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Comprehensive Textbook of SEXUAL MEDICINE

SECOND EDITION



Editors
Nilamadhab Kar
Gopal Chandra Kar

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CHAPTER 3

Genetic and Endocrinal Factors in Sexual Development

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Introduction

Life seems to have originated on earth some 2000 million years ago and is possible by the unique activity that is reproduction. Reproduction is possible due to sexual development and is practically impossible if any abnormality in sexual development occurs. It is now clear that the primitive bipotential sex gland, i.e. the gonad (gone-seed) differentiates into either a male gonad, i.e. testes or female gonad, i.e. ovary and the predominant determining factor is solely a single chromosome, i.e. the 'Y' chromosome.[1,2] The formation of genitalia depends on the secretion of the testes. In the presence of functional testes the genitalia become male type and in the absence of functional testicular tissue the general features and the genitalia become female type.[3] Hence the most important factors for the sexual development are (i) genetic, (ii) endocrinal.[4]

Genetic Role in Sexual Development

The genetic control of sexual development is very often called the sex determination. In human there are 46 chromosomes. Out of 46 chromosomes 44 are autosomes or somatic chromosomes and the other two are sex chromosomes. The genetic sex (sex genotype) depends on these two sex chromosomes. These are named as 'X' and 'Y'. In females the sex chromosomes are 'XX' whereas in males it is XY. The Y chromosome is sufficient for production of testes, i.e. male gonad. The testes determining gene product in Y chromosome is called sex determining region of the Y chromosome (SRY). SRY is a DNA binding regulatory protein. It bends the DNA and act as a factor that initiates transcription of a series of genes, which are required for testicular differentiation. The gene for the SRY is present in the tip of the short arm of Y chromosome. During the process of gametogenesis the haploid cells like ovum and sperm contain only 23 chromosomes each, i.e. 22 somatic chromosomes and one sex chromosome. In male, the spermatogonia may be 22X or 22Y but in female it is only 22X.[4] In males 50% of sperms contain 22X and the other 50% contain 22Y.

The process of gametogenesis consists of two stage meiotic division which produces the haploid cells containing 22 autosomes and a sex chromosome, i.e. X or Y. But there is a difference in male and female. In females, in the two stage division, only one cell survives as the mature ovum containing 22X and in male the two stage division produces 4 sperms, two containing 22Y and the other two containing 22X.

During fertilization if a sperm containing 22Y chromosome unites with an ovum containing 22X then the zygote formed becomes a genetic male whereas if a sperm containing 22X fertilizes with an ovum containing 22X then a genetic female is formed.

Study of Chromosomes

Chromosomes are studied in different ways. The important methods for diagnosis are buccal smear examination for the presence of Barr body or nuclear chromatin or F body fluorescence; chromosome analysis of lymphocyte culture of peripheral blood; and chromosome analysis following fibroblast culture of skin, gonad and amniotic cells.

Buccal smear: Buccal smear examination gives indirect information about a possible sex chromosome abnormality. This smear is taken by a metal spatula from inside the cheek. Then it is smeared on a slide and immediately fixed by 95% alcohol and ether. Then it is stained and examined. Barr bodies are densely stained mass of 0.8 - 1 micro meter size. In normal males and persons with 45 XO, there is no Barr body. In individuals with 47 XXY cases there is only one Barr body; 47XXX show two barr bodies and 48XXXX show 3 Barr bodies. As the buccal smear is not reliable, the chromosome analysis is preferred.

Lymphocyte culture: The culture of the blood cells is done by taking an EDTA blood which is stored at –20°c. Then the leukocytes are separated and lysed. The lysate is mixed with phenol which dissolve the proteins and lipids leaving behind the DNA material. By using RNAses the RNA is destroyed and DNA is isolated by precipitation with alcohol. By using restriction endonucleases, the DNA is cut into pieces at selected sites. Then by electrophoresis the fragments are separated in agarose gel medium. The DNA fragments are visualized under ultraviolet light after staining with ethidium bromide which fluoresces under ultraviolet light. The specific regions of DNA are identified by southern blot technique. The specific regions of RNA are identified by northern block technique.

Fibroblast culture: Many tissues like skin, amniotic fluid, gonads and rectus sheath are used for culture and chromosome analysis. Fibroblasts grow rapidly and are hence preferred for culture. The human cells, i.e. fibroblast are first cultured and grown. A drug, i.e. colchicine is added to stop the mitosis at metaphase. Then a hypotonic solution is added to make the chromosomes swell and disperse. Then fluorescent and other staining techniques on a slide study the chromosome.

Clinical Application of Chromosomal Study

Major indications for analysis of chromosome are suspected cases of:

1. Gonadal dysgenesis
2. Testicular feminization
3. Congenital absence of vagina
4. Virilization at puberty
5. Ambiguous sexual development
6. Superfemales and Klinefelter's syndrome
7. Recurrent abortion
8. Malformed infants.

Diagnosis of Genetic Sex: Sex Chromatin

The two sex chromosomes of the cell are derived from paternal and maternal sources. In females, in 50% cells, X chromosome from one source is active, while the other X chromosome remains active in the rest. One X chromosome always remains inactive. The active chromosome is randomly selected. The chromosome is inactivated by the gene X_{ist}. This inactive chromosome usually condenses and is visualized as a Barr body in the buccal smear or drumstick in neutrophils. The condensed chromatin is called a sex chromatin. By investigating the number of sex chromatin, the diagnosis of genetic sex can be determined.

Abnormalities in Chromosome Number

During the process of sex determination the chromosome numbers may be abnormal, i.e. there may be addition or omission of a

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chromosome and the total chromosomes thus may become 45, 47, 49 or 50. For the first time in 1959, sex chromosome aneuploidy in man was demonstrated by Jacobs and Strong in Edinburgh and Ford and his colleagues at Harwell. Aneuploidy is any deviation from the normal 46 chromosomes in a cell, e.g. monosomy, trisomy and polyploidy.

Monosomy: It is the absence of one of a pair of chromosomes, e.g. 45XO, 45YO. Except 45XO all others are lethal. The incidence of 45XO, i.e. the Turners syndrome is 1:2500.

Trisomy: It is the presence of an extra chromosome, which arise as a result of nondisjunction due to failure of separation of homologous chromosomes during meiosis. It results in cell containing 24 chromosomes and 22 chromosomes. Non-disjunction of any chromosomes can occur. Except trisomy of 21, 18 and 13, the rest are lethal in utero. Trisomy 21 is Down's syndrome and trisomy 18 is Edward syndrome. Trisomy 13 is the Patau's syndrome. These three conditions are associated with severe mental retardation and various malformations. Trisomy of sex chromosomes is compatible with life.

Mosaicism: In mosaicism an individual has got two cell populations each with a different genotype. It happens when non-disjunction occurs during the early cleavage of zygote.

Polyploidy: In these cases the cells contain extra copies of all the chromosomes, e.g. triploidy 69. Polyploidy usually causes abortion. If live birth occurs then the child is severely malformed and dies early in postnatal period. The extra set of chromosomes is usually derived from the father.

Turner's Syndrome

This happens due to a defect in gametogenesis called non-disjunction. Here during division a pair of chromosomes fails to separate. As a result both the chromosomes go to one cell and the total number of chromosomes become 24 (22XX) and a cell with one less chromosome is formed, i.e. 22 (22O). After fertilization the zygote formed will have chromosome pattern 45XO or 45YO. The 45YO pattern is always lethal. The 45XO pattern is called the Turner's syndrome or gonadal dysgenesis; and buccal smear becomes negative for the sex chromatin.

Clinical features: Persons with Turner syndrome are usually of short stature, have webbed neck, wide carrying angle, and coarctation of aorta. There is no gonadal differentiation, i.e. they contain only streaks of tissues; the gonads are not well developed and may be absent so that the female external genitalia develop. At puberty there is no secondary sex development. There is low hairline (trident), low set ears, hypoplastic nails, edema of dorsum of hand and feet. They have shield like chest, cubitus valgus, pigmented navel and keloid formation. The girls often present with primary ammenorrhea. They usually abort if they get pregnant.[5-8]

Klinefelter's Syndrome

It is also called seminiferous tubule dysgenesis. The karyotype is 47XXY. The man is tall. Testes are infantile. There occurs development of male breast (gynecomastia);[9] and female type of fat deposition. There is eunuchoid habitus, and increased carrying angle at elbow. There is usually infertility and mental retardation. The serum testosterone is low and buccal smear is positive. Seminiferous tubule dysgenesis is seen in 2 in 1000 males.

Structural Chromosome Abnormality

Structural abnormality of the chromosomes occurs when there is a break of chromosome and the nature of chromosomal abnormality depend upon the fate of the broken piece.[10] The examples are deletion, duplication and inversion.

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Deletion: Deletion occurs when the broken piece is lost. On analysis the chromosome is usually coiled and is called a ring chromosome. Identification of the missing portion is difficult in a ring chromosome. Otherwise they are identified by 'G' banding.

Duplication: Duplicated material may get attached to any portion of the chromosome. It is compatible with life as there is no loss of genetic material.

Inversion: It occurs when one chromosome segment is reversed in the orientation. For example, paracentric inversion, i.e. involving one single arm of chromosome and another pericentric inversion, i.e. involving both arms of the chromosome.

Isochromosome: During meiosis when the chromosome divides transversely rather than dividing longitudinally isochromosomes develop. So one set contains two short arms; and another contains two long arms. It may result in the Turner phenotype.

Translocations: Translocations occurs when chromosomes become broken during meiosis and the resulting fragment gets attached to another chromosome. In a reciprocal translocation the genetic material exchange occurs between two chromosomes, i.e. a portion of one chromosome exchange with a portion of another chromosome. So, total number of chromosomes are 46 but two abnormal chromosomes exist. If there is no genetic loss then the development is balanced. If a loss of genetic material occurs then it can be identified by 'G' banding and lead to developmental abnormality. Translocation usually occurs between homologous chromosomes. There is a chance of the offspring to get two normal chromosomes or two abnormal chromosomes or an addition of genetic material or reduction in genetic materials, e.g. in Down syndrome there is addition of 21 chromosome material which is attached to chromosome No 11. In Robertsonian translocation two chromosomes adhere to each other and become one. So, total number of chromosomes becomes 45.

49XXX/50XXXX

These individuals are females and chromosome pattern is multiple X. They are also referred as 'superfemales'. The affected persons are usually mentally retarded and the retardation is proportional to the number of X chromosome. But their children are usually normal. The intelligence quotient (IQ) is usually average. The features are typically wide set eyes, low set nasal bridge, epicanthus, mandibular prognanthism, low set ears, short neck. There are radioulnar synostosis, cryptorchidism and congenital heart defects.

True Hermaphroditism

In these persons there are features of both male and female, e.g. an ovotestis or a testis in one and an ovary on the other side. The other secondary sex characters are mixed type and cannot be determined. It is due to XX/XY mosaicism, i.e. faulty mitosis in early zygote with non disjunction. Most common karyotype is 46 XX (58%), others are 46 XX/XY (13%), 46XY (11%), 46XY/47XXY (6%). Diagnosis is made by gonadal biopsy. The sex of child is determined on the functional capability of the external genitalia and subsequently inappropriate organs are removed. An ovotestis may be removed with hormone therapy at puberty. If a child is brought up as male but breast develop and menstruation occurs then mastectomy and ovariectomy are advised and the child is allowed to live as male.

Testicular Feminization (Androgen insensitivity)

In testicular feminization the karyotype is that of a normal male, i.e. 46XY; so they are genetically male. But they fail to develop normal male secondary sex characters due to deficiency of male sex hormone, i.e. testosterone. The probable cause may be absence of enzymes for testosterone synthesis or absence of testosterone receptors in target cells or nonconversion of testosterone to dihydrotestosterone due to deficiency of 5α

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reductase enzyme.[11] Most patients with androgen insensitivity present for the first time at puberty with amenorrhea. On examination there is scanty pubic hair, a normal vulva with a blind short vagina with no cervix. On laparoscopy the uterus is absent, and testes are present in abdomen or groin or labia. The testosterone level is normal, estrogen level is male or female type and LH level is usually raised. The management depends on age at which it presents. If after puberty, then testis may be removed and estrogen therapy given to maintain femininity only, as the uterus is absent; and reconstruction of the genitalia to female type is advised.

Endocrinal Factors in Sexual Development

Once the genetic sex is determined, the sex differentiation is done by hormonal factors. In human beings, the sex differentiation is so marked that it is easy even for a child to differentiate the sex of an individual. In humans, the female is considered as the standard model of human species and male is only a deviation of it. This deviation, to maleness is due to the male hormone. Hence, if the male hormone is present in the embryo then the genetic male embryo develops to a male, or male like characters develop in a genetic female. In absence of the hormone the female characters develop.

The hormonal dependence is evidenced by the following facts: (i) If the gonad of early-undifferentiated embryo is removed then embryo develops to a female. (ii) Removal of testes in adult causes regression of male secondary sex characters where as removal of ovary in adult has no such effect. (iii) Exposure of a genetic female embryo to androgens leads to development of male genitalia.

Male Sex Differentiation

If the genetic sex is male, i.e. 46XY, then the bipotential gonad of embryo under the influence of SRY (sex determining region of Y);[2] which is a DNA binding regulatory protein, helps in transcription of genes required for testes differentiation, which subsequently secrete mullerian inhibiting substance (MIS), testosterone and dihydrotestosterone (DHT). The MIS is a 536 amino acid homodimer and a member of TGF-β family of growth factors including activin and inhibins.[12] The MIS helps in regression of mullerian duct system and prevents development of uterus, fallopian tubes and upper part of vagina.[13,14] Testosterone helps in development of Wolffian duct system, i.e. vas, epididymis and seminal vesicles of male internal genitalia. The DHT helps in development of male external genitalia, i.e. penis, scrotum and prostrate **(Fig. 3.1)**.

Another important role of testosterone is development of a male brain; which means there is a greater density of neuronal synapses in amygdala and left hemisphere dominance develops early.[1] The hypothalamus gets programmed for continuous gonadotropin releasing hormone secretion, contrary to the cyclical secretion in females. The testis, at puberty, secretes DHT which develop the secondary sex characters.

Female Sex Differentiation

If the genetic sex is female, i.e. 46XX then there is no SRY and no testicular tissue. The absence of male hormones causes the embryo to develop to female. The bipotential primordia develops the female internal genitalia, i.e. uterus, tubes and vagina and external genitalia like labia, clitoris and lower part of vagina. The embryonic ovary is converted to adult ovary, which secretes estrogen, which is responsible for secondary sex characters in female **(Fig. 3.2)**.

Abnormalities of Sex Differentiation

Female Pseudohermaphroditism

The basic abnormality is an excess of androgens in utero or after birth, which leads to ambiguous

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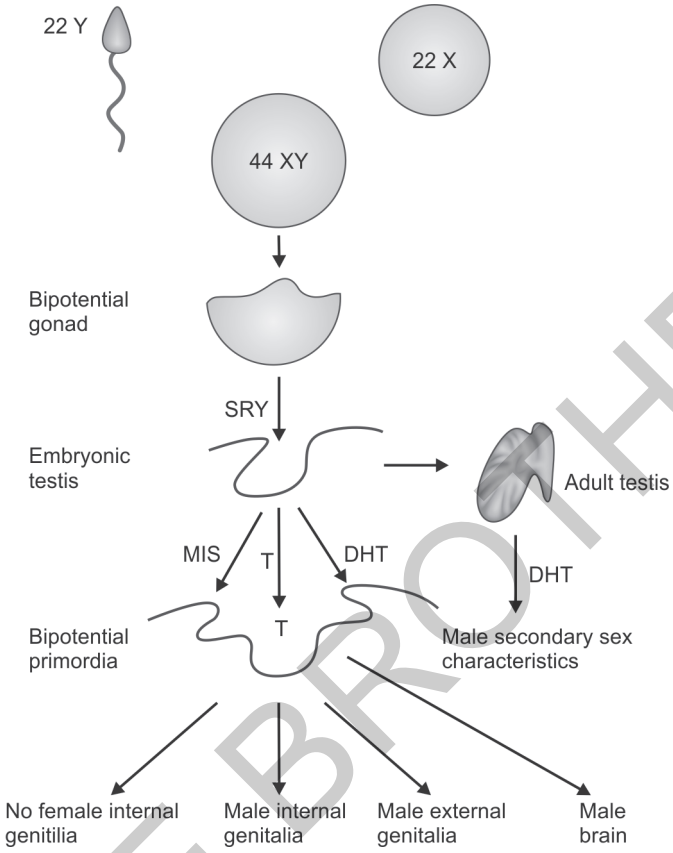

Figure 3.1 Male sex differentiation

external genitalia in the female fetus. The major causes are congenital virilising adrenal hyperplasia of fetus, maternal androgen excess, virilizing ovarian tumor and treatment with androgen or synthetic progesterone (iatrogenic).

Male Pseudohermaphroditism

The causes are resistance to androgens, defective testicular development, congenital 17 α hydroxylase deficiency, and congenital adrenal hyperplasia due to blockage of pregnenolone formation.

Congenital Adrenal Hyperplasia (CAH)

It is the most common cause of female intersex and is an autosomal recessive disorder resulting in enzyme deficiency related to the biosynthesis of cortisol and aldosterone.[15] The most common enzyme defect is 21-hydroxylase deficiency resulting in a failure of conversion of 17 α hydroxyprogesterone to deoxycortisol and also failure of conversion of progesterone to desoxycorticosterone. As a result of no negative feedback regulatory mechanism

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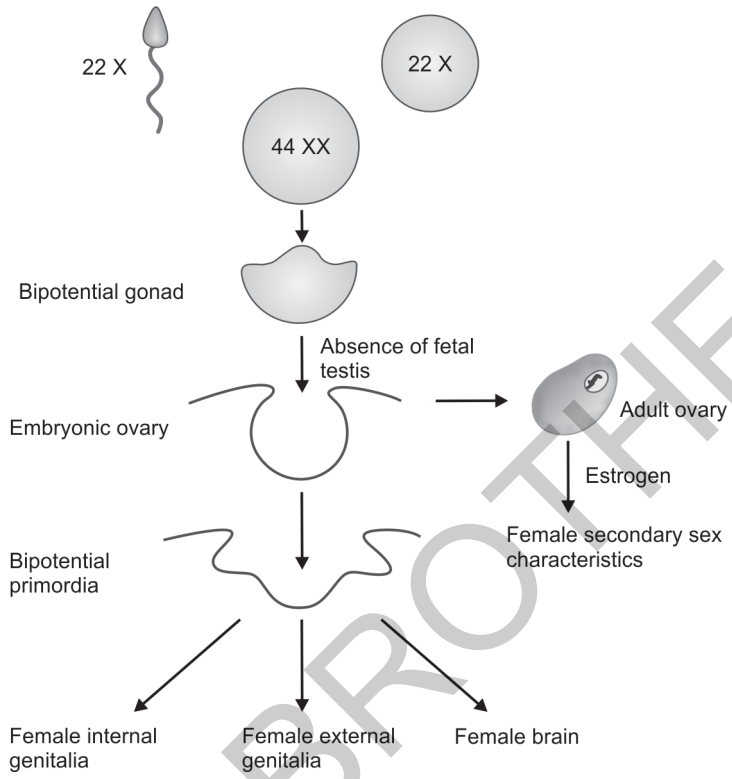

Figure 3.2 Female sex differentiation

the adrenocorticotropic hormone (ACTH) production becomes high and the intermediary metabolites accumulate in the body. These metabolites are androgenic in nature and are also converted to androgen in the body and produce virilization. Depending on the enzyme deficiency the cortisol deficiency may be severe or mild or may be normal due to hypertrophied gland. Deficiency of the other two enzymes, i.e. 3 β hydroxysteriod hydrogenase and 11 β hydroxylase deficiency are also noted. The 21 hydroxylase deficiency accounts for 90% of the CAH. The deficiency leads to increase level of progesterone and 17 α hydroxyprogesterone which is converted to androstenedione and subsequently to testosterone and there is increased urinary pregnanetriol.[16]

The relationship of the 21-hydroxylase deficiency to HLA type was established in 1977 by Dupon et al.[6] The gene is located in the short arm of chromosome 6 between HLA-B and HLA-DR. The salt losing types of CAH is due to HLA-B subgroup of gene disorder and HLA-BW51 is related to simple virilizing forms. Donohoue et al showed that there are two 21 hydroxylase genes. One is 21 hydroxylase A and the other 21 hydroxylase B. The 21 hydroxylase B is active and lie between the 4th components of C4A and C4B. The incidence of 21 hydroxylase deficiency is 1:5000 to 1:15000.

The other enzyme deficiencies producing CAH may be C 20–22 desmolase, 18 hydroxylase, and 18 dehydrogenase.

Features: Due to exposure to androgen, the child at birth shows signs of virilization, i.e. hypertrophied clitoris and labial fusion. So, there is a resemblance to male external genitalia. The male child usually dies due to cortisol deficiency. There is usually enlargement of clitoris, and

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excessive fusion of genital folds leading to obscure vagina and urethra, leading to only one artificial urogenital sinus opening to the perineum. There is thickening and rugosity of labia having some resemblance to scrotum. This thickness of the labial folds leads to narrowing of the vagina. The vagina opens at some point of the urogenital sinus. The uterus and tubes are always present.

In some infants due to associated aldosterone deficiency a salt losing syndrome may arise. The child may die of wasting and vomiting within a few weeks of life. The same virilization occurs in an ovarian tumor producing androgens and also if androgenic drugs or progesterone are given. In 17 α hydroxylase deficiency the corticosterone and deoxycorticosterone level increases and simultaneously the androgen of the testes and estrogen of ovary are deficient.[17] It leads to primary hypogonadism in female and pseudohermaphroditism and gynecomastia in male. In male pseudohermaphroditism, the defect is due to an abnormal testicular differentiation, abnormal testicular function or abnormalities of target tissue response, i.e. resistance to androgens.

Investigations: Investigation of a suspected case of congenital adrenal hyperplasia should include:

1. Chromosome analysis to establish karyotype.
2. Measurement of urinary androgen level by assay of urinary 17-oxosteroids.
3. Assay of 17 α hydroxy progesterone in blood or urinary pregnanetriol estimation.
4. Estimation of serum electrolytes like Na^+, K^+, and Cl^- to establish a salt losing types of CAH.
5. Ultrasonography of pelvis to detect organ anomalies.

Management: The mainstay of treatment is to suppress the ACTH secretion by cortisol therapy and correction of the anomalies by surgery in cooperation with the pediatrician. The surgery includes reduction in size of clitoris and division of labial fold to expose the urethra and vagina.

Prenatal Screening and Diagnosis of Fetal Abnormalities

Ultrasound

Ultrasound diagnosis of genetic disease has gained prominence in the last few years. Fetuses with cytogenetic defects having external or internal defects can be seen with the ultrasound. The markers like increased nuchal thickness, pyelectasis, and short femur are mainly associated with chromosomal abnormality. The central nervous system (CNS) markers are holoprosencephaly, agenesis of corpus callosum, ventriculomegaly, and choroid plexus cyst. The cardiovascular system (CVS) markers are tetralogy of Fallot, ventricular septal defect (VSD), atrial septal defect (ASD) or a double outlet ventricle. The gastrointestinal (GI) markers are duodenal atresia, esophageal atresia, omphalocele, and diaphragmatic hernia. The genitourinary markers are pyelectasis, multicystic dysplastic kidney, nephromegaly. The facial markers are cleft lip, cleft palate, ocular and ear abnormality. The limb markers are club foot, limb length discrepancy, sandal toe and rocker bottom foot.

Maternal Blood Test

The study of fetal cells in maternal circulation by polymerase chain reaction or fluoroscent in situ hybridization (FISH) or specific DNA probes and biochemical testing of maternal serum are done to detect the chromosomal abnormality.

Amniocentesis

It was first used for the management of hydramnios and subsequently used as a diagnostic procedure.[18-22] In 1966 Steel and Berg first determined successfully the fetal karyotype.[23] Jacobson and Barter in 1967 first made a diagnosis of chromosomal anomaly by using the amniocentesis method.[24]

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Indications: It is indicated in the late first and second trimester for diagnosis of cytogenetic defects, neural tube defects and metabolic disorders. In the late second and third trimester it is useful for evaluation of severity of iso-immunization and fetal lung maturity. The time during which amniocentesis is done for the cytogenetic diagnosis is between 15 and 17 weeks of gestation. It is usually done in women of 35 years and above. It is also indicated in the women with previous offspring affected by chromosomal abnormality or in a woman with X linked disorder. The study takes an average time of 1–2 weeks. So, it allows sufficient time for medical termination of pregnancy (MTP) if indicated. But the main disadvantage is that when it is done in second trimester of pregnancy, the termination of pregnancy becomes difficult and more traumatic both mentally and physically.

Procedure: An ultrasound is done to determine the number of fetuses, placental location and the amount of amniotic fluid.[25] Then puncture site is determined. A 20–22 gauge needle is selected with a length of 8.89 cm. Then under strict aseptic procedure and under the guidance of the ultrasound the amniocentesis is done. The fluid is then subjected to culture and chromosomal analysis.

Usual complications: The main complications are fetal injury leading to fetal loss, subsequent respiratory complications, leakage of the amniotic fluid, and fetomaternal transfusion.

Fetal Blood Sampling

It was first done in 1973 by a fetoscope-guided trocar. Blood was collected from fetal heart and also intra-abdominal part of the umbilical vein. Recently percutaneous umbilical blood sampling (PUBS) is done under the ultrasound guidance.[26-29]

Indications: The main indications of the PUBS are chromosomal analysis and mendelian genetic disease, diagnosis of congenital viral and parasitic infections, evaluation of blood disorders and perinatal fetal assessment.

The best time for amniocentesis for cytogenetic analysis is 18 weeks onwards. By ultrasonography the sampling site is detected and then under strict aseptic precaution a 20–22 gauge needle is inserted and blood is drawn from umbilical vein. Then it is subjected to chromosomal analysis.

Chorionic Villus Sampling

Kazy was the first to suggest the use of ultrasound guided biopsy of the chorion frondosum.[30] Subsequently, it was modified by Humphrey ward in London and refined by Bruno Brambatti in Milan.[30-34]

Procedure: As the chorion frondosum contains the mitoticaly active cells it is the preferred site of sampling. Prior to sampling, gestational age and fetal viability are confirmed by ultrasound. The procedure is best performed between 70–91 days of last menstrual period. By ultrasound guidance the chorionic frondosum is identified and by using a transcervical catheter or transabdominal needle the sample is taken and subjected to chromosomal analysis.

Biochemical Markers

The biochemical markers useful for the cytogenetic analysis and diagnosis are maternal serum alpha-fetoprotein (AFP), amniotic fluid AFP, maternal serum human chorionic gonadotropin (hCG) and maternal serum unconjugated estriol. Maternal serum AFP is best done at 15–18 weeks. An increased level of AFP is seen in neural tube defect. AFP is a fetal specific glycoprotein which reaches the amniotic fluid by the fetal urine, GI secretions and transudation from the vessels and then subsequently diffuses to maternal blood via placenta. Amniotic fluid AFP estimation is undertaken on the cell free fluid using the rocket method of counter immunoelectrophoresis. The height of the rocket is directly proportional to the

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level of AFP in the sample, which is calculated by reference to known standards.

Genetic Counseling

Following the birth of a severely handicapped child, the possibility of genetic cause should be thought, as there may be high recurrence risk in future pregnancies. Hence, it is important to look for a genetic cause and establish a correct diagnosis so that accurate genetic prognosis can be given to parents. Genetic counseling clinics are now providing the information regarding the recurrence risks to parents. In any case of fetal abnormality the parents should undergo complete investigation including chromosome analysis.[35] The important role and responsibility of the genetic counselling is that if an inherited disorder is detected then the whole family are also counselled for risk of genetic disorder and may get the benefit of genetic advice.

Conclusion

Genetic and endocrinal factors in sexual development are important to understand the cause of handicapped child so as to help the child as well as the parents. The problem of fetal handicap concerns not only the obstetricians but the pediatricians, the family, and the society. Major fetal abnormalities are an important cause of perinatal death to the tune of around 20%. It is seen that if a handicapped child is born then the chance of subsequent child being handicapped is more. Antenatal diagnosis should be offered to mothers 35 years of age and above, mothers with previous mongoloid child, mothers liable to have chromosomally unbalanced child due to parental chromosome translocation, mothers with previous child affected with autosomal recessive metabolic disorders, mothers who are known carriers of X-linked recessive disease and mothers with high level of AFP. Ideally prenatal diagnosis of abnormality should be made as early as possible in pregnancy before the age of viability and preferably before fetal movements are felt. Hence the results of the tests performed should be available by 20–22 weeks of gestation.

The decision to have a prenatal diagnosis or not; and to accept the selective MTP or not; are to be strictly taken by parents. Most mothers at risk of bearing a handicapped child are always eager to know the possibility of testing their pregnancy to make certain whether their child is healthy or normal. Most of them are prepared to accept a selective MTP if a lethal and severe fatal abnormality of the child is detectable in pregnancy. Though most of the conceptions affected by chromosomal abnormality are spontaneously aborted; those who escape spontaneous abortion need the MTP.

Starting from the patient's obstetric history, antenatal screening for chromosomal abnormalities by amniocentesis, chorionic villus sampling, ultrasonography, biochemical analysis of maternal and fetal blood are the mainstay of diagnosis of disorders. Some degree of complications like fetal injury, fetal abnormality and even fetal death limit the wide use of these procedures. For those concerned with the cost of such analysis it will be appreciated from even the most superficial cost-benefit analysis that this is a fraction of the expense required for looking after children born with abnormalities. It is clear that prenatal diagnosis has an important role to play.

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