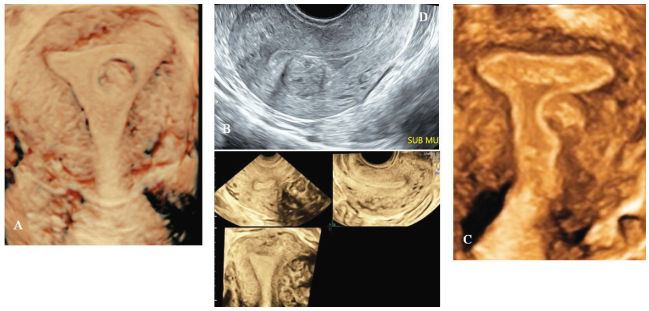

Figure 9: The International Federation of Gynecology and Obstetrics (FIGO) classification of Submucous uterine fibroids. A. Completely Intracavitary (Type 0) B. > 50% Intracavitary (Type 1) C. <50% Intracavitary (Type 2) D. Completely Intramural (type 4)

Endometrial polyps are benign nodular protrusions of the endometrial surface. These have a higher prevalence in women with pregnancy loss.[1]

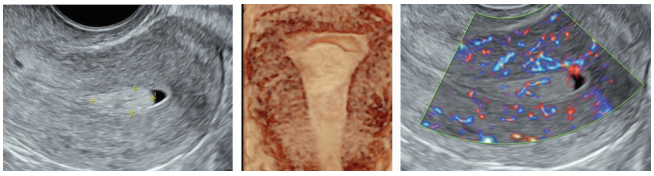

Figure 10: TVS & 3D rendered images of a homogenous echogenic polyp completely within the endometrial cavity & showing a vascular pedicle on color flow.

Uterine synechiae are seen as thin or thick strands of tissue crossing the endometrium. These are better seen when there is fluid in the endometrial cavity. Women with RPL are more likely to have uterine synechiae because of curettage; the prevalence ranges from 0.5 to 28%.[12] The probable pathophysiology in RPL is due to an abnormal functional endometrium which may interfere with the invasion and normal development of the placenta.

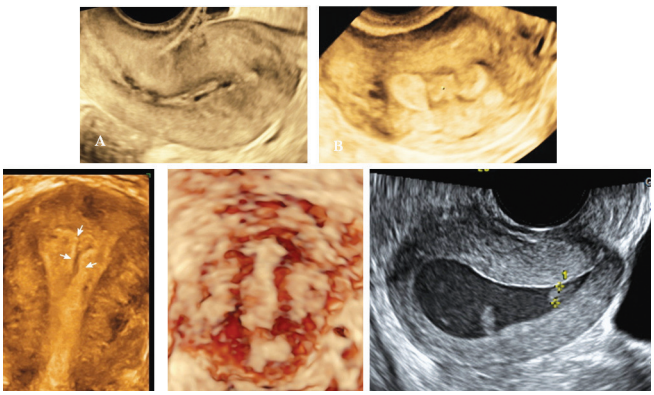

Fig 11 : Uterine Synechiae A: TVS: thin endometrium &linear hypoechoic scar.

B. Thick bands distorting endometrium C. Mild adhesions D. Extensive Dense adhesions completely distorting the endometrial cavity. E. Synechiae seen better with fluid in the endometrial cavity

Chronic endometritis refers to inflammation or infection involving the endometrium. This has a prevalence of 12-13% in RPL.[13]

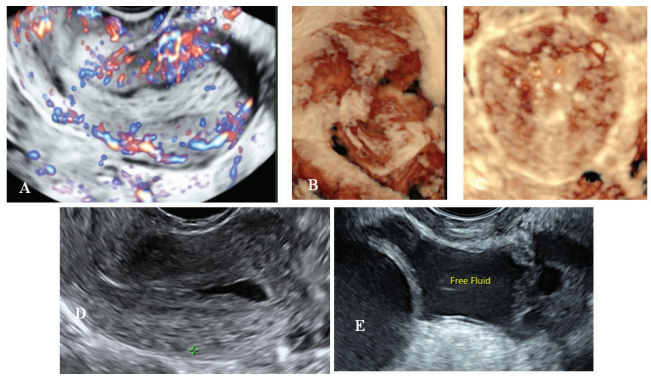

Figure 12: A: Thick, heterogeneous endometrium; increased vascularity on color flow B: Loss of normal myometrial endometrial interface C: Intra uterine air D: Intracavitary fluid E: Fluid in the pouch of Douglas

CERVICAL INCOMPETENCE

Cervical incompetence is painless cervical dilatation in the absence of uterine contractions due to a functional or structural defect resulting in a second-trimester miscarriage. This may be due to embryological maldevelopment of the Müllerian ducts or acquired from cervical trauma. TVS is modality of choice for assessment.[1]

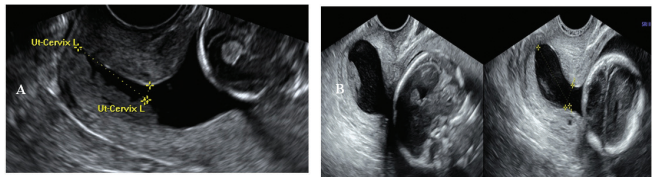

Figure 13: Cervical incompetence
A. Short cervical length < 25mm and widening of internal os
B. Complete bulging giving an hour glassappearance.

CONCLUSION

Ultrasonography has a significant role in detecting abnormal uterine morphology as a cause for RPL.

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FETAL MICROGNATHIA CENTRAL DIAGNOSTICS ONWARDS

Alok Varshney
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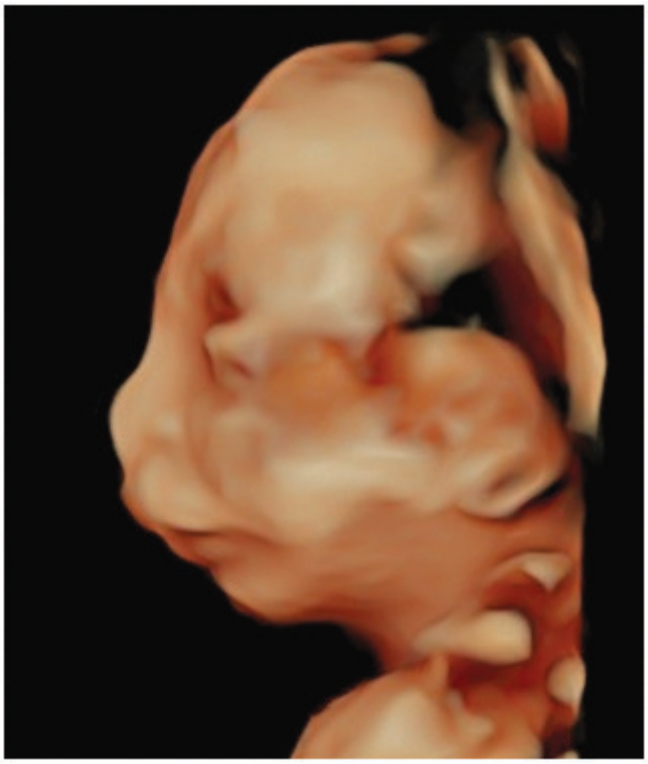
MICROGNATHIA is a relatively common anomaly with a reported incidence of 1 in 1,500 births.[1] Abnormal or arrested development of the fetal mandible due to genetic or environmental factors results in its abnormal size and shape. This may lead to micrognathia (short mandible) or retrognathia (posterior displacement of the mandible in relation to the maxilla), although these two anomalies are commonly concurrent.[2]



PRENATAL DIAGNOSIS is usually made subjectively on the mid-sagittal view of the fetal face. A facial profile with a prominent upper lip and receding chin is characteristic of this anomaly. Objective diagnosis is made by using **biometric techniques,** which may be helpful in diagnosing less severe cases. These include:

Inferior facial angle is the angle formed in a mid-sagittal facial profile view by a line drawn perpendicular to the forehead at the level of the nasal bridge and another line through the tip of the chin and the more protrusive lip, usually the upper lip (Fig1). Normal value is 65°±16°. Micrognathia is diagnosed when the angle is less than 49°.[3]

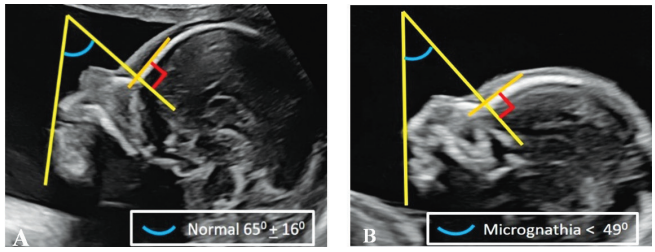


Figure 1: Inferior Facial Angle in a normal fetus (a) and an abnormal fetus (b)

Jaw Index is calculated in the axial plane by measuring the anteroposterior diameter of the fetal mandible from the symphysis menti to a line joining the bases of the two mandibular rami and expressing it as a percentage of the biparietal diameter (anteroposterior mandibular diameter / BPD x 100) (Fig.2). This provides an index that is independent of gestational age.[3] A value less than 23 suggests micrognathia.[2]

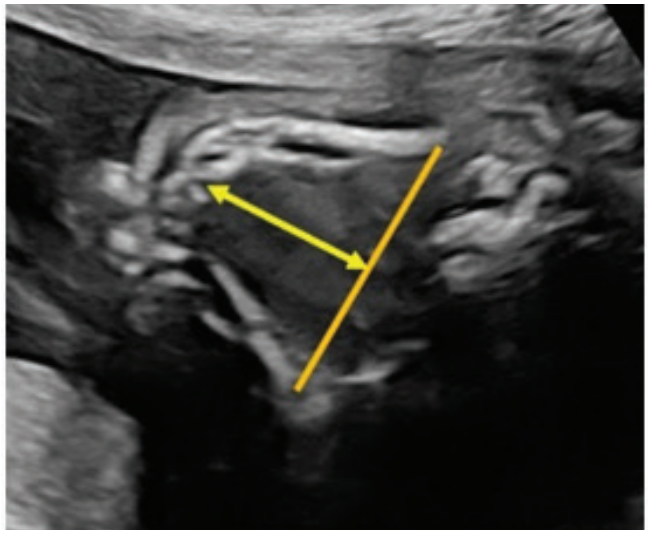


Figure 2: Jaw Index

Frontal nasomental angle is the angle between a line drawn from the tip of the nose and frontal bone and another line from the nasal tip to the mentum (Fig. 3). Its normal mean value is 147°±5°. An angle less than 142° is consistent with the diagnosis of micrognathia. However,

since many normal fetuses may have a low frontal nasomental angle, a more specific cut-off is 134°.[3]

Mandible width/maxilla width ratio: Axial sections of the mandible and the alveolar process of the maxilla are taken. The width of the mandible and maxilla are measured at the level 10 mm from the anterior osseous border (Fig. 4). This ratio is found to be constant over the second trimester. The mean value of this ratio is 1.02 ± 0.12 (SD). A ratio less than 0.78 (below the 5th centile) is used to define micrognathia.[4]

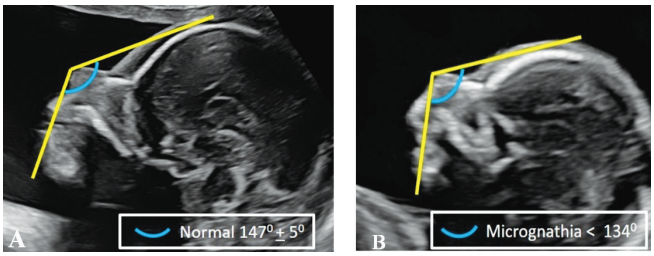


Figure 3: Frontal nasomental angle in a normal fetus (A) and an abnormal fetus (B)

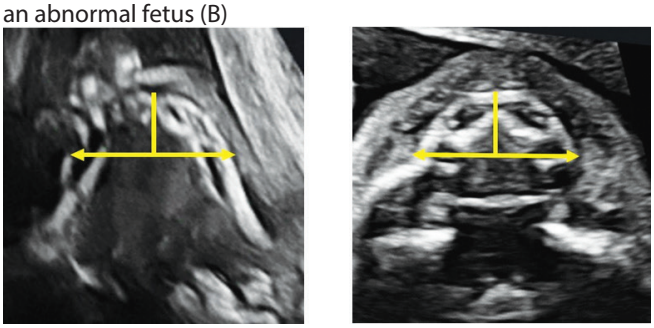


Figure 4: Mandible width (a) and Maxillary width (b) in a normal fetus

FURTHER INVESTIGATIONS

Detailed ultrasound examination to look for other anomalies.

Micrognathia is rarely isolated; presence of other anomalies suggests syndromic association. **Invasive testing for karyotyping/ Microarray** should be offered even in isolated cases. In many cases of seemingly isolated prenatal micrognathia an underlying syndromic cause is found after birth.[2]

SOME COMMON ASSOCIATED SYNDROMES

Pierre-Robin syndrome: Micrognathia + Glossoptosis + cleft palate. No other anomalies.

Nager syndrome: Micrognathia + ectrodactyly + mesomelic limb shortening + ear anomalies.

Treacher-Collins Syndrome: Micrognathia + external ear anomalies + large mouth (macrostomia) + downslanting palpebral fissures.

Campomelic dysplasia: Micrognathia + bowed bones.

FADS: Micrognathia + joint contractures + thoracic hypoplasia + club feet + reduced movements.

Neu-Laxova syndrome: Micrognathia + severe FGR + microcephaly + short limbs + diffuse edema + joint contractures.

FOLLOW UP

Ultrasound scans every 4 weeks.

DELIVERY

At 38 weeks at a hospital with facilities for neonatal intensive care.

Method: Induction of labor aiming for vaginal delivery.

A pediatrician should be present in the delivery room and be prepared to intubate the neonate.

PROGNOSIS

Neonatal mortality: >80% due to associated abnormalities.

In Pierre–Robin anomalad survival is good.

RECURRENCE

Isolated: No increased risk of recurrence. Part of trisomies: 1%.

Part of genetic syndromes: 25% to 50%.

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False positive NIPS- Maternal aneuploidies and need for invasive testing

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Non-invasive prenatal testing has been a revolution in the field of prenatal diagnosis. Over the years NIPS has been integrated into routine clinical practice. NIPS is a high sensitivity and specificity prenatal screening test for trisomy 21, 18, and 13.[1] Due to the genome-wide properties of NIPS, the scope of screening was widened to include sex chromosome aneuploidies, autosomal trisomies, and sub-microscopic copy number variants. These still remain to be universally validated.[1,2]

Even with the increasing sensitivity and specificity of NIPS, it is still an advanced screening method at best. The results can be discordant from the amniotic fluid culture analysis. The discordant NIPS results can be attributed to several factors. Hartwig et al. clarified that confined placental mosaicism, maternal copy number variations (CNVs), maternal aneuploidies, maternal malignancy, vanishing twin, and technical, bioinformatics, or human errors were found to be reasons for discordance between NIPS results and fetal karyotype (3). Accordingly, a positive NIPS result should always be confirmed by an invasive test like amniocentesis (4).

A 33-year-old primigravida was referred for a genetic counseling in view of her non invasive prenatal screening results showing a positive screen for Monosomy X (Turner syndrome). This pregnancy was conceived by in-vitro fertilization using a donor egg. The artificial reproductive technology was used in view premature ovarian failure (AMH<0.1) On detailed history taking and examination it was found that the woman had short stature, with a short neck and increased carrying angle. It was suspected that the woman herself has a mosaic form of Turner syndrome which may be causative of the positive NIPS report. Amniocentesis was recommended for confirmation of the ploidy of the fetus. It showed a normal autosomal as well as sex chromosomal complement in the fetus. Karyotype of the woman revealed 45,X (figure 1).

The circulating cell free fetal DNA (cffDNA) in maternal plasma is a combination of maternal and fetal DNA, in which the fetal fraction is only 3-10%. Therefore, abnormal maternal chromosome complements may lead to discordant NIPS results. Maternal aneuploidies or low level maternal mosaicism result in variations of the maternal contribution to circulating DNA, which could impact NIPS results. This case brings out the need of detailed clinical evaluation while analysing any NIPS report and a mandatory invasive testing after positive NIPS.

Figure 1- Karyotype of the pregnant woman, 45,XO

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Fetal Skeletal dysplasia: the long and short of long bones

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Introduction:

Skeletal dysplasias (SD) or osteochondrodysplasias are a heterogeneous group of rare genetic diseases that affect the growth and development of bone and cartilage.[1,2,3]

As per recent data review, birth prevalence is estimated as 2.1 to 4.7/10,000.[4-9] Duarte et al. studied 1,663,610 births from 160 hospitals, and estimated the prevalence of osteochondrodysplasia in Argentina as 2.20 per 10,000 births [10]. Barbosa-Buck et al. reported the birth prevalence as about 3.2 per 10,000 in in South America[11].

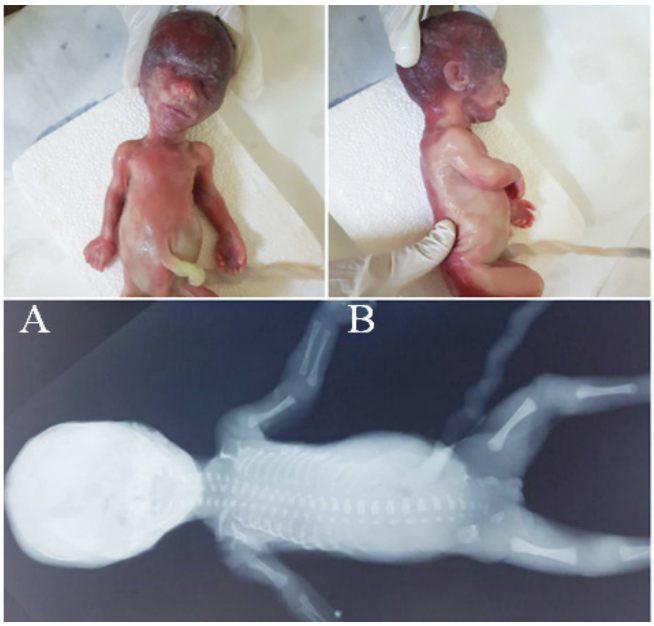

Fig. 1 : Asphyxiating Thoracic Dystrophy
A&B- Fetus with a narrow thorax and short limbs. C- X-ray showing handle bar clavicle, narrow thorax and squared ilium.

Indian statistics on the prevalence of skeletal dysplasia are scant. A hospital based prospective study from Davangere estimated the incidence of SD as 19.6/10,000 births and lethal dysplasias as 5.2/10,000 births [12]. In a recent study by Nampoothri et al, common lethal skeletal dysplasias in Indian population are OI Type 2, Thanatophoric dysplasia I, Hypochondrogenesis, Spondylocostal dysostosis, Achondrogenesis type 2, Achondrogenesis I , Campomelic dysplasia, and Roberts syndrome. Nonlethal skeletal dysplasias commonly described in this study are Achondroplasia, Brachytelephalangic chondrodysplasia punctata, Asphyxiating thoracic dystrophy, Kniest syndrome , MPS 3 A, GM1 gangliosidosis, and Mucolipidosis type 2.[13]

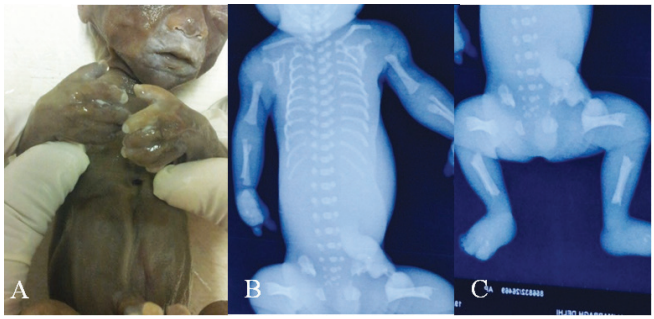

Fig. 2: Short Rib Polydactyly type 3
A: Fetus after termination showing polydactyly. B and C: X-Ray of the fetus showing short long bones, narrow thorax and metaphyseal spur

Currently, the genetic skeletal disorders classifications are based on combination of skeletal dysplasias and dysostoses, and include 461 different disorders classified into 42 nosology groups. This newest and tenth version of the Nosology is based on their clinical, radiographic, and/or molecular phenotypes. Pathogenic variants affecting 437 different genes have been found in 425/461 (92%) of these disorders.[14]

Fetal skeletal dysplasia has a prevalence of 2.4–4.5 of 10,000 births[6,18-20]. At present, the prenatal diagnosis of fetal skeletal dysplasia mostly relies on ultrasound, X-ray and magnetic resonance imaging. However, ultrasound cannot differentiate among the different types of skeletal dysplasias in 40-50% cases. [21-23]

Short long bones is a very common presentation on antenatal ultrasound. The various causes of short long bones would include maternal causes or fetal disorders. Among maternal causes uteroplacental insufficiency is commonly cited as

one of the causes or it can be constitutional. Fetal causes comprise FGR, aneuploidy, infections such as CMV, skeletal dysplasia or genetic syndromes such as primordial dwarfs or chromosomal breakage syndromes. Differentiating constitutional, aneuploidy and skeletal dysplasia clinically is most relevant clinically so as to prognosticate the outcomes. In cases with constitutional shortness, the long bones would be at around 5th centile. In FGR due to aneuploidy, long bones are short -2 SD/-3 SD but BPD, HC and AC all would be small and this will be supported by presence of oligohydramnios, soft markers or abnormal maternal serum screening and doing microarray testing will be most appropriate in these settings. Diagnosis of skeletal dysplasia requires objectively assessing the shortness and is suspected if long bones are at -3 SD/-4 SD particularly in the presence of normal BPD/HC. In cases of suspected skeletal dysplasia, other objective criteria are important so as to detect lethal skeletal dysplasia.

1. Narrow thorax <5th centile
2. Femur / Foot ratio <1
3. TC/AC <1

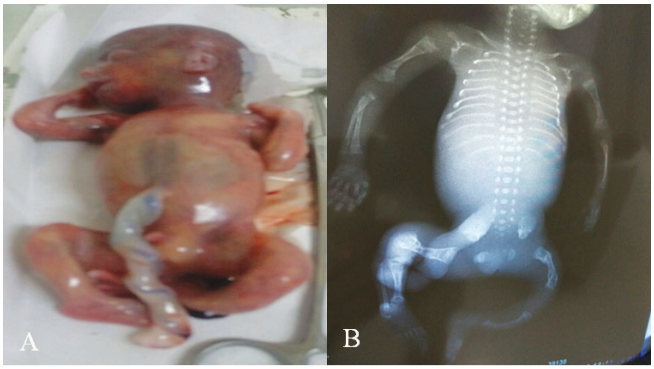

Fig. 3: Osteogenesis Imperfecta
A- Fetus with bowed and short femur B: X ray findings:
Skull bones-Decreased mineralization; Long bones - bent broad and small, metaphyseal widening, reduced bone density; Chest- narrow thorax, thin and beaded ribs; Spine - Platyspondyly

Once clinical diagnosis of skeletal dysplasia is established its important to subclassify further into different types. Five common skeletal dysplasia in north India include- OI, thanatophoric dysplasia, Short rib polydactyly, Asphyxiating thoracic dystrophy and achondroplasia. (Fig1-Fig 5). Presence of bowing of long bones/ fractures or defective mineralization should be looked for to detect osteogenesis imperfecta. The diagnosis of OI can be made by postnatal X-ray on the basis of wide bones with defective mineralization. Thanatophoric dysplasia (TD) is suspected in cases of bowed femur as telephone receiver type on postnatal X-ray and histopathology of bones suggestive of TD. SRP/ATD are distinguished by X-ray findings postnatally and molecular testing will confirm the diagnosis. In cases with achondroplasia shortening of long bones is noted after 20 weeks and usually after 25 weeks. Molecular diagnosis for common FGFR3 mutation will confirm the diagnosis of achondroplasia.

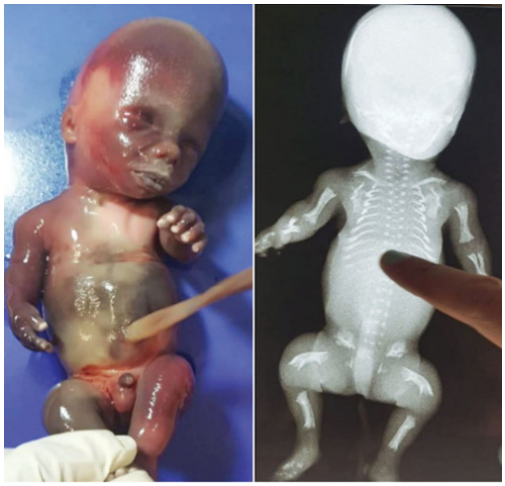

Fig. 4: Thanatophoric Dysplasia
A- Fetus after termination showing polydactyly
B- X-Ray of Fetus showing rhizomelic shortening of the long bones, bilateral short and bowed femora(telephone receiver type), platyspondyly and narrow thorax

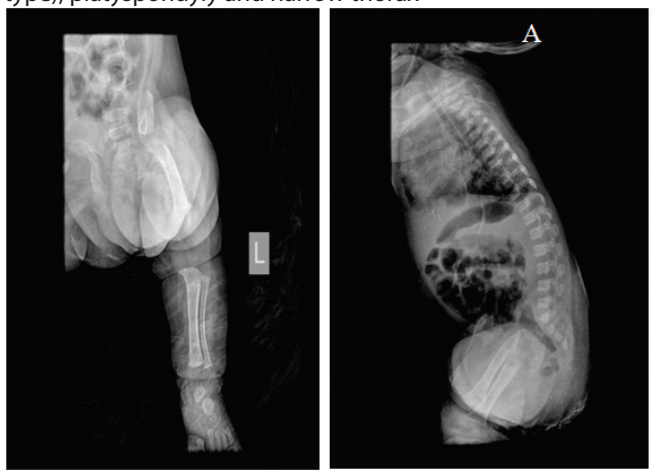

Fig. 5: A-Short long bones and sloping metaphyses
B- Gradual narrowing of interpeduncular distance

To conclude, etiology of short long bones is heterogenous and a systematic approach by clinical, radiological and molecular testing will

lead to correct diagnosis. Making a correct diagnosis is essential for genetic counselling and accurate prenatal diagnosis.

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STATISTICS

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Why do we need statistical literacy?

If we don't understand statistics how are we going to explain numbers to the patients?

What about **informed consent** and **informed decision**?

Understanding results in confident advising of tests, better communication which translates into less headache, guilt and lesser number of medicolegal issues.

Good communication is highly dependent on statistical literacy.

Data are numbers with a context but has no meaning by itself.

Statistics is the science of learning from data by organizing and interpretation.

Applications:

- It allows to measure & control uncertainty.
- Objective tool to assess the results
- Support the conclusions

Statistical techniques are broadly grouped in two categories:

-descriptive

-inferential

Descriptive: raw data is **organised** to create / discover **patterns** and understand better. **These include:**

• Graphs, frequency tables, charts etc to **display data** so that it is easier to understand.

• Measures of central tendency

• Measures of dispersion

Inferential: when decisions are made on the basis of organised data.

STATISTICAL GLOSSARY USED IN DAY-TO-DAY PRACTICE:

Percent: one part in every hundred.

Percentile: measure used in statistics indicating the **value below which a given percentage of observations** in a group of observations falls.

Applications: Fetal biometry including weight to assess growth in comparison to national / population averages. **Percentiles are found in growth charts**, e.g. the 20th percentile is the value below which 20% of the observations may be found

- fetal weight below 3rd percentile is treated as abnormally low.
- Amniotic fluid above 95th percentile is treated as abnormally high.

Quartile: type of quantile which divides the number of data points into four more or less equal parts-quarters.

Applications: used in defining interval growth of fetal weight. **Loss of a quartile of fetal weight is also labelled fetal growth restriction (FGR) even if it is within the normal range.**

For example, if fetal weight percentile was on the 70th percentile to start with and reduced to 45th percentile at follow-up examination, it is considered suboptimal growth, even though the latter cut-off still lies within the normal range.

Screening: a systematic attempt to find from an apparently healthy population, people at high risk of a specific condition to warrant further action. Most screening tests serve only to mark the condition and *are not diagnostic.*

Screen positives: are offered further work up which is impossible to offer universally due to limited financial resources, harmful effects or both.

Sensitivity/detection rate: refers to the test's ability to correctly detect ill patients.

Sensitivity: number of true positives/number of true positives + number of false negatives = number of true positives /total number of sick individuals in a population

Specificity: Ability to correctly detect the individuals without the condition i.e. true negatives

Positive predictive value signifies the likelihood that an individual who obtains a positive

screening result actually has the condition

Likelihood ratio is used for assessing **the value of performing** a diagnostic test.

An LR of **> 1** indicates test result is associated with disease.

LR **< 1** indicates that the result is associated with **absence** of disease.

Tests where LR lie close to 1 have little practical significance as the post-test probability is not different from the pre-test probability. **Pre-test probability** refers to the chance that an individual has a disorder or condition prior to the use of a diagnostic test.

Positive predictive value/Positive likelihood ratio tells how much to **increase the probability** of aneuploidy if soft marker is **present.**

Negative predictive value/Negative likelihood ratio tells how much to **decrease the probability** of aneuploidy if soft marker is **absent.**

Prevalence refers to the **number of cases** of a disease present in a particular population at a given time.

Incidence refers to the number of **new cases** that develop in a given period of time.

Background/baseline risk is the incidence of a disease in exposed group

I**ncreased risk** implies added risk

Relative risk estimates the magnitude of disease based on the incidence of disease **in the** exposed group **relative** to the unexposed group

Attributable risk or **risk difference** is the absolute **difference** in incidence **between** an exposed and unexposed group.

Measures of central tendency of the data are as follows:

Mean: Arithmetic average where all numbers are added up and then divided by the total number of variables.

Median: **Middle value** of a data set. All values are listed in numerical order from smallest to largest and the list is arranged in ascending or descending order in order to find the median

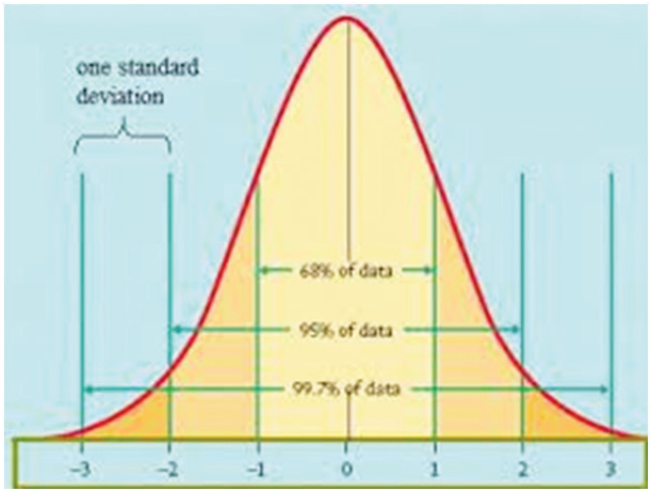
Mode: It is the most commonly occurring value. *If no number in the list is repeated, then there is no mode for the list.*

Multiple of the median (**MoM**) is a method to **normalize data**. It is a measure of how far an individual test result deviates from the median. *MoM is commonly used to report the results of medical screening* tests, particularly where the results of the individual tests are highly *variable.*

For example, maternal serum alpha-fetoprotein (AFP) testing is used to screen for a neural tube defects (NTDs) during pregnancy. If the median AFP result at 16 weeks of gestation is 30 ng/ml and a pregnant woman's AFP result at that same GA is 60 ng/ml, then her MoM is equal to two (60/30 = 2.0) i.e.her AFP result is 2 times higher than "normal."

Measures of dispersions:

Normal distribution curve is a probability distribution that is symmetric about the mean, showing that data near the mean are more frequent in occurrence than data far from the mean. In graphical form, the normal distribution appears as a "bell curve".



Variance and Standard deviation *both measure how spread out data is.*

•**Variance** = average difference of all data points from the sample mean squared

•**Standard deviation** is the square root of variance

•68% of sample data falls within 1 SD

•95% of sample data falls within 2 SD

•99.7% of sample data falls within 3 SD

The greater the standard deviation, more spread out is the data set.

Z-scores are a way of comparing the results with a normal population. It tells where the score lies on a **normal distribution curve**. **A positive**

score represents data above the mean and a **negative score** represents data below the mean. e.g. z-score of **zero** means values is the same as average while a score of +3 means that the value is much higher than the average, a score of -1.8 is 1.8 standard deviations below the mean.

Percentiles use the median as the average (50th percentile) while z-scores use the mean as average (Z-score of 0).

"I AM A FETUS"

I am a fetus .. unborn
Innocent yet forlorn

I began my sojourn
With a happy face
I was hope, I was joy
was laughter & grimace

Until someone peeped
through the window
And saw me as a
black n' white shadow

I had potential.. In plenty
And countless jubilations
But for this person I became
a bundle of calculations

Soon so many of them
Began to stand and stare
Before I knew it I was
Part of a software

From no where my Mom
Began shedding tears
I was supposed to bring
Immense joy for years

And then I heard this
She was about to get pricked
I could sense it all
As all around me panicked

The doctor had said,
"it wasn't necessary"

But everyone wanted
A certificate in a hurry

Piles and piles of
Guidance poured
Google, twitter, WhatsApp
The info just soared ..
I was silently watching
This utter dismay
Wondering What had led
to this sudden disarray

Until I realised they didn't want
To take an iota of chance
No one was now ready
To even give me a glance

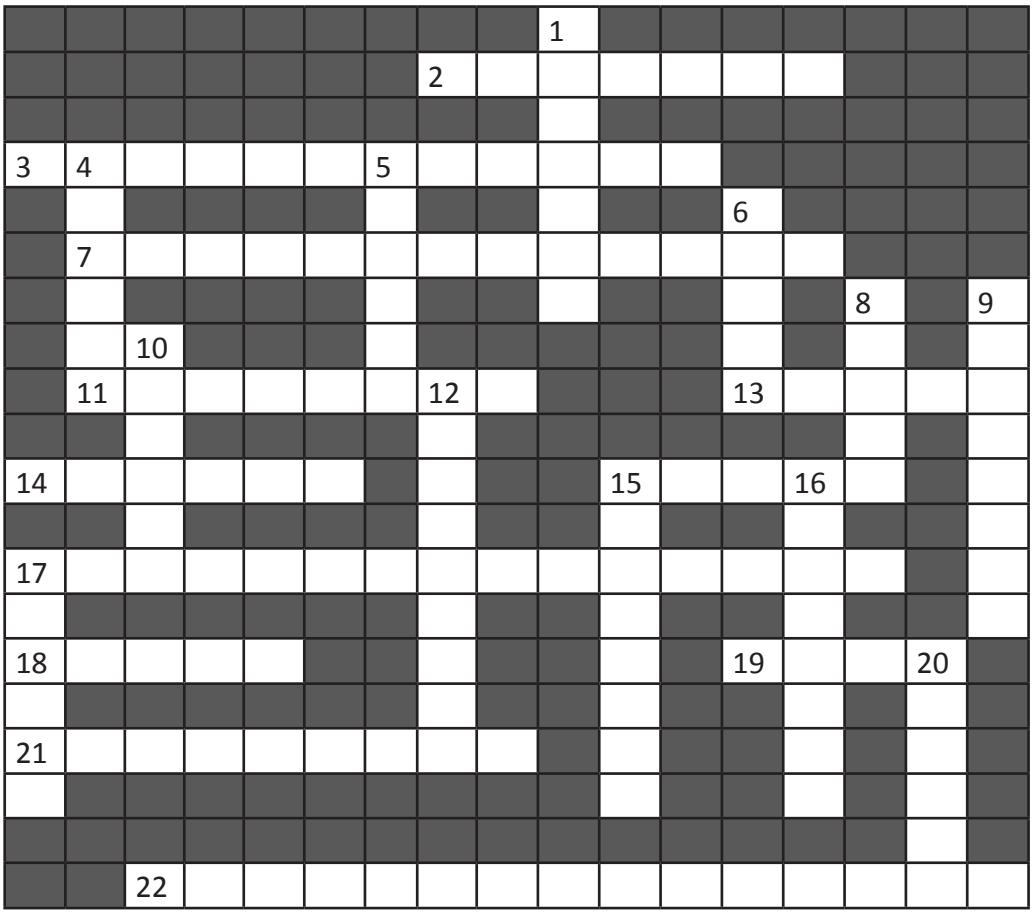
No they won't let me be born
Won't like to see who I was
The desire for a perfect child
Had brought me to this chaos

All the family put
Their thinking hats on
And decided in a jiffy
I was dangerous to be born

I was .. without any further testing ..
too dangerous.. to be born .

Dr Jaya Chawla,
Associate Professor,
Deptt of Obstetrics and Gynaecology,
ABVIMS & Dr RML Hospital,
New Delhi

CROSSWORD PUZZLE



Across

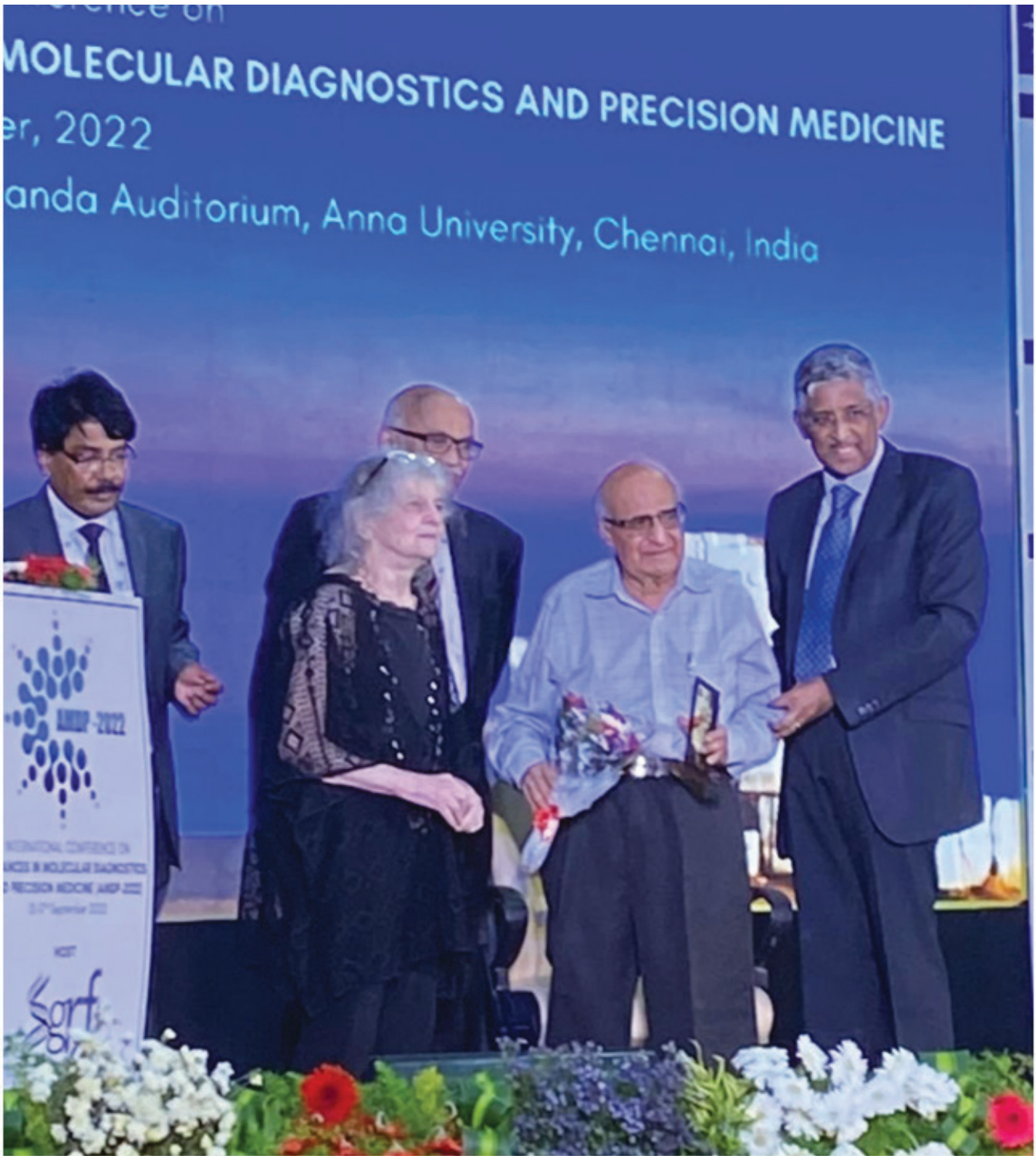
2. Cranio-facial syndrome with Craniostenosis, hypertelorism, proptosis and midfacial hypoplasia.
3. Encephalocele, polydactyly and polycystic kidneys.
7. Lissencephaly, ocular anomalies and Z -shaped brain stem
11. X-linked muscular dystrophy
13. Overgrowth syndrome with brain anomalies.
14. Characteristic flat facial profile
15. _____Cheney
17. Growth restriction, abnormal facial profile, synophrys, heart defects, hand abnormalities, CDH
18. Craniostenosis, mitten hands
19. Commonest aneuploidy
21. Facial asymmetry, ear, vertebral and ocular anomalies.
22. Cranio-facial dysostosis with micrognathia, hypoplastic zygomas and low set ears.

Down

1. Molar tooth sign
4. A common aneuploidy with micrognathia, heart and limb anomalies.
5. A form of distal arthrogryposis with cleft palate.
6. Diaphragmatic hernia, distal limb hypoplasia, cranio-facial anomalies
8. Aneuploidy with holoprosencephaly, facial anomalies, polydactyly and heart defects.
9. Facio-cutaneous-skeletal syndrome with nuchal thickening, macrocephaly, short long bones, tachycardia and polyhydramnios.
10. Monosomy with cystic hygroma and left-heart defects.
12. Severe growth restriction, diffuse edema, akinesia, contractures, microcephaly, marked ocular proptosis.
15. A type of Heart-hand syndrome
16. Heart defects, thymic abnormalities, cleft lip-palate.
20. Suspect when increased Nuchal translucency but normal karyotype.

Answer in the Next Editon of the Newsletter

Image Gallery



Prof. IC Verma, Founder President of the Society of Fetal Medicine receiving Lifetime Achievement Award from Nobel Laureate Prof. Ada E Yonath during International Conference on Advances in Molecular Diagnostics and Precision Medicine organised by SGRF in Chennai.

Some glimpses from the first quarterly meeting organized by the Delhi Chapter of the Society of Fetal Medicine in association with Indian Fertility Society at Sir Ganga Ram Hospital, New Delhi.
Topic: Genomic medicine and assisted reproduction – changing scenario.

Delhi Chapter Events' Calendar

Month/Year	Faculty	Theme/Topic	Site
24th July, 2022	Dr Ratna Puri, Dr Veronica	ART Pregnancies	SGRH
October, 2022	Dr Chanchal	Prevention of Preterm Birth	Rainbow
January, 2023	Dr Vatsla Dadhwal	Fetal Growth Restriction	AIIMS
April, 2023	Dr Jaya Chawla	Multifetal Pregnancy	RMLH
July, 2023	Dr Akshatha Dr Alok Varshney	Demystifying Neurosonogram	Apollo
October, 2023	Dr Ratna Puri Dr Sumitra Bachani	Fetal Autopsy/Postnatal Evaluation in Still Birth	SJH
January, 2024	Dr Manisha Dr Sucharita Jain	Revisiting Basics Including PE Screening	LHMC
April, 2024	Dr Sangeeta Gupta	Mixed Bag	MAMC

"Do not go where the path may lead, go instead where there is no path and leave a trail."

-Ralph Waldo Emerson

Please send your comments, feedback and suggestions to
delhichaptersfm@gmail.com
We also look forward to submissions to be included in subsequent editions

www.societyoffetalmedicine.org