A STUDY ON THE INFECTIOUS ETIOLOGY OF ABNORMAL VAGINAL DISCHARGE IN WOMEN OF REPRODUCTIVE AGE GROUP ATTENDING A TERTIARY CARE HOSPITAL

Dissertation submitted to

THE TAMILNADU DR.M.G.R.MEDICAL UNIVERSITY

In partial fulfillment of the regulations

for the award of the degree of

M.D. (MICROBIOLOGY)

BRANCH - IV

GOVT. STANLEY MEDICAL COLLEGE & HOSPITAL

THE TAMIL NADU DR. M.G.R. MEDICAL UNIVERISTY

CHENNAI

MAY - 2018

CERTIFICATE

This is to certify that this dissertation entitled **"A STUDY ON THE INFECTIOUS ETIOLOGY OF ABNORMAL VAGINAL DISCHARGE IN WOMEN OF REPRODUCTIVE AGE GROUP ATTENDING A TERTIARY CARE HOSPITAL"** is the bonafide original work done by Dr. MAJEETHA BANU S., Post graduate student in the Department of Microbiology, Govt. Stanley Medical College, Chennai in partial fulfillment of the regulations of the Tamil Nadu Dr. M.G.R. Medical university, Chennai towards the award of M.D., Degree in Microbiology (Branch IV).

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I, Dr. Majeetha Banu S., solemnly declare that the dissertation entitled **"A STUDY ON THE INFECTIOUS ETIOLOGY OF ABNORMAL VAGINAL DISCHARGE IN WOMEN OF REPRODUCTIVE AGE GROUP ATTENDING A TERTIARY CARE HOSPITAL"** is the bonafide original work done by me in the Department of Microbiology, Govt. Stanley Medical College, Chennai during the year October 2016 to September 2017 under the guidance and supervision of **Dr.N.Thilakavathi M.D.,** Associate Professor, Department of Microbiology, Govt. Stanley Medical College, Chennai.

This dissertation is submitted to Dr. MGR Medical University towards partial fulfillment of requirements for award of MD Degree Branch IV Microbiology.

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INSTITUTIONAL ETHICAL COMMITTEE, STANLEY MEDICAL COLLEGE, CHENNAI-1

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The request for an approval from the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 25.10.2016 at the Council Hall, Stanley Medical College, Chennai-1 at 2PM

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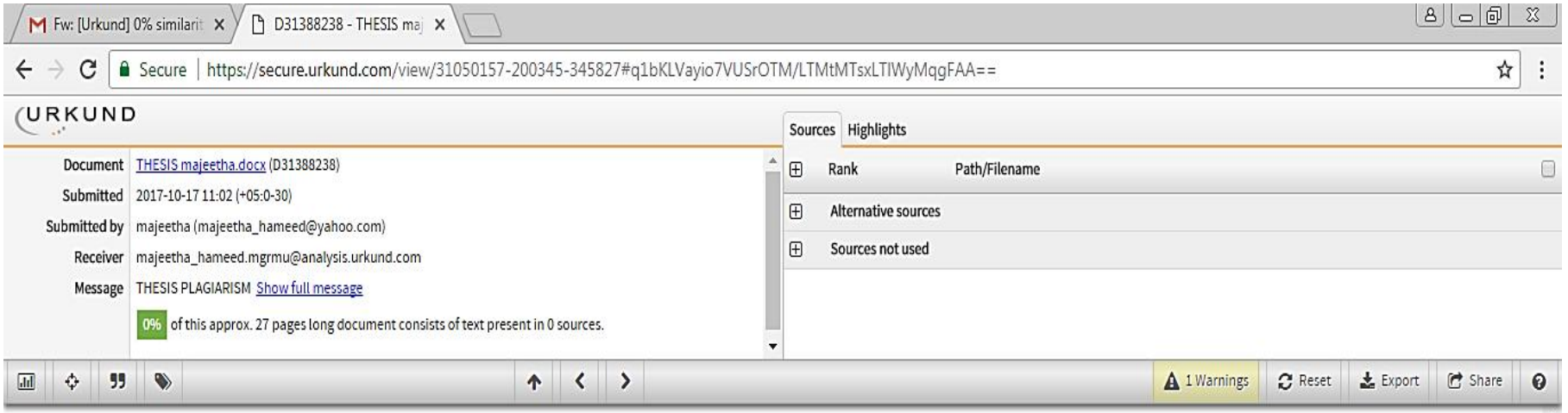

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TABLE OF CONTENTS

LIST OF TABLES

LIST OF CHARTS

LIST OF COLOUR PLATES

LIST OF ABBREVIATIONS

AVD - Abnormal Vaginal Discharge

BV - Bacterial Vaginosis

VVC - Vulvo Vaginal Candidiasis

TV - Trichomonas vaginalis

HPV - Human Papilloma virus

HSV - Herpes Simplex Virus

DIV - Desquamative Inflammatory Vaginitis

HRG - High Risk Groups

STD - Sexually Transmitted Diseases

IUCD - Intra Uterine Contraceptive Devices

OCP - Oral Contraceptive Pills

NACO - National AIDS Control Organization

CDF - Cell Detaching Factor

CPLM - Cysteine Peptone Liver Maltose

HRT - Hormone Replacement Therapy

NAC - Non albicans Candida

AFST - Anti-Fungal Susceptibility Testing

MHA - Mueller Hinton Agar

AV - Aerobic Vaginitis

MTM - Modified Thayer Martin medium

NYC - New York City medium

RCUT - Rapid Carbohydrate Utilisation Test

NAAT - Nucleic Acid Amplification Tests

PID - Pelvic Inflammatory Disease

SDA - Sabouraud's Dextrose Agar

BAP - Blood Agar Plate

PMN - Polymorphonuclear Neutrophils

RPR - Rapid Plasma Reagin

TPHA - Treponema pallidum HaemAgglutination

INTRODUCTION

INTRODUCTION

Abnormal vaginal discharge is a common clinical problem causing distress among reproductive age group women. Genitourinary symptoms in women usually lack specificity[1]. All women of reproductive age group have vaginal secretions, but they do not generally have the ability to discriminate normal from abnormal discharge. Some meticulous women may consider even normal physiological vaginal discharge to be abnormal while some ignorant women may disregard pathological vaginal discharge to be normal.

Abnormal vaginal discharge is fairly common and results from a variety of infectious and non-infectious causes. Infectious vaginal discharge can be caused by bacterial, viral, fungal or parasitic infections, the three most common causes being Bacterial Vaginosis (BV), VulvoVaginal Candidiasis (VVC), and Trichomoniasis[2].

Gonorrhoea and Chlamydia affect only the cervical columnar epithelium causing cervicitis but may cause vaginitis when the stratified squamous epithelium of vagina is damaged due to other predisposing causes. Some viral infections like Human Papilloma virus (HPV), Genital Herpes Simplex Virus (HSV) can also cause vaginitis and vaginal discharge. Syphilis is an ulcerative sexually transmitted disease caused by

treponemes which can also cause vaginal discharge if any vaginal ulcer is present.

Aerobic vaginitis is also seen in a small proportion of women caused by facultative anaerobic or aerobic bacteria mostly belonging to S. aureus, Group B Streptococci, Enterococci, E.coli and Klebsiella spp.

Non- infectious causes of abnormal vaginal discharge are many including an idiopathic condition known as desquamative inflammatory vaginitis (DIV), chemical irritants, vulvovaginitis associated with estrogen deficiency and other gynaecological conditions.

Abnormal vaginal discharge due to sexually transmitted infections is more common in women with high risk behavior (those having multiple sexual partners) than in normal women of reproductive age group. The common etiologies of vaginal discharge can also vary between the two groups of women.

Infection of the genito-urinary tract if left untreated or if inappropriately treated can lead to serious complications like pelvic Inflammatory disease (PID) that causes salpingitis, endometritis and tubo-ovarian abscess leading to scarring of the fallopian tubes. This

increases the risk of ectopic pregnancies, infertility, abdominal discomfort and chronic pelvic pain.

Genito-urinary infections cause increase in risk of invasive cervical cancer, cystitis, ascending pyelonephritis, pyrexia of unknown origin and most importantly increased susceptibility to HIV transmission.[3] Toxic Shock Syndrome by gram positive bacteria like Staphylococcus and Streptococcus spp. is especially associated with prolonged tampon use and lack of proper genital hygiene.

Genito-urinary infections in pregnancy can lead to increased risk of chorioamnionitis, Premature Rupture of Membrane (PROM), preterm labour, Intra-Uterine Growth Retardation (IUGR), Intra-Uterine Death (IUD), spontaneous abortions and repeated miscarriages.[4]

STI has an adverse consequence in the health of both mother and newborn. Vertical transmission of HIV, Congenital syphilis, Ophthalmia Neonatorum, Neonatal herpes simplex virus (HSV), Mental Retardation and delayed milestones are some of the serious problems in the child which can be prevented by treating the mother during pregnancy.

Identifying the etiological agent through laboratory methods and then administering appropriate treatment to the patient will help to

prevent such complications. But as this is not always possible in sexually transmitted infections patients due to loss of follow up, in many cases an empirical diagnosis is made and syndromic management is given to the patient and contacts. This syndromic management being presumptive can sometimes be inaccurate leading to treatment failure.

On the other hand, many women with vaginal discharge self-treat with over-the-counter drugs making the pathogen more resistant to treatment. So, it is of great importance to determine the causative agent and give appropriate treatment to the patient to avoid complications and prevent unnecessary over-usage of drugs.

Abnormal vaginal discharge being easily diagnosable and treatable, these serious complications can be avoided to a great extent. This study is undertaken to address these issues and determine the etiological diagnosis of abnormal vaginal discharge to give appropriate treatment to the patient.

AIMS
AND
OBJECTIVES

AIM

To determine the infectious etiology of abnormal vaginal discharge in women of reproductive age group attending Gynaecology OPD in Govt. RSRM Hospital and Sexually Transmitted Diseases OPD in Govt. Stanley Medical College

OBJECTIVES:

1. To determine the pathogenic organism causing abnormal vaginal discharge in women of reproductive age group attending Gynaecology OPD in Govt. RSRM Hospital and Sexually Transmitted Diseases OPD in Govt. Stanley Medical College.

2. To make a comparison between the etiologic agents in the two groups i.e. between women in general population having abnormal vaginal discharge and women in High Risk Groups (HRG) having abnormal vaginal discharge.

3. To study the demographic profile and predisposing factors in women presenting with abnormal vaginal discharge.

4. To make a comparison between Amsel's criteria and Nugent's criteria for Bacterial Vaginosis.

5. To determine the prevalence and role of Candida albicans and Non albicans Candida species in Vulvo-Vaginal Candidiasis.

REVIEW OF LITERATURE

REVIEW OF LITERATURE:

EPIDEMIOLOGY:

Sexually transmitted diseases (STD) refers to clinical syndromes caused by organisms that are acquired or transmitted through sexual contact.[5] Sexual transmission occurs when one person harboring the infectious agent engages in sexual practice with another person who is susceptible to that agent.

The term STD has widely replaced the earlier term venereal diseases (VD), a name derived from Venus, the goddess of love of the ancient Romans. STDs have been known to mankind from time immemorial. The first ever recorded case of STD occurred in ancient Greece, where the Herpes virus got its name meaning "to creep or crawl". Shakespeare referred to Herpes as "blister plagues", which shows the extent of the epidemic.

Abnormal vaginal discharge as a separate disease was described in ancient Thai medicine called ‘Muttakit’ and categorized into 4 groups resembling Bacterial vaginosis, cervicitis, carcinoma of cervix or endometrium with herbal cures for each group[6].

Hippocrates described vaginal discharge “leucorrhoea” as a consequence of anatomic characteristics of women and even distinguished different forms of discharge as albus, rufus, ruber, and niger. In ancient Babylon and Rome, there is documentation of both male and female prostitution. Homosexuality was also present in those days as is evident from a number of paintings and pottery.

In the Holy Bible, there is mention of gonorrhea and syphilis showing that these diseases are rampant from ancient times. Before the days of microscopy, Gonorrhoea was also mistaken to be syphilis as they both produced very similar symptoms and were often silent. These diseases were called ‘The great imitators’ by William Osler, an eminent physician and they were rampant during the times of World Wars I and II. Syphilis and Gonorrhoea were treated with orally ingested mercury before antibiotics were discovered leading to many complications.

‘Malariotherapy’ was considered as a treatment for Syphilis in olden days, in which malaria was induced by mosquito bites as a therapy in patients suffering from syphilis.

In India, Vedas are the oldest sacred books and the Atharva Veda contained information on venereal diseases, treatment of sexual dysfunctions and aphrodisiacs. There has also been clear documentation

of female genital infections with herbal cure in an ancient ayurvedic book called Bhavaprakasha. The Charaka Samhita has mention of different genital diseases, genital discharge and their therapy.

STD ranks one among the five major problems in developing nations according to the WHO. In 1912, the chair of the US committee, Prince Morrow looking into the problem of STD said “It is a conservative estimate that one-eighth of human suffering comes from this source alone”.

In 1996, WHO global estimates for the four major venereal diseases suggested that there were more than 333 million new cases of syphilis, gonorrhoea, chlamydia, and trichomoniasis in adults aged 15-49 years.[7] 75-85% of them were from the developing countries and the highest number of new infections were from South and South-East Asia (45.6%), followed by sub-Saharan Africa (19.7%), Latin America and Caribbean (10.9%).

Though adolescents and young adults (15-24 years) make up only 25% of the sexually active population, they represent around 50% of all newly acquired STDs.

It is estimated that reported cases of STDs represent only 50%-80% of reportable STD infections, reflecting on limited screening and low disease reporting. Individuals infected with STDs are 5-10 times more likely than normal individuals to acquire or transmit HIV through sexual contact.

According to CDC, during 2014–2015 the rate of reported gonorrhea increased 18.3% among men and 6.8% among women. In 2015, a total of 23,872 syphilis cases were reported, and the national primary and secondary syphilis rate increased to 7.5 cases per 100,000 population, a 19% increase from 2014.

Women are more susceptible to contact STD than men due to anatomical, hormonal, physiological and immunological factors. The problem is further complicated in women by the social stigma in seeking healthcare for STD.[8]

STDs in women are divided into ulcerative and non-ulcerative types. Abnormal vaginal discharge is the most common among the non-ulcerative STD. The prevalence of abnormal vaginal discharge is found to be 30% in normal population[9] and around 50% in commercial sex workers.[10]

A Female Sex Worker by definition is a woman who provides sexual favours in exchange for immediate cash or in kind returns. These sex workers are the core group and their clients form the bridge group for STD transmission to the general population.

ANATOMY OF THE VAGINA:

The perineum is a diamond-shaped region bounded by the symphysis pubis anteriorly, coccyx posteriorly, and ischial tuberosities laterally. The external female genital organs are vulva (labia majora, labia minora, clitoris, mons pubis, skene's glands, bartholin's glands), vagina and urethra. The internal genital organs include cervix, uterus, fallopian tubes and the ovaries. Vagina is a fibromuscular tube that is about 9 cm long anteriorly and 11.5 cm long posteriorly. It forms a cuff around the lower part of cervix and forms 4 fornices, an anterior, posterior and two lateral fornices. The vaginal wall has three layers[11]:

(1) The mucous membrane- stratified squamous non keratinized epithelium and an underlying lamina propria of connective tissue;

(2) The muscular layer- composed of smooth muscle fibers disposed both longitudinally and circularly;

(3) Adventitial layer- a dense connective tissue that fuses with the surrounding fascia.

NORMAL PHYSIOLOGICAL VAGINAL DISCHARGE:

There are no sweat, sebaceous or other types of glands in the vaginal wall. The fluid production is due to transudation across the vaginal wall. Physiological discharge also comprises secretions of the endocervix and the Bartholin's gland along with cells that are shed from the vaginal walls. These secretions are affected by hormonal changes during the menstrual cycle. The normal vaginal discharge is a physiologically important biomass. It is a white or clear discharge that varies with the menstrual cycle and does not cause discomfort. The physiological discharge is increased during sexual arousal and pregnancy.

Physiological pH varies in different age groups. At the time of birth, vagina of the female baby is colonized by anaerobic and aerobic bacteria acquired during passage through the vagina. The epithelium of newborn’s vagina is rich in glycogen due to influence of maternal estrogens. This results in a low pH (3.7-6.3).

Weeks after birth the epithelium becomes atrophic and devoid of glycogen, the pH rises to 6-8 (remains so till puberty) and the predominant flora are Gram-positive cocci and bacilli.

At puberty, there is proliferation of vaginal epithelial cells and glycogen content in the cells increases due to influence of oestrogen. Vaginal cells containing glycogen are shed into the lumen of the vagina. As the cells are autolysed, glycogen is converted to glucose, serving as an energy source for lactobacilli. Lactobacilli convert glucose to lactic acid, which results in a normal vaginal pH of 3.5 to 4.6 (acidic). This is the normal vaginal environment, which inhibits the growth of pathogens.

AGE	**Newborn to neonate**	**Neonate to puberty**	**Puberty to menopause**	**Pregnancy**	**Post menopause**
Ph	3.7 - 6.3	6 - 8	3.5 - 4.6	4 - 4.2	6 - 7.5

Lactobacilli also produces hydrogen peroxide, which is bactericidal when combined with physiologic amounts of myeloperoxidase and chloride. When the normal Lactobacillus-dominated vaginal microbiota is lost, the chance of infection on exposure to sexually transmitted pathogens increases. The risk for infections in association with pregnancy

and gynecologic surgery is also increased when normal vaginal microbiota is altered[8].

ABNORMAL VAGINAL DISCHARGE (AVD):

AVD is defined as any one of the three presentations:

1) Excessive vaginal discharge not associated with menstruation

2) Offensive or malodourous discharge

3) Yellowish mucopurulent discharge

Abnormal vaginal discharge can be due to infectious or non-infectious causes.

CAUSES OF ABNORMAL VAGINAL DISCHARGE:

INFECTIOUS CAUSES:

VAGINITIS:

1. Bacterial Vaginosis

2. Trichomoniasis

3. Candidiasis

4. Aerobic gram positive bacterial infection (Staphylococcus spp., Streptococcus spp., Enterococcus spp.)

CERVICITIS:

1. Gonorrhoea

2. Chlamydiasis

3. Viral cervicitis due to genital herpes(HSV) or genital warts(HPV)

4. Syphilis

5. Tuberculous cervicitis

NON INFECTIOUS CAUSES:

1. Gynaecological conditions: (cervical polyp, cervical ectropion, Cervical Intraepithelial Neoplasia (CIN), post operative or post radiation leucorrhoea, fistulae)

2. Foreign bodies: (Intra Uterine Contraceptive Devices IUCD, retained tampons, retained tissues post partum)

3. Chemical irritants: (perfumed soaps, antiseptics, douches, lubricants, spermicides)

4. Hormonal imbalance

5. Medications: Oral Contraceptive Pills (OCPs), oestrogen creams.

BACTERIAL VAGINOSIS (BV):

Bacterial vaginosis is one of the most prevalent causes of reproductive tract infections among women of childbearing age. It is also known as anaerobic vaginosis. It is a polymicrobial syndrome. BV results from the replacement of the normal vaginal flora (Lactobacillus sp. like L.crispatus, L. jensenii, L. gasseri, L.iners) with a mixed flora consisting of Gardnerella vaginalis, Mobiluncus sp., anaerobes like Prevotella and Porphyromonas, Bacteroides ureolyticus, Fusobacterium nucleatum, peptostreptococcus and genital mycoplasmas.

EPIDEMIOLOGY:

Gardner and Dukes in 1955 described ‘Haemophilus vaginalis vaginitis’ as a sexually transmitted disease as the causative agent was found in male contacts of the patients as well. BV is considered to be the most common etiology of abnormal vaginal discharge throughout the world. Incidence is higher with increasing years since marriage, parity of greater than two and lower socio-economic status. In rural South India, the prevalence among reproductive age group women was found to be

20%[12]. In a study of antenatal women from Chennai, 38% of symptomatic women and 16% of asymptomatic women were found to have BV.[13]

PATHOGENESIS:

It is hypothesized that inadequate IgA response against Gvh (a hemolytic toxin by Gardnerella vaginalis) increases risk of BV. Presence of hydrolytic enzymes like sialidase and protein dipeptidase lyse mucin and help in adherence of bacteria. Production of amino acids by anaerobes and ammonia by Prevotella is also said to increase Gardnerella vaginalis growth.

Vaginal mucosa and vulva appear normal as the inflammation of the vaginal epithelium in the form of leucocytes is seldom seen and so it is termed vaginosis instead of vaginitis.

CLINICAL FEATURES:

BV manifests as a non-viscous, white, homogenous, malodorous, non inflammatory discharge which smoothly coats the vaginal walls causing pruritis, pain during coitus and lower abdominal pain. Patients may complain of foul odour immediately after intercourse due to

alkalination of vaginal secretion by semen producing volatile polyamines.[14]

DIAGNOSIS:

Self-diagnosis of bacterial vaginitis is usually not correct. In a study of 552 women aged 16 years or older, only 3–4% of women could accurately identify the symptoms associated with BV. So the diagnosis is to be made based on clinical and laboratory methods.

AMSEL'S CRITERIA:

Amsel's criteria for Bacterial Vaginosis is based on clinical findings and has four criteria. When at least three out of these four criteria are fulfilled, it is suggestive of Bacterial Vaginosis:

1. A thin homogeneous, white, uniformly adherent vaginal discharge without granular material;

2. Elevation of the pH of vaginal fluid above 4.5; This has the highest sensitivity but least specificity.

3. Development of a dead-fish odor after mixing vaginal fluid with 10% KOH;

4. “Clue cells” on microscopic examination of vaginal fluid. Clue cells are epithelial cells heavily coated with bacteria sufficient to obscure the cell borders. Detection of clue cells is the single most useful procedure for diagnosis of BV. [15]

Vaginal pH has the greatest sensitivity of the four criteria but lowest specificity.[14] Detection of ‘Clue cells’ is the single most useful and specific criterion to diagnose BV and when other three clinical criteria are fulfilled in absence of ‘clue cells’ the diagnosis of BV based on Amsel’s criteria becomes questionable.

Gram stain of normal vaginal fluid shows a predominance of lactobacilli with normal epithelial cells, whereas the Gram stain of vaginal fluid from a woman with BV shows a decrease or absence of lactobacilli and a predominance of Gram-variable cocco-bacilli.

Nugent’s criteria uses gram stained smears of vaginal discharge to diagnose Bacterial Vaginosis. Various morphotypes are identified and graded according to Nugent’s scoring system. Lactobacilli type (large gram-positive rod), Gardnerella type (small gram-negative or gram variable rod) and Mobiluncus type (Curved negative/variable rod) are the morphotypes included.

A score of 0-3 is normal vaginal flora, 4-6 is considered intermediate and 7 to 10 is reported as Bacterial Vaginosis. Intermediate flora also needs to be intimated to the clinician as in 32% of cases, intermediate flora progresses to Bacterial Vaginosis.

NUGENT'S CRITERIA:

BACTERIAL MORPHOLOGICAL TYPE	**SCORE**				
	NONE	**1+**	**2+**	**3+**	**4+**
LACTOBACILLI TYPE(Large Gram-positive rod)	4	3	2	1	0
GARDNERELLA TYPE(Small Gram-negative/variable rod)	0	1	2	3	4
MOBILUNCUS TYPE (Curved negative/variable rod)	0	1	2	3	4

SCORE KEY:

<1/oil immersion field	1+
1-5/ oil immersion field	2+
6-30/ oil immersion field	3+
>30/ oil immersion field	4+

INTERPRETATION OF NUGENTS'S SCORE:

NUGENTS'S SCORE	INTERPRETATION
0-3	NORMAL
4-6	INTERMEDIATE-to be repeated later
7-10	BACTERIAL VAGINOSIS

Nugent's Scoring has sensitivity of 86-89% and specificity of 94-96% when compared to Amsel's criteria.

HAYS/ISON SCORING is based on gram staining to estimate ratio of observed monotypes rather than exact number of bacteria. This has 5 categories:

Group 0 : no bacteria at all

Group 1 : normal

Group 2 : intermediate

Group 3 : Bacterial Vaginosis

Group 4 : numerous gram positive cocci present

SCHMIDT'S SCORING of wet mount preparations is validated for diagnosis of BV at primary health care level. It is similar to Nugent's scoring but uses wet mount preparation instead.

Though clinical methods like Amsel's criteria are useful for bedside diagnosis and treatment, Gram-stained smear grading method appears to be more accurate.

SEROLOGIC METHODS:

Gas liquid chromatographic identification of fatty acids showing ratio of succinate to lactate peaks of more than 0.4 is highly suggestive of Bacterial vaginosis. Rapid nucleic acid hybridization test, proline aminopeptidase activity, amine test are other tests used. But the sensitivity of serologic tests in diagnosis of BV is poor.

TREATMENT:

National AIDS Control Organisation NACO (2004) regimen - T. Metronidazole 400 mg orally twice daily for 7 days or 2 gm orally single dose or T. Tinidazole 2 gm orally as a single dose.

TRICHOMONIASIS:

Trichomonas vaginalis is a pathogenic protozoan parasite of the human urogenital tract. It is transmitted by sexual route and causes vaginitis in women and urethritis in men. A proportion of infections are also asymptomatic. According to the World Health Organization (WHO)

trichomoniasis causes more than half of all curable sexually transmitted infections (STI) worldwide[16].

HISTORY:

T. vaginalis was first described in 1836 by French physician Alfred Donné. He observed the organisms in a preparation of fresh vaginal discharge and regarded it to be a commensal of vaginal tract. But in the 1940s, T. vaginalis was established as an etiologic agent of vaginitis as it fulfilled the Koch's postulates.

EPIDEMIOLOGY:

In general, T. vaginalis is highly prevalent among sexually active women. Among women presenting with vaginal complaints, the reported prevalence of trichomoniasis can be as high as 75%[17]. However, approximately 50% of infections with T. vaginalis may be subclinical in presentation[18].

T. vaginalis is transmitted almost exclusively by sexual intercourse. Although nonsexual transmission by contaminated fomites may explain reports of trichomoniasis in a few patients, such as sexually mature virgins, the data suggest that non-sexual transmission of T. vaginalis is rare.

Three species are found in humans- Trichomonas vaginalis is a parasite of the genitourinary tract, T. tenax is found in the oral cavity, Pentatrichomonas hominis is seen in large intestine. Trichomonads exist only in the motile, vegetative trophozoite form, and true cysts stages have not been described. They are primitive eukaryotes and cannot synthesize macromolecules de novo.

PATHOGENESIS:

Trichomonas vaginalis produces many proteolytic enzymes involved in cytotoxicity and haemolysis. Both contact dependent and independent mechanisms are seen. It has adhesion proteins AP 23, 33 and 65 using which it attaches to epithelial cells. They attach not only to epithelial cells but also to extracellular matrix substances like fibronectin and laminin.

The Cell detaching factor (CDF) which has trypsin like activity also acts as a virulence factor. Some women become symptomatic after menstruation, suggesting a role for iron in the pathogenesis of trichomoniasis[19]. Trichomonas evades immune response by molecular mimicry, its ability to coat itself with host proteins and complement mediated destruction.

CLINICAL PICTURE:

The spectrum varies from asymptomatic disease to severe vaginal infection. However, vaginal discharge can be seen in more than 50% of patients. Vaginitis is the most common presentation followed by infection of the urethra, Bartholin's gland, Skene's gland and endocervix.

Trichomoniasis is characterized by malodorous greenish-yellow frothy discharge with intense itching. Dyspareunia, burning micturition and abdominal pain may be present. Colpitis macularis also called 'Strawberry cervix' is due to microscopic punctate hemorrhages over the cervix. T. vaginalis is associated with Bacterial vaginosis- like vaginal flora showing reduced lactobacilli and colonization with Gardnerella vaginalis[20].

DIAGNOSIS:

Direct microscopic examination of wet mount preparation:

Diagnosis by wet mount is by observing viable active trophozoites with characteristic twitching type of motility. Trichomonads are around 15 μm in size, pyriform in shape with anterior tuft of flagella and a lateral undulating membrane. Wet mount microscopy is about 50–70% sensitive in women with trichomoniasis [21]. But since the parasite loses viability in normal saline if left more than 20 minutes at room temperature[22], Wet

mount examination is to be done as early as possible. Samples should not be centrifuged before processing as the fragile flagella are detached during centrifugation. The parasite can be stained by various staining methods like Gram stain, Giemsa stain, periodic acid-Schiff, Papanicolaou , acridine orange, fluorescein, and immunoperoxidase staining.

CULTURE:

Culture remains the gold standard for diagnosing trichomoniasis among women[23]. It is more sensitive than the wet mount examination as it can detect as low as 300-500 trophozoites/ml. But the time duration required is a minimum of 2 days to a maximum of 7 days. The culture media used are either semi-solid or liquid media. The most commonly used media are Diamond's media, Modified Diamond's media, CPLM (Cysteine Peptone Liver Maltose) media, modified CPLM media, In-pouch Trichomonas media, Kupferberg media, Feinberg Whittington media etc. A combined approach of wet mount examination followed by culture if microscopy is negative is very useful[24].

CELL CULTURE:

It is superior to both wet mount microscopy and regular culture methods as it can pick up trophozoites as low as 3/ml.

RAPID DIAGNOSTIC TESTS:

Oligonucleotide probe tests, immuno-chromatographic assays that detect trichomonas antigens in vaginal swabs and PCR assays have been developed. Rapid tests may be useful in settings where culture and microscopy are not available.

TREATMENT:

NACO (2004) regimen Metronidazole 500 mg orally twice daily for 7 days or 2 gm orally single dose/ Tinidazole 2 gm orally as a single dose.

CANDIDIASIS:

Candidiasis is the most common fungal disease in humans affecting skin, mucosa, nails and internal organs. It is also called Moniliasis. It is caused by a group of yeasts belonging to genus Candida with prototype species Candida albicans. Several non-albicans Candida species have been described and their incidence has been on the rise in recent days.

Women having Vulvo Vaginal Candidiasis (VVC) present with a spectrum of manifestations ranging from asymptomatic colonisation to severe acute symptomatic infection. Certain patients may develop mainly

vulvar symptoms like soddening of skin and vulvar pruritis instead of vaginal manifestations.

Candida species causing Vulvo-Vaginal Candidiasis:

- Candida albicans
- Candida tropicalis
- Candida krusei
- Candida glabrata
- Candida haemulonii
- Candida guilliermondii
- Candida parapsilosis
- Candida lusitaniae
- Candida kefyr (formerly C. paratropicalis)
- Candida dubliniensis

Around 85% of candidal strains cultured from the vagina are Candida albicans.[25] The rest are non-albicans, the commonest of which is C. glabrata. Non-albicans vaginitis is not clinically distinguishable from vaginitis caused by C. albicans but it is more resistant to therapy. The use of single dose oral anti-fungal regimens and over-the-counter anti-mycotics, are responsible for this increase.

PATHOGENESIS:

Candida colonize the vaginal epithelium after adhering to it. The yeast adhesin is seen in surface manno-protein. Germination of yeast cells increases colonization and invasion of tissue. Ibrahim *et al.*, have strongly implicated phospholipases as a virulence factor in the pathogenesis of *C. albicans*.[26] Candida usually gain access to vagina from the adjacent perianal region.

RISK FACTORS:

Host factors include immunodeficiency (HIV), pregnancy, immunosuppressive drugs, chemotherapy, uncontrolled Diabetes, corticosteroids therapy, Oral Contraceptive Pills (OCP), broad spectrum antibiotic therapy, Hormone Replacement Therapy (HRT), oro-genital sex practices etc.

Progesterone is shown to have suppressive effect on anti-Candida action of neutrophils and estrogen reducing the ability of vaginal epithelial cells to inhibit candida colonization. This could explain the higher incidence of Candidiasis in pregnancy, OCP usage and HRT.

CLINICAL PICTURE:

Vaginal discharge is characteristically curdy, cottage cheese like in appearance but can vary from watery to homogenous thick discharge. However, vaginal discharge is not always present and is mostly minimal. Vaginal soreness, burning, dysuria, dyspareunia are major complaints and symptoms are more in the week preceding menstruation.

On examination vulvar erythema and pustulo-papular lesions may be seen. VVC is divided into uncomplicated and complicated disease based on host or microbial factors and these factors have a profound impact on outcome of therapy.

FACTORS	UNCOMPLICATED VC	COMPLICATED VVC
Frequency	<4 episodes per year	≥ 4 episodes per year
Severity	Mild to moderate	Moderate to severe
Microscopy	Pseudophypae /hyphae seen	Budding yeast only
Host	Healthy	Pregnant females, diabetes, immune compromised patients.

DIAGNOSIS:

Direct microscopy:

Wet mount microscopy of vaginal discharge with a drop of normal saline shows budding yeast cells with pseudohyphae.

This is better identified by a 10% potassium hydroxide (KOH) preparation which is more sensitive in identifying germinated yeast cells (sensitivity of 65–85%). KOH is a strong alkali helping in clearance of epithelial cells and digestion of keratin material, thus the fungal elements can be clearly seen. This clearing can be hastened by slightly heating the KOH mount over a flame.

Vaginal pH in VVC is almost normal (4.0–4.5) and if pH is in excess of 5.0 usually it is BV, trichomoniasis, or a mixed infection.

Culture methods:

Culture of vaginal discharge is the most sensitive and specific method for detecting Candida, but a positive culture alone cannot confirm VVC since 10–15% of normal asymptomatic women are colonized with Candida and are invariably culture positive. Positive culture results are to be correlated with direct microscopy showing high number of yeast cells and polymorphonuclear cells to confirm VVC.

There is no reliable serologic method or antigen-detection technique for diagnosing VVC.

SPECIATION OF CANDIDA:

Cultures positive for yeast cells can be differentiated into C. albicans and non albicans Candida (NAC) by the Germ Tube Test also called as Reynolds-Braude phenomenon. C. albicans and C. dubliniensis alone form germ tubes within 2 hours of incubation in serum. A germ tube is a lateral hyphal extension from the yeast cell without any constriction at the base.

CHROMagar is a newer culture medium that distinguishes different species of Candida by a characteristic colour for each species.[51] This is based on detection of specific enzymatic activities by addition of substrates of fluorochromes to the media.[27]

Candida species	**Colour on CHROMagar**
Candida albicans	Light green to bluish green
Candida tropicalis	Dark blue
Candida glabrata	Pink to purple
Candida parapsilosis	Cream coloured
Candida dubliniensis	Dark green
Candida krusei	Pinkish to purplish

Triphenyl Tetrazolium Chloride (TTZ) is another chromogenic medium that differentiates Candida species based on the colour produced.

Culture of suspected Candida strains are inoculated in Corn Meal Agar or Rice Starch Agar by Dalmau technique for 48-72 hours to show production of chlamydospores in C.albicans and C. dubliniensis. The tween present in corn meal agar and the sub surface inoculums provides a low surface tension favouring development of hyphal and pseudohyphal forms, chlamydoconidia and blastoconidia.

Candida species	**Morphology on Corn meal Agar**
Candida albicans	Terminal chlamydospores
Candida tropicalis	Branching pseudohyphae and blastoconidia
Candida glabrata	No pseudohyphae, only blastoconidia
Candida parapsilosis	Curved pseudohyphae and blastoconidia
Candida dubliniensis	Terminal chlamydospores
Candida krusei	Pseudohyphae and blastoconidia resembling crossed match sticks

Of late, tobacco agar and casein agar are reported to selectively induce chlamydospore formation in C. dubliniensis differentiating it from C. albicans. Other species of Candida can be differentiated based on pseudohyphae, arthrospores and blastospores formation.

Carbohydrate assimilation tests are also used to speciate Candida isolates based on the sugars utilised.

ANTI-FUNGAL SUSCEPTIBILITY TESTING: (AFST)

Disc Diffusion Test: MHA with 2% glucose and GMB (Gomorri Methylene Blue) medium

According to the CLSI M44-A2, Mueller Hinton Agar (MHA) is considered to a good choice for AFST procedure. When supplemented with glucose to a concentration of 2%, it provides for fungal growth. The addition of Methylene Blue Dye (GMB) to a concentration of 0.5 µg/ml enhances the zone edge definition.[28] The pH is adjusted between 7.2 and 7.4

Inoculum is standardised to 0.5 McFarland standard and after placing anti-fungal discs plates are incubated at 35C for 24 hours.

Interpretive zone diameter:

Drug tested	RESISTANT	S-DD(Susceptible Dose Dependant)	SENSITIVE
Fluconazole 25 µg	≤14	15-18	≥19
Itraconazole10 µg	≤13	14-22	≥23
Voriconazole 1 µg	≤13	14-16	≥17
Amphotericin B 20 µg	≤10	10-14	≥15

Candida krusei shows intrinsic resistance to Fluconazole and should not be interpreted with this scale for Fluconazole. Other non albicans Candida also show varying degrees of azole resistance. This highlights the importance of routine speciation and anti-fungal susceptibility testing for all Candida cultures.

Minimum Inhibitory Concentration by Microbroth dilution:

According to M27-A3[29], MIC testing is done using Rosewell Park Memorial Institute medium (RPMI-1640 medium) with glutamine without bicarbonate. It is supplemented with 0.2% glucose and buffered to the pH of 7.0. Inoculum is standardised to 0.5 McFarland standard added to RPMI medium in a microtitre plate. To this, liquid preparation of drugs in the concentration to be tested are added in doubling dilution. The plate is incubated at 35C for 24 hours and interpretation done using a reading mirror.

The MIC for azoles is the lowest concentration with an optical score of 2. (prominent decrease in turbidity). MIC for Amphotericin B is the lowest concentration with score of 0 (optically clear).

TREATMENT:

Fluconazole 150 mg single dose therapy is indicated in uncomplicated VVC and intensive regimen is recommended for complicated VVC with longer duration of treatment and higher dosage of drugs.

AEROBIC VAGINITIS:

In 2002, Donder suggested the term Aerobic Vaginitis(AV) and described it as being caused by enteric commensal flora like Streptococcus agalactiae, Escherichia coli, Enterococcus spp., Staphylococcus spp. AV was determined based on Donder's criteria:

- Enhanced yellowish vaginal discharge
- pH value >/= 5
- Increased number of leucocytes >10
- Negative whiff test
- Absence of lactobacilli
- Most commonly isolated organisms- Staphylococcus aureus, Escherichia coli, Enterococcus, Group B Streptococcus etc.

Modified Donder's grading of gram stained smears for diagnosis of Aerobic Vaginitis:

Score	Lacto bacillary grade	No. Of leucocytes	Proportion of toxic leucocytes	Background flora	Proportion of parabasal cells
0	I, IIA	</=10/ HPF	None or sporadic	Unremarkable	none
1	II B	>10/HPF or </=10 /EPI CELL	</= 50% of leucocytes	Small coliforms	</= 10%
2	III	>10 /EPI CELL	>50% of leucocytes	Cocci in chains	>10%

SCORING:

<3 : Normal

3-4 : Slight aerobic vaginosis

5-6 : Moderate aerobic vaginosis

6-10 : Severe aerobic vaginosis

CERVICITIS:

Cervicitis is the inflammation of the cervical epithelium. It can be gonococcal cervicitis or non-gonococcal cervicitis.

GONOCOCCAL CERVICITIS:

Gonorrhoea is derived from – ‘gonos’ meaning ‘seed’ and ‘rhoea’ meaning ‘flow’. It was thought that syphilis and gonorrhoea were caused by same organism until Neisseria gonorrhoea was identified as the causative organism of gonorrhoea in 1879 by Albert Neisser.

Nowadays gonorrhoea commonly referred to as ‘the Clap’ or ‘the Drip’ is presently the second most commonly reported notifiable disease in the US. The WHO estimates worldwide, 78 million people get gonorrhoea each year with majority of cases affecting the young under the age of 25.

In women, the endocervical canal is the most common site of gonococcal infection but only 50% of infected women have symptoms. Urethral colonization is also seen in 70–90% of infected women, but is not common in the absence of cervicitis. Infection of the skene’s and bartholin glands is also seen. In most patients, there is mucopurulent cervical discharge, edema and erythema of cervix and mucosal bleeding on swabbing the endocervix.

PATHOGENESIS:

Primary infection occurs in columnar epithelium of urethra, cervix, conjunctiva etc. Penetration of cocci occurs through intercellular spaces and reaches the sub- epithelial tissue by third day followed by adherence, invasion and tissue damage due to lipo-oligosaccharides and peptidoglycans. In pre-pubertal girls, rarely primary infection may also occur in stratified squamous epithelium of vagina.

CLINICAL FEATURES:

The common symptoms in females are burning micturition, mucopurulant cervical discharge, erythema and edema of cervix which may bleed on touch. The rectal mucosa may be infected in 35-50% of women. Pharyngeal infection as a result of oro-genital sex can also occur.

DIAGNOSIS:

Microscopy:

Direct gram staining of endocervical secretion reveals numerous polymorphonuclear cells in gram negative intracellular diplococci. The diplococci are characteristically kidney bean shaped and around 0.8 µm × 0.6 µm in size. The specificity of gram stain is 95-97% in male urethral discharge but only 40-60% in endocervical discharge.[30]

CULTURE:

Culture is the most specific investigation and done on Modified Thayer Martin medium (MTM), New York City (NYC) medium, Chacko Nayar medium and Martin Lewis medium under 5-10% CO2 incubation.

MTM is incorporated with VCN supplement. (Vancomycin to inhibit gram positive bacteria, Colistin to inhibit gram negative bacteria and nystatin to inhibit fungal growth). Culture yields Neisseria gonorrhoea with a diagnostic sensitivity of 80–95% for promptly incubated samples.[30]

Confirmatory Test for Neisseria gonorrhoea:

RAPID CARBOHYDRATE UTILISATION TEST: (RCUT)

This is a reliable method to distinguish Neisseria gonorrhoea from other Neisseria spp. It is a non growth dependent method and tests both carbohydrate utilisation and beta-lactamase production. Carbohydrates routinely tested include glucose, maltose, sucrose and lactose.

Neisseria gonorrhoea utilizes only glucose and hence shows a color change for glucose alone.

Non culture methods like Nucleic acid amplification tests (NAATs) for diagnosing gonorrhoea are also available now.

COMPLICATIONS:

Salpingitis leading to PID, bartholin gland abscess, disseminated gonococcal infection, Gonococcal arthritis, meningitis, endocarditis, ophthalmia neonatorum are all complications of gonorrhoea.

TREATMENT:

The currently recommended therapy for uncomplicated gonorrhea by the CDC is a single i.m injection of ceftriaxone 125 mg or Cefixime 400 mg orally single dose along with a single 1-g oral dose of azithromycin.

Gonorrhoea was initially treated with sulphonamides and when resistance developed, Penicillin became the drug of choice. Within 10-15 years, penicillin resistance by Penicillinase producing Neisseria gonorrhoea (PPNG) had spread to a great extent and fluoroquinolones were the treatment widely used.[31] Since the spread of fluoroquinolone resistance in 2010, the cephalosporin antibiotics have been the foundation of treatment. The emergence of extended spectrum cephalosporin-

resistant ‘SUPERBUG’ gonococcus is highly detrimental, since there are very few antibiotic options left to treat these gonorrhoeas.

As of July 2017, there are three cases of totally untreatable gonorrhoea cases identified in which all known antibiotics were found to be resistant. This was caused by newly discovered H041 strain of Neisseria gonorrhoea. One of the cases was in France, one in Japan and one in Spain. This is an imminent threat to the community as there will be no antibiotic left to treat Gonorrhoea if this superbug spreads around the world.

DIFFERENTIAL DIAGNOSIS OF ABNORMAL VAGINAL DISCHARGE:

Diagnostic criteria	**Normal**	**Bacterial Vaginosis**	**Trichomonas vaginitis**	**Vulvovaginal candidiasis**
Discharge	White, thin, flocculent	Thin, white homogenous, grey	Yellow, green, frothy	White, curdy, "cottage cheese"
Vaginal pH	3.8 - 4.2	> 4.5	4.5	< 4.5 (usually)
Microscopic	Lactobacilli, epithelial cells	Clue cells, no WBC's	Trichomonads, WBC's >10/ hpf	Budding yeast, hyphae, pseudohyphae
Amine odor "whiff" test	Absent	Fishy	Fishy	Absent

SYNDROMIC MANAGEMENT (NACO):

As per NACO guidelines in 2007 any patient presenting with abnormal vaginal discharge is categorized into VAGINAL DISCHARGE SYNDROME and a pre-determined single dose drug combination is administered to collectively treat all probable causes of vaginal discharge. This is labeled the GREEN KIT and consists of 2 drugs:

- T. Secnidazole 2gm orally, single dose to treat BV and TV;
- T. Fluconazole 150 mg orally, single dose to treat Candidiasis.[11]

MATERIALS AND METHODS

MATERIALS & METHODS

STUDY PLACE:

1. Department of Microbiology, Government Stanley Medical College, Chennai.

2. Department of Obstetrics & Gynaecology, Govt. RSRM Lying-in Hospital, Chennai.

3. Department of Sexually Transmitted Diseases, Govt. Stanley Medical College, Chennai.

STUDY DESIGN:

Prospective study.

SAMPLE SIZE : 200 IN NUMBER.

STUDY PERIOD :

October 2016 to September 2017

INCLUSION CRITERIA:

1. Women of reproductive age group (18-45) belonging to normal population who present with abnormal vaginal discharge to the Gynaecology OPD in Govt. RSRM lying-in Hospital

2. Women of reproductive age group (18-45) belonging to high risk groups who present with abnormal vaginal discharge to the STD OPD in Govt. Stanley Medical College Hospital.

HIGH RISK GROUP

includes- Female Sex Workers

Females having multiple sexual partners

Females with high risk partners

Females with known venereal diseases

3. Women of reproductive age group who presented with other complaints like lower abdominal pain, infertility or irregular menstruation but had abnormal vaginal discharge on examination.

EXCLUSION CRITERIA:

1. Women who are not willing for examination
2. Pregnant women
3. Post-menopausal women
4. Women below the age of 18 and over the age of 45
5. Women on antibiotic course
6. Women on their menstrual period

STATISTICAL ANALYSIS :

Statistical Analysis was done using IBM-SPSS Statistics – 20 statistical package. Variables were analysed using student 't' test, Chi square test and Z test of proportions wherever necessary. The P value less than 0.05 ($P<0.05$) were treated as significant in two tail test.

ETHICAL CONSIDERATION:

This study has been approved by the Institutional Ethic Committee of Govt. Stanley Medical College and informed consent was obtained from the patient before inclusion in the study.

MATERIALS:

Sample Source: Cervico-Vaginal discharge

Materials Required: Sterile dacron swabs, sterile glass test tubes, clean glass slides, pH indicator strips(narrow range 3.5 to 6), chemicals and reagents like normal saline,10% Potassium hydroxide.

Media Required:

5% Sheep Blood agar

Modified Thayer Martin Medium

Cysteine Peptone Liver Maltose (CPLM)

Sabouraud's Dextrose Agar

CHROMagar for Candida

Corn Meal Agar

Gram staining reagents

Giemsa staining reagents

METHODS:

This study has been approved by the Institutional Ethic Committee of Govt. Stanley Medical College.

After informed consent from the patient is obtained, the patient details are collected and presenting complaints are noted.

The details about quantity of vaginal discharge, mode of onset, relation to menstrual cycle, duration, presence of itching/irritation (localized or generalized, provoked, intermittent, or chronic), colour, consistency, odour of discharge, associated abdominal pain, dysuria and dyspareunia are elicited.

The medical history elicited includes all of the usual gynecologic parameters, including menstrual history, pregnancies, contraception, sexual preference, past and current sexual relationships, vaginal hygiene practices (douching) and prior genitourinary infections. The patient is also asked about underlying medical conditions such as allergies, diabetes, malignancies, and immunodeficiency syndromes.

General examination of the patient is done and relevant systemic examination is done. The patient is then made to lie comfortably in supine position on an examining table in a covered cubicle.

Inspection of external genitalia is done; the pubic hair is examined for the presence of pthirus pubis or nits. The inguino-femoral area is palpated for adenopathy. Suprapubic and lower abdominal tenderness or masses are sought by palpation.

The patient is then asked to lie in lithotomy position and the external genitalia should be carefully inspected. The patient is asked to point to any areas of external itching, irritation, or other discomfort. The appearance of the vulval skin and vestibular mucous membranes is noted, with careful attention to ulcers or other discontinuity of the skin or mucosa, mucosal erythema, labial edema and visible secretions.

By spreading the labia with the gloved hand, the urethral meatus is examined for any discharge. The introitus and the internal surfaces of the labia minora are examined for lesions.

Under speculum examination, the vaginal mucosa and cervix are inspected for any erosion or ectropion and nature of vaginal discharge is noted.

Two high vaginal swabs are taken from the lateral fornices and one endo-cervical swab is taken under strict aseptic precautions. Finally, bimanual examination for adnexal tenderness and masses is done.

One high vaginal swab is used to inoculate 5% Sheep Blood Agar Plate (BAP), Sabouraud's Dextrose Agar (SDA) and Cysteine Peptone Liver Maltose (CPLM) medium. These are incubated at 37C.

The 2^{nd} high vaginal swab is used to make a wet mount with normal saline, a wet mount with 10% KOH, a smear for gram staining and the vaginal pH is tested using a pH indicator strip of range 3.5 to 6.0. The cervical mucus is avoided since it has a higher pH (pH 7.0) than the vaginal fluid.

The sample is then examined for odor (whiff test) by placing a drop on a microscope slide, adding a drop of 10% potassium hydroxide (KOH), and smelling the resultant mixture. Normal secretions have no odour and a fishy odor is indicative of BV. The Amsel's criteria is thus applied to diagnose BV.

The endo-cervical swab is first inoculated onto Modified Thayer Martin medium (MTM) and incubated at 37^0C in 5-10% CO_2 atmosphere by placing the streaked plates in a candle jar with a piece of moist cotton to provide moisture.

The swab is then used to make a smear for gram staining by gently rolling the swab in one direction onto a grease-free clean glass slide.

This uni-directional smearing technique helps to minimize distortion and breakage of polymorphonuclear leukocytes (PMNL) thus preserving the characteristic intracellular appearance of gonococci.

For Gram staining the smears were fixed with alcohol, flooded with methyl violet for one minute and washed in running tap-water and then flooded with Gram's iodine for one minute. The films were then decolourized with acetone, washed immediately with water and counter-stained with dilute carbol fuschin for one minute. The films were then washed with water and allowed to air dry.

OBSERVATION:

The wet mount is examined under high power with a bright-field microscope for presence of epithelial cells, clue cells, polymorphonuclear neutrophils (PMNs) and budding yeast cells with pseudo-hyphae. The relative numbers of epithelial cells and PMNs is noted.

The wet preparation is also scanned for trophozoites of Trichomonas vaginalis which are identified by their characteristic twitching motility.

The KOH mount is examined for budding yeast cells with or without pseudo-hyphae.

The gram-stained high vaginal smear is examined for presence of epithelial cells, clue cells, gram positive lactobacilli, gram variable cocco-bacilli, PMNs, budding yeast cells with pseudohyphae and is also scored according to Nugent's score for Bacterial Vaginosis.

The gram stained endocervical smear is examined for PMNs and intracellular gram negative diplococci which suggest gonorrheal cervicitis.

Presence of >30 PMN/ high power field with no gram negative diplococci is an indirect clue for the diagnosis of Chlamydial Cervicitis.

The BAP incubated at 37^0C is examined after 24 hours and any colony grown is identified by gram staining and biochemical reactions.

The SDA tube is examined for creamy-white colonies suggestive of Candida and gram stain is done. Any budding yeast cells isolated are subjected to germ tube test (Reynauld's braude phenomenon) and streaked on CHROMagar and Corn meal agar to speciate Candida.

The MTM incubated in candle jar is examined after 24-48 hours for tiny translucent water-drop like colonies.

Gram stain and oxidase test is done and if gram negative diplococci which are oxidase positive is seen, further confirmation is done using RCUT (Rapid Carbohydrate Utilisation Test) to confirm Neisseria gonorrhea.

The CPLM medium is examined for motile trophozoites of trichomonas by wet mount examination every alternate day for 7 days before it can be declared negative. The motile trophozoites found are fixed in smear and stained by Giemsa stain to study the morphology.

RESULTS

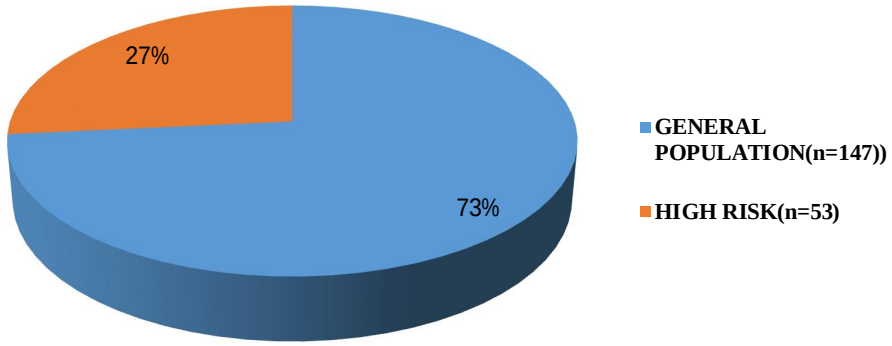
RESULTS

The total number of patients included in the study was 200 in which 147 patients belonged to general population from Gynaecology OPD and 53 patients belonged to High Risk Groups from STD OPD.

TABLE 1: DISTRIBUTION BASED ON RISK FACTORS:

STUDY POPULATION	NO. OF PATIENTS	PERCENTAGE
GENERAL POPULATION	147	73.5%
HIGH RISK GROUPS	53	26.5%
TOTAL	200	100%

CHART 1: STUDY POPULATION



Based on the presenting complaints the patients are classified into:

- Those who presented with abnormal vaginal discharge as the chief presenting complaint and
- Those who presented with other complaints like lower abdominal pain, infertility, dysuria etc. and were found to have abnormal vaginal discharge on examination.

TABLE 2:

DISTRIBUTION BASED ON PRESENTING COMPLAINTS

Chief presenting complaint	**General population**	**High-risk groups**	**Total**
Abnormal vaginal discharge	102(69%)	44(83%)	146(73%)
Infertility/ lower abdominal pain/ Irregular menstruation	45(31%)	9(17%)	54(27%)
Total	147	53	200

TABLE 3: DISTRIBUTION BASED ON AGE

AGE (YRS)	GENERAL POPULATION	HIGH RISK GROUP
18-29	58(39.5%)	18(33%)
30-39	61(41.5%)	25(47%)
40-45	28(19%)	10(20%)
TOTAL	147	53
Mean ±SD	31.6 ±7.2	32.2 ±6.9
Significance	t-value=1.62 degree of freedom=198 p value = 0.0534 p value > 0.05	

The study included menstruating women of reproductive age group from 18 years to 45 years. The most common age group in both the groups was 30-39 years. Mean age among general population was 31.6 ±7.2 and among high risk groups was 32.2 ±6.9. Since the p-value was >0.05, the two groups were similar in terms of distribution of age and thus a comparison can be made between them.

CHART 2: AGE-WISE DISTRIBUTION

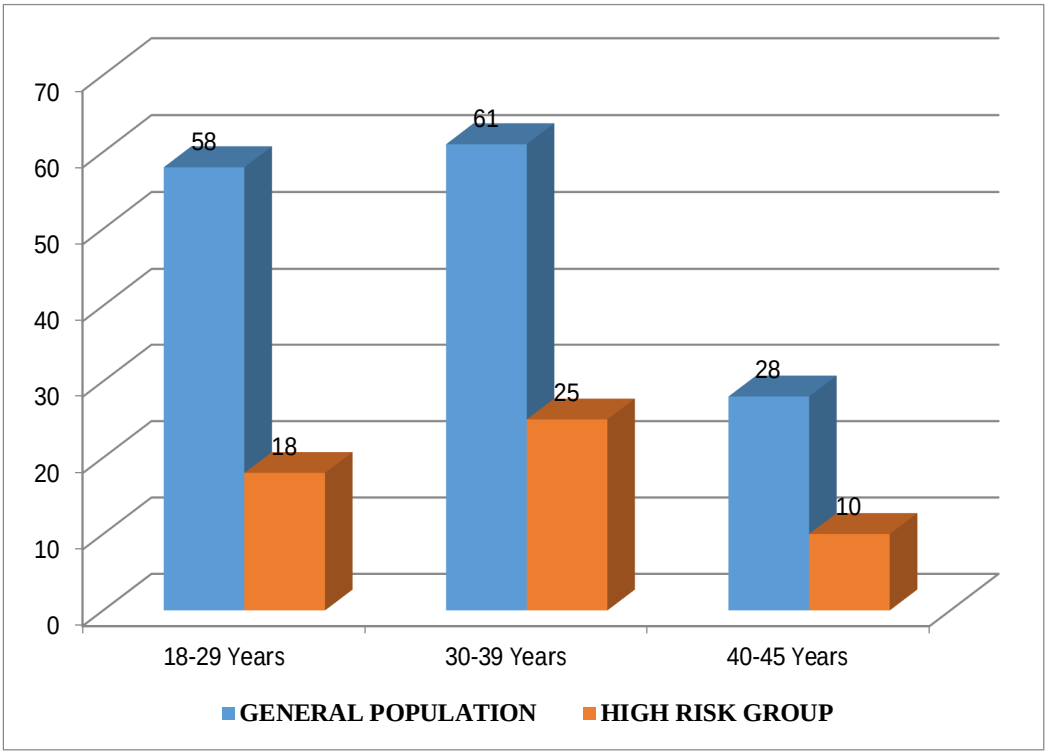


TABLE 4: DISTRIBUTION BASED ON MARITAL STATUS

MARITAL STATUS	GENERAL POPULATION	HIGH RISK GROUP
Unmarried	0	5(9.4%)
Married	136(92.5%)	24(45.2%)
Widowed	3(2.0%)	7(13.2%)
Separated	8(5.4%)	17(32.2%)
Total	147	53
Significance	$\chi^2 = 50.032$ P value <0.00001 P<0.05	

There is a significant difference between general population and high risk group based on their marital status ($p<0.05$). The percentage of separated and divorced women in high risk groups was significantly more than general population.

CHART 3: DISTRIBUTION BASED ON MARITAL STATUS

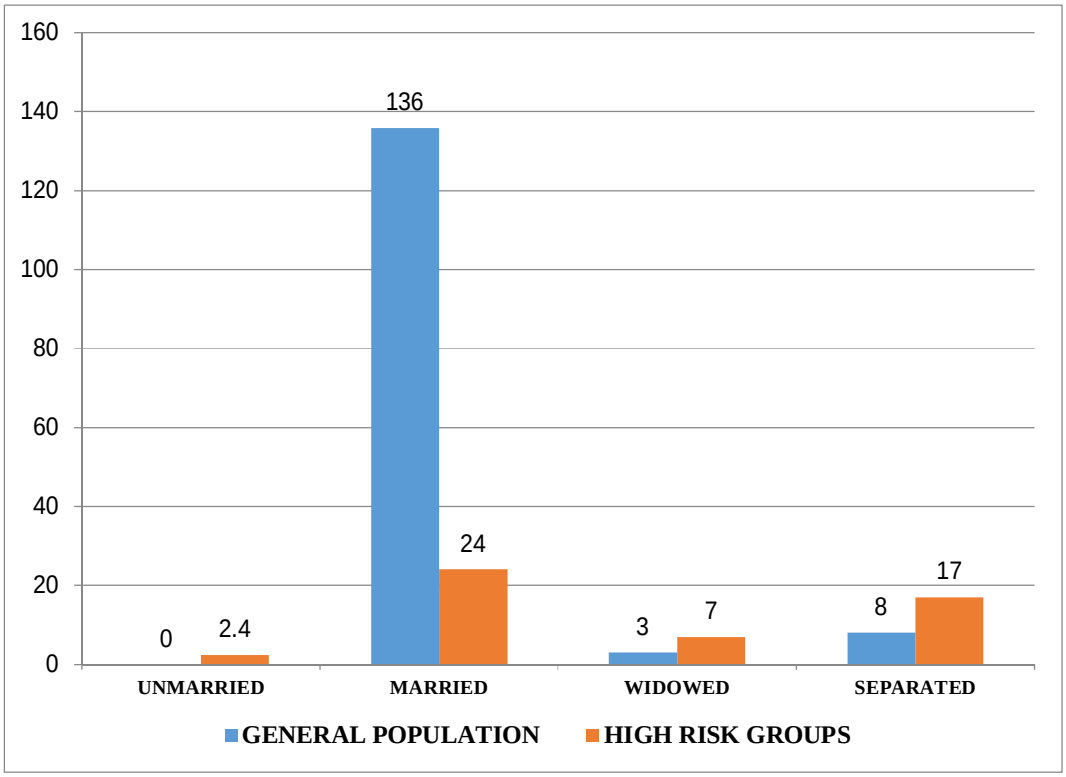


TABLE 5: DISTRIBUTION BASED ON EDUCATION

EDUCATION	GENERAL POPULATION	HIGH RISK GROUP
Illiterate	6(4.1%)	5(9.4%)
Primary school	23(15.7%)	13(24.6%)
Middle school	19(12.9%)	17(32.1%)
High school	46(31.3%)	11(20.8%)
Higher secondary	32(21.7%)	6(11.3%)
Degree	21(14.3%)	1(1.8%)
Total	147	53
Significance	χ^2 =4.25 Degree of freedom=5 P value = 0.514 p>0.05	

Most common education level in general population was high school education while in high risk group it was middle school level. The general population had a higher level of education but statistically the two groups did not show any significant difference (p>0.05).

CHART 4: DISTRIBUTION BASED ON EDUCATION

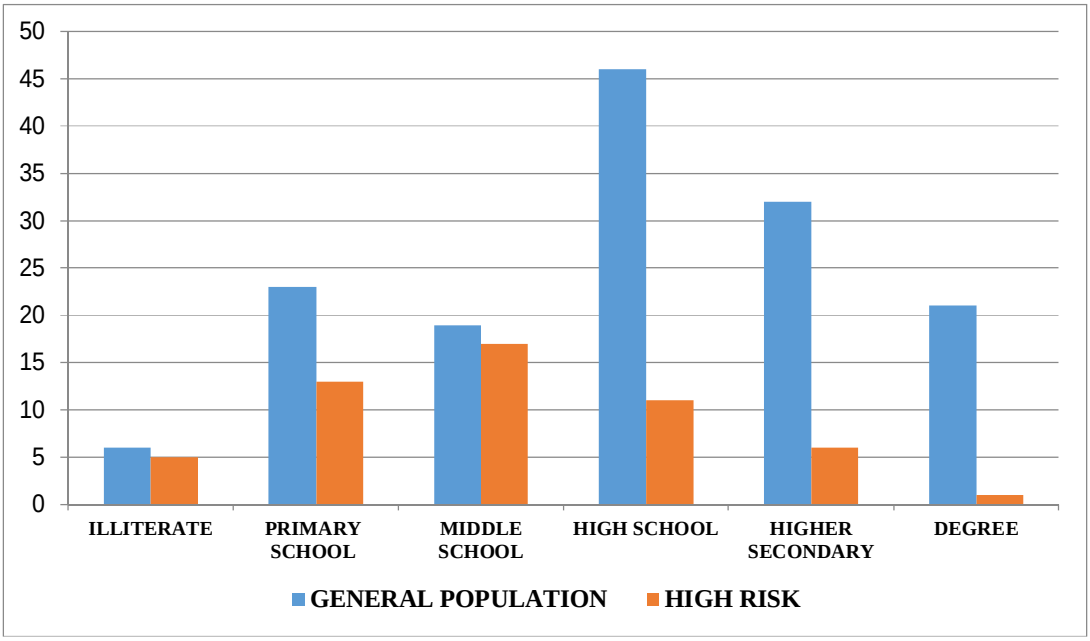


TABLE 6: DISTRIBUTION OF HIGH RISK PATIENTS BASED ON RISK FACTORS ASSOCIATED

RISK FACTOR	NO. OF PATIENTS IN HIGH RISK GROUP
Female sexual worker	18(33.9%)
Female having multiple sexual partners	17(32.1%)
Females with high risk partner	9(16.9%)
Female with other known venereal diseases	5(9.4%)
Female with past history of treated venereal diseases	4(7.5%)
Total	53

Female Sex Workers were the most common group among high risk women as they are mobilized by NGOs periodically to attend STD OPD for their sexual health.

TABLE 7: DISTRIBUTION BASED ON PARITY

PARITY	GENERAL POPULATION	HIGH RISK GROUP
Nulliparous	33(22.4%)	13(24.6%)
Women with single child	36(24.4%)	8(15.0%)
Multiparous	78(53.2%)	32(60.4%)
Total	147	53

Multiparous women were the most common among both the groups of women corresponding to the most common age group of 30-39 years.

TABLE 8: METHODS OF CONTRACEPTION USED

CONTRACEPTIVE	GENERAL POPULATION	HIGH-RISK GROUP
None	37(25.3%)	5(9.5%)
Barrier method(Condom)	10(6.8%)	19(35.9%)
Oral Contraceptive pills	19(12.9%)	9(16.9%)
Intra-Uterine Contraceptive Device	24(16.3%)	6(11.3%)
Permanent Sterilisation (Tubectomy)	57(38.7%)	14(26.4%)
Total	147	53

Permanent sterilisation by tubectomy was the most common contraceptive method used in general group corresponding to more number of multiparous women in this group. Barrier method by male Condom usage was the common mode of contraception in high risk group as it also serves to protect against STIs and is being promoted by NACO and NGOs among these groups.

CHART 5: METHODS OF CONTRACEPTION USED

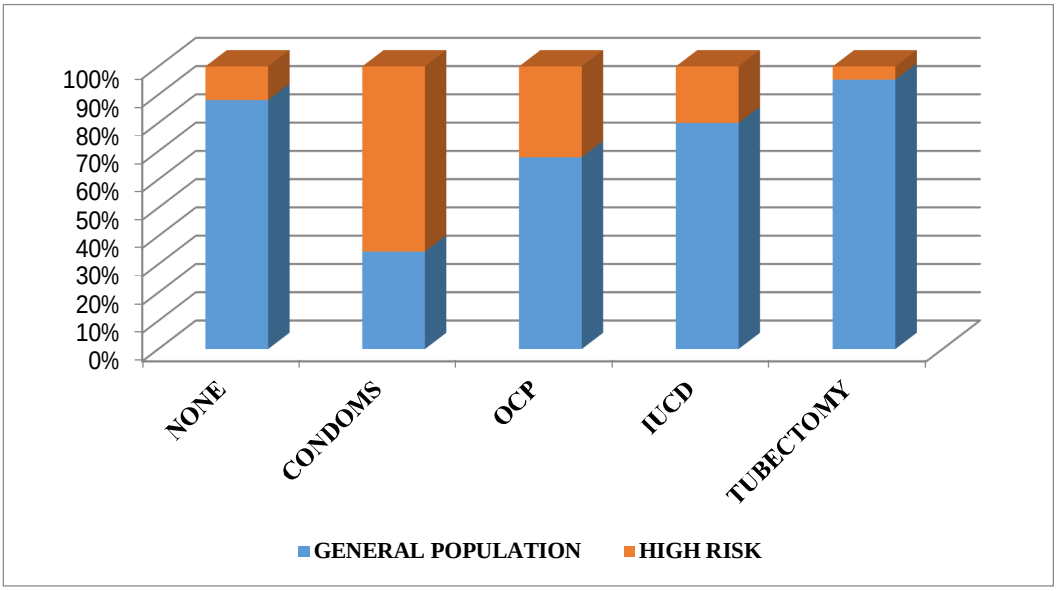


TABLE 9:

DISTRIBUTION BASED ON LABORATORY TEST RESULTS

	GENERAL POPULATION	HIGH-RISK GROUPS	TOTAL
NO. OF SAMPLES POSITIVE FOR PATHOGEN	68(46.2%)	37(69.8%)	105(52.5%)
NORMAL VAGINAL FLORA	79(53.8%)	16(30.2%)	95(47.5%)
TOTAL	147	53	200
SIGNIFICANCE	$\chi^2 = 8.665$ P value = 0.0032 P<0.05		

The percentage of pathogens identified by wet mount, gram stain and culture was significantly higher (p value<0.05) in HRGs when compared to the general group who had normal vaginal flora in more than half of the cases.

CHART 6:

DISTRIBUTION BASED ON LABORATORY TEST RESULTS

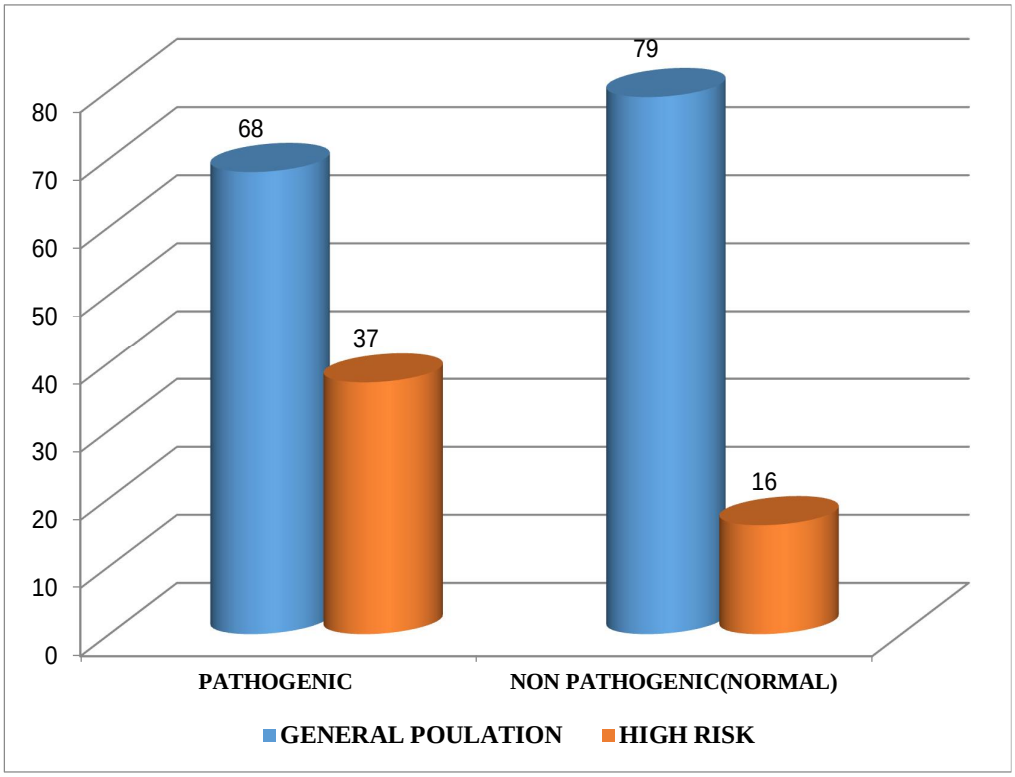


CHART 7:

DISTRIBUTION OF ABNORMAL VAGINAL DISCHARGE BASED ON PROBABLE INFECTIOUS ETIOLOGY

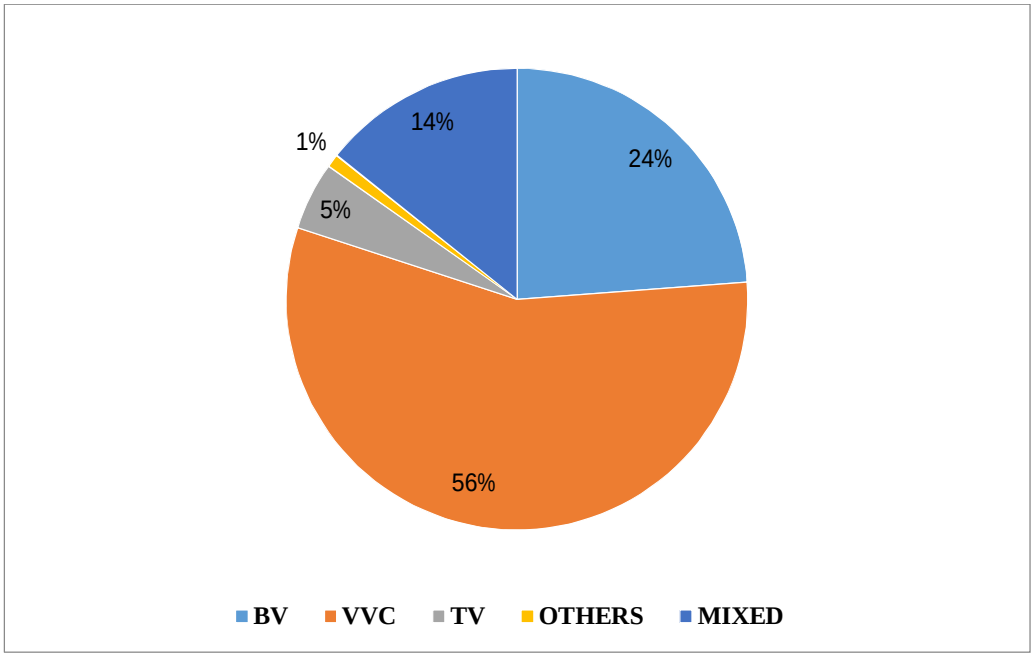


TABLE 10:

DISTRIBUTION OF ABNORMAL VAGINAL DISCHARGE BASED ON PROBABLE INFECTIOUS ETIOLOGY:

	GENERAL POPULATION	HIGH-RISK GROUP	TOTAL
BACTERIAL VAGINOSIS	17(25%)	8(21.6%)	25(23.8%)
VULVO-VAGINAL CANDIDIASIS	42(61.8%)	17(46%)	59(56.3%)
TRICHOMONIASIS	0	5(13.5%)	5(4.7%)
OTHERS	1-Enterococcus (1.5%)	0 (0%)	1(1%)
MIXED INFECTIONS	8 (11.7%)	7(18.9%)	15(14.2%)
TOTAL PATIENTS TESTED POSITIVE	68	37	105
TOTAL ISOLATES	76	44	120

The most common etiological agent in both the groups was Candidiasis (56.3%) followed by Bacterial Vaginosis (23.8%). Trichomoniasis was not identified in General group whereas it constituted 13.5% of pathogens isolated in HRG. Mixed infections were found in both groups constituting about 14.2%.

TABLE 11: BACTERIAL VAGINOSIS BY AMSEL'S CRITERIA

AMSEL'S CRITERIA	GENERAL POPULATION	HIGH-RISK GROUP	TOTAL
3 or more positive	36(24.5%)	18(33.9%)	54(27%)
Less than 3	111(75.5%)	35(66.1%)	146(73%)
TOTAL	147	53	200

According to Amsel's criteria if 3 out of 4 criteria are fulfilled, it is suggestive of BV. Amsel's criteria identified 27% of patients as having BV.

TABLE 12:

BACTERIAL VAGINOSIS BY NUGENT'S SCORING OF GRAM STAINED VAGINAL SMEARS

NUGENT'S SCORE	GENERAL POPULATION	HIGH-RISK GROUP	TOTAL
0-3(Normal Flora)	74(50.3%)	20(37.7%)	94(47%)
4-6(Intermediate)	48(32.6%)	18(34%)	66(33%)
7-10(BV)	25(17.1%)	15(28.3%)	40(20%)
TOTAL	147	53	200

Nugent's score identified 20% of samples to be BV, 33% intermediate flora and 47% as normal flora.

CHART 8: BACTERIAL VAGINOSIS – NUGENT'S SCORE

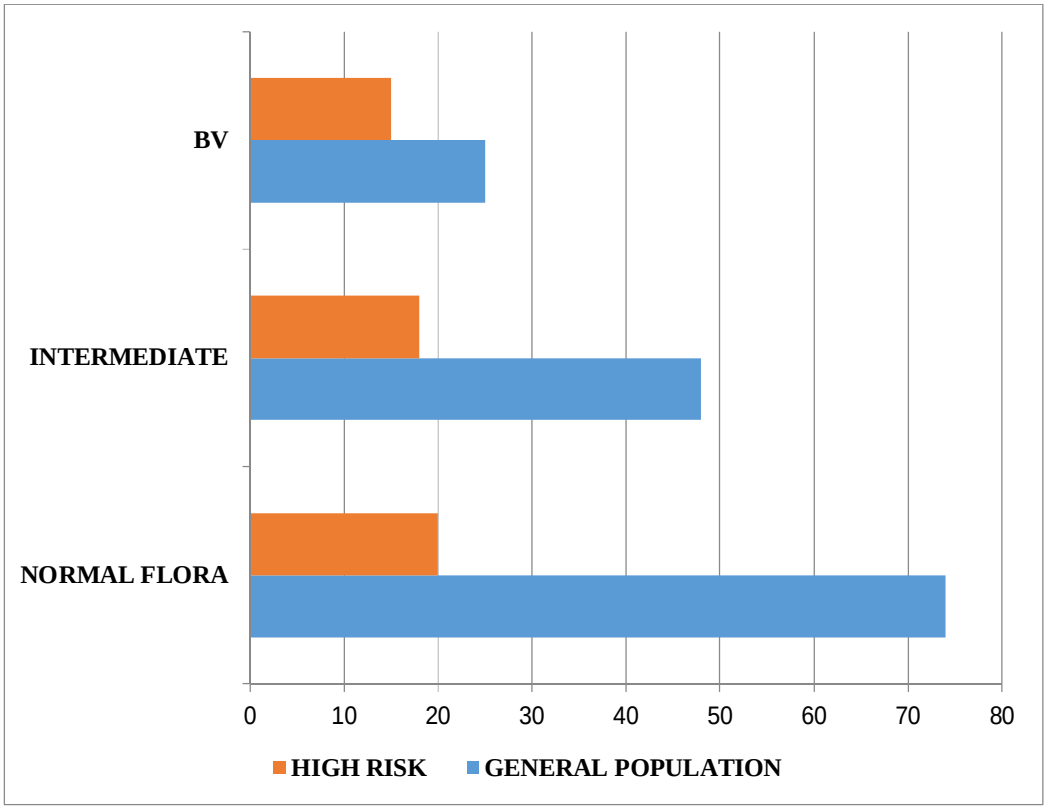


TABLE 13:

COMPARISON BETWEEN AMSEL'S CRITERIA & NUGENT'S CRITERIA

BACTERIAL VAGINOSIS	BY AMSEL'S CRITERIA	BY NUGENT'S CRITERIA
GENERAL POPULATION	36	25
HIGH RISK GROUP	18	15
TOTAL	54	40

Amsel's criteria identified more cases of BV than Nugent's criteria but Nugent's scoring is widely accepted as the Gold standard for diagnosis of BV.

TABLE 14: DISTRIBUTION OF CANDIDIASIS

VULVO-VAGINAL CANDIDIASIS	GENERAL POPULATION	HIGH-RISK GROUP	TOTAL
Candida albicans	21(42%)	11(47.8%)	32(43.8%)
Non albicans Candida	29(58%)	12(52.2%)	41(56.2%)
TOTAL	50	23	73

NAC(56.2%) were more commonly isolated than C. albicans (43.8%) among both the groups.

CHART 9: VULVO VAGINAL CANDIDIASIS

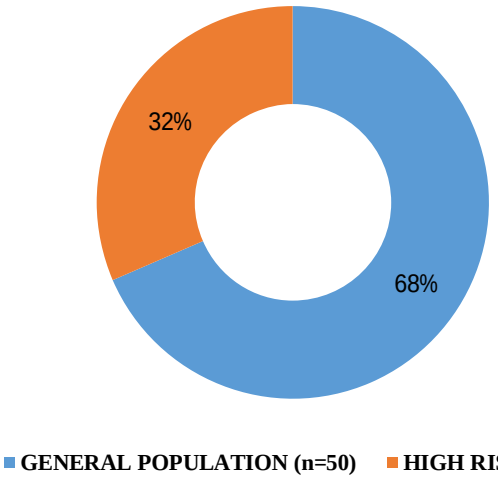


TABLE 15: CANDIDA SPECIATION

CANDIDA SPECIES	NO. OF ISOLATES		
	NORMAL POPULATION	HIGH RISK GROUPS	TOTAL
C. Albicans	21(42%)	11 (47.8%)	32 (43.8%)
C. Tropicalis	10(20%)	4 (17.4%)	14 (19.3%)
C. Glabrata	9(18%)	3 (13.1%)	12 (16.4%)
C. Krusei	6(12%)	3 (13.1%)	9 (12.3%)
C. Parapsilosis	4(8%)	2 (8.6%)	6 (8.2%)
TOTAL	50	23	73

Speciation of Candida was done based on Germ tube test, CHROMagar and CornMeal agar. C. albicans was the most common species (43.8%)isolated in both the groups. Among the NAC, C. tropicalis (19.3%) was most commonly isolated.

CHART 10: CANDIDA SPECIATION

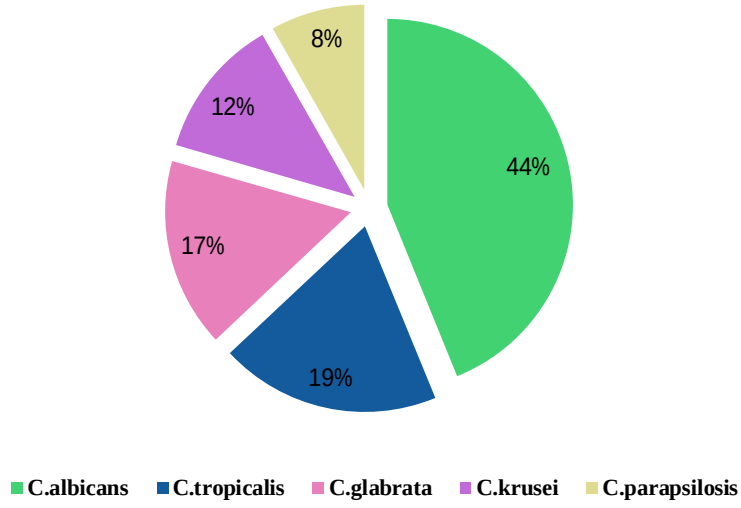


TABLE 16: DISTRIBUTION OF TRICHOMONIASIS

	BY WET MOUNT	BY GIEMSA	BY CULTURE
NORMAL POPULATION	0	0	0
HIGH RISK GROUP	5	5	6
TOTAL	5	5	6

Culture of Trichomonas in CPLM broth was the most sensitive as one case missed by wet mount examination and giemsa stain probably due to low parasite load or reduced motility was identified after culture of vaginal discharge for 4 days.

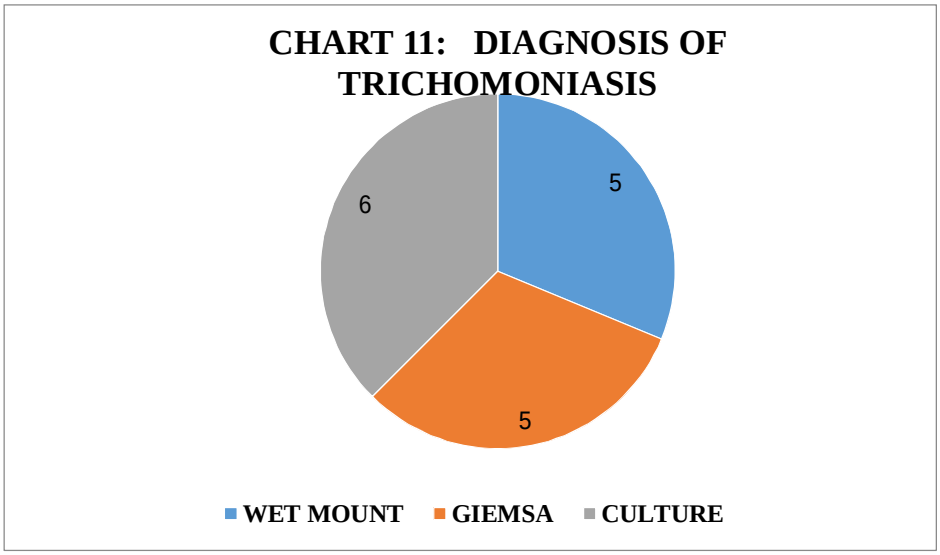


TABLE 17:

DISTRIBUTION BASED ON NO. OF PATHOGENS ISOLATED FROM A PATIENT

PATHOGEN	GENERAL POPULATION	HIGH-RISK GROUPS	TOTAL
SINGLE	60(88.2%)	30(81.1%)	90
MIXED INFECTION	8- 8 (BV & Candida) (11.7%)	7(18.9%) -1(2.7%) (TV&BV) -6(16.2%)(BV & Candida)	15
TOTAL	68	37	

Mixed infections were found in both groups (14.2%). Of this, co-infection of BV and Candidiasis was most common- 11.7% in general group and 16.2% in HRG.

CHART 12:

DISTRIBUTION BASED ON SINGLE OR MULTIPLE INFECTIONS

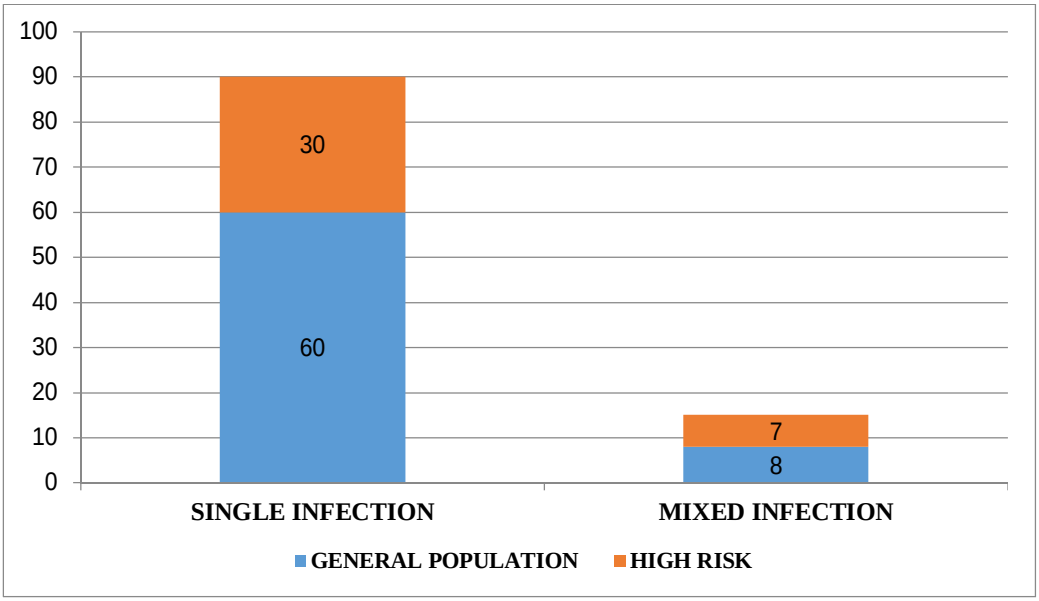


TABLE 18: DISTRIBUTION BASED ON HIV STATUS

HIV STATUS	NORMAL POPULATION	HIGH RISK GROUPS
NEGATIVE	147(100%)	51(96.2%)
POSITIVE	0	2(3.8%)
TOTAL	147	53

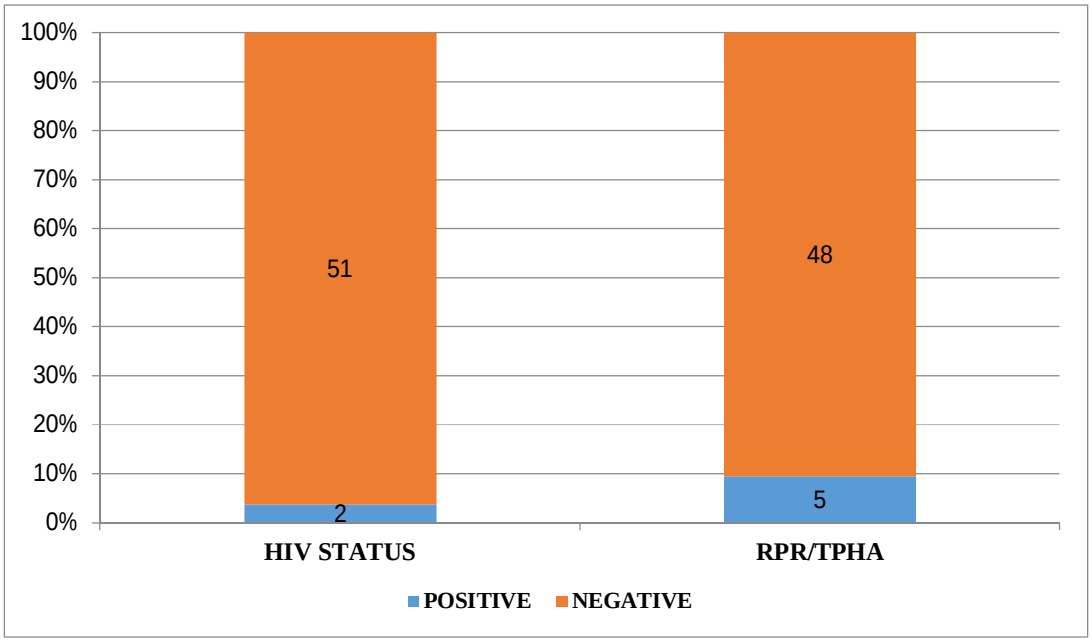
TABLE 19: DISTRIBUTION BASED ON RPR/TPHA POSITIVITY

SYPHILIS	NORMAL POPULATION	HIGH RISK GROUPS
NEGATIVE	147 (100%)	48 (90.5%)
POSITIVE	0	5 (9.5%)
TOTAL	147	53

None of the patients in general group had syphilis or HIV. This shows low prevalence of these diseases in general population without any risk factors.

CHART 13:

HIV AND SYPHILITIC STATUS OF HIGH RISK INIDIVIDUALS



DISCUSSION

DISCUSSION

Abnormal Vaginal discharge is one of the most common but often neglected health problem in women in their reproductive age. Therefore it is important to know the exact prevalence of AVD in women with genital tract infections. This study is carried out to determine the characteristics of vaginal discharge and the prevalence of pathogens causing infectious vaginal discharge.

In the present study, total number of patients were 200 of which 147 (73.5%) patients belonged to general population with no significant risk factors and 53 (26.5%) patients belonged to high risk groups (Table 1).

Among these 200 patients included in the study, 146 (73%) presented with abnormal vaginal discharge as the chief presenting complaint whereas 54(27%) presented with other complaints like lower abdominal pain, infertility, irregular menstrual cycle and dysuria and were found to have abnormal vaginal discharge on examination. (Table 2) In a study by Das et al, 68% were symptomatic with AVD and 32 % were asymptomatic which is similar to the finding in this study.[32]

Among the 200 patients, 102(69%) from Gynaecology OPD and 44(83%) from STD OPD had abnormal vaginal discharge as the chief presenting complaint whereas 45(31%) from Gynaecology OPD and 9(17%) from STD OPD presented with other complaints like lower abdominal pain, infertility or irregular menstruation and were found to have AVD on examination. (Table 2) This shows higher awareness among women in high risk groups about the nature of vaginal discharge and for seeking medical services.

The study included menstruating women of reproductive age group from 18 years to 45 years as they are most vulnerable to acquire STIs. The commonest age group in both the study groups was 30-39 years. Of the 147 women from Gynaecology OPD, 61(41%) belonged to age group of 30-39 and of the 53 women from STD OPD, 25 (47%) belonged to age group of 30-39. The next common age group was 18-29 years, 58(39%) of general population and 18(33%) of high risk groups belonged to this age group.(Table 3)

The most common age group coincides with the study by Samia S. Khamees et al. in which the mean age was 30.8 and the highest

prevalence of STD (32%) has been found in the age group of 29 - 33 years.[33]

This is also in accordance with a study by R Sivaranjani et al. where the mean age was 35.24 ± 4.37 years.[34] This is attributed to higher sexual activity in this age group.

Based on marital status (Table 4), in both the groups married women formed the majority. 92.5% of general group and 45.2% of high risk group were married and living with their husband. There were no unmarried women in general group whereas 9.4% of high risk group were unmarried. The widowed (13.2%) and separated women (32.2%) were more in number in the high risk group than general group [widowed (2%), separated (5.4%)]. These findings are similar to a study by Madeline Sutton et al.[35] where percentage of widows and separated females was higher in high risk groups.

The educational status analysis (Table 5) of both the groups showed that majority (31.3%) of general population had completed high school whereas middle school education was more common among high risk group (32.1%). Overall, Literacy was significantly lower in the high risk group than general population. This is similar to the findings of the

study by Bambara Moussa et al. where 31.8% of high risk women had only middle school education.[36] Many of the female sex workers also cited lack of education and means of livelihood as one of the reasons for entering sex work.

Among the high risk groups,(Table 6) most common were the female sexual workers (33.9%), next common high risk factor was females with multiple sexual partners (32.1%), followed by females with high risk partners (16.9%). This is because of the efforts put in by the Non Government Organisations (NGO) in mobilizing the Female Sexual Workers (FSW) to attend STD OPD and creating an awareness about sexual health among them.

Comparing the parity (Table 7), in both groups multi-parous women formed the majority with 53.2% of general group and 60.4% of high risk group. In the general group permanent sterilization by tubectomy was the most common method of contraception whereas barrier method by condom usage was most common contraceptive method used in high risk group (Table 8).

This is similar to the study by Bambara Moussa et al. where male condom was the most commonly adopted practice among high risk group

women.[36] No contraceptive methods were adopted in 25.3% of general group and 9.5% of high risk group. In this context, Barrier methods are considered to be better as they provide protection against transmission of STD in addition to contraception.

Of the 200 patients tested (Table 9), 105 (52.5%) were positive for potential pathogens and 95 (47.5%) did not show any pathogenic organisms and had only normal vaginal flora. Of the 105 patients tested positive for pathogens, 68 were from general group and 37 were high risk individuals. 79(53.8%) of general group and 16 (30.2%) of high risk group showed normal vaginal flora. This is similar to a study by R Sivaranjani et al. in which infectious agents were found in 51.75% patients.[34]

Of the 146 patients who presented with AVD, 76(52.1%) patients tested positive for pathogens. Of the 54 patients who presented with other complaints like lower abdominal pain, infertility or irregular menstruation and found to have AVD on examination, 29(53.7%) tested positive for pathogens.

Out of the 105 patients (52.5%) positive for pathogens, 25 patients (23.8%) were found to have Bacterial Vaginosis, 59(56.3%) patients had

VVC, 5(4.7%) had Trichomoniasis, 1(1%) had Enterococcal infection, 15(14.2%) had mixed infections with more than pathogen. (Table 10) This is similar to a study by Fonck et al.[37] where Candidiasis was found to be more prevalent (50%) among reproductive age group women. It is in contrast to a study by Bhalla et al.[38] where the most common infection was BV (32.8%), followed by candidiasis (16.9%) and trichomoniasis (2.8%) cases.

The rise in VVC as a major cause of AVD could be due to increased use of broad spectrum antibiotics for various health reasons which alter the normal vaginal flora leading to positive shift in colonization of yeast.[39] This could account for an increase in VVC over BV as the leading cause of abnormal Vaginal Discharge.

In this study, (Table 11) Amsel's criteria identified 54 patients (27%) to have clinical findings suggestive of BV.

By Nugent's scoring, (Table 12) 40 smears (20%) had a score of 7-10 and confirmed to have BV in which 25(17.1%) belonged to general group and 15 (28.3%) belonged to high risk group. 94 smears (47%) showed a score of 0-3 (Normal Flora). 66 smears(33%) had a intermediate score of 4-6 and these patients should be followed up and

gram stain should be repeated to detect progression to BV. This is in accordance with a study by P Madhivanan et al. in which 19.1% had BV, 65.4% had normal flora and 15.4% had intermediate flora.[41]

Bacterial Vaginosis can be diagnosed bedside by clinical criteria such as Amsel's criteria but Nugent's laboratory method is the Gold standard for diagnosis of BV. Rangari et al. in their study reported Nugent score had a higher sensitivity in diagnosis of BV while Amsel's criteria had less sensitivity but higher specificity. They concluded Amsel's criteria without using staining methods for detection of 'clue cells' could be misleading.[40]

Total number of Candida spp. isolated were 73 out of which 32(43.8%) were Candida albicans and 41(56.2%) were non albicans Candida. (Table 14) Of the non albicans Candida, most common was C. tropicalis(19.3%), followed by C. glabrata (16.4%), C. krusei(12.3%) and C. parapsilosis(8.2%). (Table 15) Thus, Candida albicans is the most common species isolated followed by C. tropicalis and C.glabrata and C. krusei.

This is in accordance with a study by Oyeyipo et al.[50] in which Candida albicans (35.0%) was the predominant species followed by C.

tropicalis (8.3%), C. glabrata (6.7%) and C. krusei (3.3%). Also, the study by Doddaiah Vijaya et al. showed C. tropicalis to be the most common non albicans species isolated from VVC.[42]

This study shows a marked rise in proportion of NAC infections as compared to previous studies like the study by Twinkle n. Gandhi et al. where C. albicans was 66.39% of Candida isolates and NAC formed 33.61%.[43]

The reason for this may be the indiscriminate over-the-counter usage of antifungals like Fluconazole which destroys the more sensitive Candida albicans and selects the less sensitive non albicans species. The resistance to azole groups among the non albicans Candida is on the rise and thus their identification becomes mandatory.

The speciation was done with commercially available CHROMagar which has several advantages like direct identification of species and rapidity, rendering it useful for early identification and treatment of Candidial infection.[44] These results also correlated well with the Corn meal agar test and the Candida speciation was thus confirmed with the results of both CHROMagar and Corn meal agar.

Trichomoniasis was better identified by culture in CPLM than by direct examination by wet mount. (Table 16) Total isolates were 6(5.7%) in which 5(4.7%) were identified by wet mount examination, giemsa stain and culture. One isolate was identified only after culture on CPLM. This shows greater sensitivity by Culture methods which are the gold Standard for diagnosis of trichomoniasis. This is similar to a study by Van der Schee C et al. in which wet mount microscopy detected 3.8% Trichomonas infection as opposed to 4.9% positives in modified Diamond's medium.[45]

Mixed infections with more than 1 pathogen were seen in 15 patients (14.2%). 8 patients in the general population and 6 patients in high risk group had co-infection of BV and Candidiasis. One patient in the high risk group had co-infection of TV and BV. (Table 17)

None of the women in general population were HIV positive whereas 2(3.8%) patients in high risk group were known HIV positive. (Table 18)

Similarly, none of the women in general population were positive for RPR/TPHA whereas 5(9.5%) patients in high risk group were positive. (Table 19) These served as additional risk factors in the high

risk groups consistent with promiscuous sexual practices not seen in general population.

PREDISPOSING FACTORS:

Majority of the women reported usage of sanitary napkins during menstruation than cloth napkins or tampons. None of the women reported douching practice. This improvement in personal hygiene could also be one of the reasons for decrease in BV prevalence in this locality since douching is consistently associated with BV as shown in the study by Mark A. Klebanoff et al.[46]

There was a positive correlation between Diabetes mellitus and Candidiasis in this study. Out of the 73 patients with candidiasis, 13(17.8%) patients gave a positive history of Diabetes. Different studies such as the study by Reza Faraji et al. have indicated VVC is more common in women with diabetes than in the normal population. [47]

In this study, Trichomonas vaginalis was isolated only from High risk women and not from general population. This shows Trichomoniasis being a sexually transmitted disease is more prevalent in women with promiscuous sexual practices. High-risk sexual behaviour is positively

associated with Trichomoniasis as reported by Phuong Anh Ton Nu et al.[48]

One patient had Enterococcus as the sole cause of Abnormal Vaginal discharge. She was a neglected woman with history of induced abortion one month ago. Personal hygiene was very poor. On examination, there was excessive, inspissated, dirty white, foul smelling vaginal discharge. Culture showed heavy growth of Enterococcus faecalis which was sensitive to penicillin, ampicillin, high level gentamycin, vancomycin and resistant to ciprofloxacin. She was treated with Penicillin and soon symptomatically improved.

SUMMARY

SUMMARY

- This study on infectious etiology of Abnormal Vaginal Discharge (AVD) in women of reproductive age group was conducted to determine the prevalence of AVD and its etiological agents among two groups of women.

- One group of women attending Gynaecology OPD without significant risk factors were representative of general population and the other group of women with associated high risk factors attending STD OPD was representative of the High Risk Groups (HRG).

- The general group consisted of 147 women and the High risk group consisted of 53 women.

- Of the 200 patients tested, 52.5% were positive for pathogens and 47.5% did not show any potential pathogens.

- The most common etiological agent in both the groups was Candidiasis(56.3%).

- The second most common etiology was Bacterial Vaginosis (23.8%).

- Trichomoniasis was not identified in General group whereas it constituted 13.5% of pathogens isolated in HRG.

- Mixed infections were found in both groups constituting about 14.2%. Of this, co-infection of BV and Candidiasis was most common- 11.7% in general group and 16.2% in HRG.

- In BV, Amsel's clinical criteria (27%) identified more number of cases than Nugent's scoring method (20%) but the laboratory scoring of gram stained smear is considered the gold standard.

- Candidiasis was most common etiological agent causing AVD in this study. This could be due to indiscriminate use of antibiotics leading to a shift towards fungal pathology. Though Candida albicans (43.8%) was the most common species identified, there is a significant rise in non albicans Candida (NAC) species. This is probably due to over-usage of fluconazole being included in the Green kit for vaginal discharge syndrome which may selectively increase growth of fluconazole resistant NAC.[52]

- Trichomoniasis was better identified by culture in CPLM medium than by wet mount examination in which one case with low parasite load was missed and later identified in culture.

- HIV and syphilis was not documented in general population whereas 3.8% HIV positivity and 9.5% RPR/TPHA positivity was seen in high risk groups.

- Overall, the positivity for pathogens was more in HRG with 69.8% as opposed to general population (46.2%)

CONCLUSION

CONCLUSION

This study was done with the aim of determining the various causes of infectious vaginal discharge among general population and high risk groups and Candidiasis was found to be the most common etiological agent in both the groups followed by Bacterial Vaginosis. Co-infections with Candidiasis and BV were also seen in a fair proportion of patients in both groups. Trichomoniasis, HIV and Syphilis were seen only in individuals with high risk. Gonococcal infection was not detected in both the groups.

There is a significant difference between the number of pathogen positive cases among general population and high risk population. High risk individuals were significantly more prone to infections. There is an urgent need to spread awareness among high risk groups of the need for use of preventive measures and to decrease their risk activity.

Syndromic management still remains the mode of treatment for vaginal discharge syndrome and it does provide adequate antibiotic cover in many cases. But, it is mandatory to investigate the non-responders to syndromic management and give appropriate therapy.

However, in view of increasing incidence of non albicans Candida with decreased sensitivity to fluconazole, without the etiological diagnosis and susceptibility testing these patients cannot be treated properly. This is more important in case of C.krusei which is intrinsically resistant to fluconazole. In such cases, voriconazole and itraconazole are the drugs of choice which is not included in the Green kit used in Syndromic management of Vaginal Discharge syndrome.

Also, Syndromic approach causes over-exposure to drugs for infections not harboured by the patient and in the long run leads to development of resistance in the microorganisms.

Prompt diagnosis and treatment of Genito- Urinary infections will prevent a lot of distress to the patient. Delayed or inadequate treatment may lead to serious complications like PID causing infertility, increased risk of ectopic pregnancies, abdominal discomfort and chronic pelvic pain.

Infectious vaginal discharge in pregnant women can lead to PROM, IUGR, IUD, preterm labour and repeated miscarriages.

Abnormal Vaginal Discharge being an often overlooked symptom among women needs to be addressed as an important issue. Women with

abnormal vaginal discharge hesitate to seek medical assistance until the complaint becomes intolerable and hinders routine activities.[49]

Thus, it is essential to spread awareness about the predisposing factors, symptoms and easily available treatment of AVD among women particularly targeting the sexually active and reproductive age groups to reduce mortality and morbidity in mother and baby.

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ANNEXURES

PROFORMA

/200

Serial No:

1)Name:

Date:

2) Age :

3) Gender:

4) OP No:

5) PIN No:

HISTORY DETAILS:

6) H/O abnormal vaginal discharge (Yes/No): Colour, consistency, odour

7) H/O itching(Yes /No):

8) H/O foul-odour (Yes/No):

9) H/O lower abdominal pain (Yes/No):

10) H/O dyspareunia (Yes/No):

11)H/O antibiotic use (Yes/No):

12) H/O comorbid illness(Yes/No):

MENSTRUAL HISTORY:

Age at menarche:

Last menstrual period:

MARITAL HISTORY:

Married since:

No. of children:

Last child birth:

Last marital contact:

Extra marital contact:

Pre marital contact:

GENERAL EXAMINATION :

Anaemia (Yes/No):

Jaundice (Yes/No):

Generalised Lymphadenopathy (Yes/No):

SYSTEMIC EXAMINATION

RESPIRATORY SYSTEM :

CARDIOVASCULAR SYSTEM :

CENTRAL NERVOUS SYSTEM :

PER ABDOMEN EXAMINATION:

PER VAGINAL EXAMINATION :

PER SPECULUM EXAMINATION :

INVESTIGATIONS:

TREATMENT:

CONSENT FORM

I agree to participate in the study entitled **"A STUDY ON THE INFECTIOUS ETIOLOGY OF ABNORMAL VAGINAL DISCHARGE IN WOMEN OF REPRODUCTIVE AGE GROUP ATTENDING A TERTIARY CARE HOSPITAL"**. I confirm that I have been told about this study in my mother tongue and have had the opportunity to clarify my doubts.

I understand that my participation is voluntary and I may refuse to participate at any time without giving any reasons and without affecting my benefits.

I agree not to restrict the use of any data or results that arise from this study.

Name of the participant: Sign / Thumb print:

Name of the investigator:

Sign of Investigator:

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MASTER CHART

KEY TO MASTER CHART

CONTRA	-	CONTRACEPTIVE USED
EDU	-	EDUCATION
AVD	-	ABNORMAL VAGINAL DISCHARGE
LAP	-	LOWER ABDOMINAL PAIN
INF	-	INFERTILITY
IM	-	IRREGULAR MENSTRUATION
MARITAL	-	MARITAL STATUS
M	-	MARRIED
W	-	WIDOWED
S	-	SEPARATED
UN	-	UNMARRIED
PL	-	PARA LIVE
OCP	-	ORAL CONTRACEPTIVE PILLS
TUBECT	-	TUBECTOMY
IUCD	-	INTRA UTERINE CONTRACEPTIVE DEVICE
ILL	-	ILLITERATE
PRI	-	PRIMARY
MID	-	MIDDLE SCHOOL
HS	-	HIGH SCHOOL

HSEC	-	HIGHER SECONDARY
DEG	-	DEGREE
FSW	-	FEMALE SEXUAL WORKER
HRP	-	FEMALE WITH HIGH RISK PARTNER
MSP	-	FEMALE WITH MULTIPLE SEXUAL PARTNER
PVD	-	FEMALE WITH PAST VENEREAL DISEASE
VD	-	FEMALE WITH KNOWN VENEREAL DISEASE
BYC	-	BUDDING YEAST CELLS
PH	-	PSEUDOHYPHAE
PUS	-	PUS CELLS
NF	-	NORMAL FLORA
INT	-	INTERMEDIATE FLORA
BV	-	BACTERIAL VAGINOSIS
C.ALB	-	CANDIDA ALBICANS
C.TROP	-	C. TROPICALIS
C.GLAB	-	C.GLABRATA
C.KRU	-	C. KRUSEI
C.PARA	-	C.PARAPSILOSIS
VVC	-	VULVO VAGINAL CANDIDIASIS
TV	-	TRICHOMONAS VAGINALIS

MASTER CHART

S.NO	OP	AGE	SYMPTOM	MARITAL	PARITY	CONTRA	EDU	RISK	WET MOUNT	KOH	GRAM S	AMSEL'S	NUGENT'S	CULTURE	HIV	RPR	DM	DIAGNOSIS
1	31882	23	AVD	M	P1L1	OCP	HS	-	NORMAL	BYC,PH	PUS, BYC,PH	<3	NF	C.ALB	-	-	-	VVC
2	31887	19	AVD	M	P0L0	NIL	HSEC	-	CLUE	-	CLUE	3+	BV		-	-	-	BV
3	31884	20	AVD	M	P0L0	NIL	HSEC	-	NORMAL	-	NORMAL	<3	NF		-	-	-	NF
4	31888	44	LAP	W	P3L3	TUBECT	ILL	-	NORMAL	BYC,PH	PUS, BYC,PH	3+	INT	C.KRU	-	-	+	VVC
5	31831	40	AVD	M	P2L2	TUBECT	MID	-	NORMAL	-	NORMAL	<3	NF		-	-	-	NF
6	31754	41	AVD	M	P2L2	TUBECT	MID	-	NORMAL	BYC	PUS, BYC,PH	<3	NF	C.GLAB	-	-	-	VVC
7	32067	28	INF	M	P0L0	NIL	DEG	-	NORMAL	-	NORMAL	<3	INT		-	-	-	NF
8	32180	38	AVD	M	P3L3	TUBECT	PRI	-	NORMAL	-	NORMAL	3+	INT		-	-	-	NF
9	32218	24	IM	M	P0L0	NIL	DEG	-	NORMAL	-	NORMAL	<3	NF		-	-	-	NF
10	32141	30	AVD	M	P1L1	IUCD	HS	-	NORMAL	-	NORMAL	<3	INT		-	-	-	NF
11	33041	20	AVD	M	P1L1	OCP	HSEC	-	NORMAL	BYC,PH	PUS, BYC,PH	<3	NF	C.ALB	-	-	-	VVC
12	33047	31	AVD	M	P1L1	IUCD	HS	-	NORMAL	-	NORMAL	<3	NF		-	-	-	NF
13	33108	37	AVD	S	P2L2	TUBECT	MID	-	NORMAL	-	NORMAL	<3	INT		-	-	-	NF
14	33112	36	IM	M	P2L2	TUBECT	PRI	-	CLUE	-	CLUE	3+	BV		-	-	-	BV
15	33187	24	AVD	M	P1L1	OCP	DEG	-	NORMAL	-	NORMAL	3+	INT		-	-	-	NF
16	33186	35	LAP	M	P2L2	TUBECT	PRI	-	NORMAL	BYC,PH	PUS, BYC,PH	<3	INT	C.KRU	-	-	-	VVC
17	33409	40	AVD	S	P2L2	TUBECT	MID	-	NORMAL	BYC,PH	PUS, BYC,PH	<3	NF	C.TROP	-	-	+	VVC
18	33446	20	INF	M	P0L0	NIL	HSEC	-	NORMAL	-	NORMAL	<3	NF		-	-	-	NF
19	33469	41	AVD	M	P3L3	TUBECT	HS	-	NORMAL	-	NORMAL	<3	INT		-	-	-	NF
20	33430	20	AVD	M	P1L1	OCP	HS	-	NORMAL	-	NORMAL	<3	INT		-	-	-	NF
21	33454	34	INF	M	P0L0	NIL	MID	-	NORMAL	-	NORMAL	<3	NF		-	-	-	NF
22	33435	38	AVD	M	P2L2	TUBECT	ILL	-	NORMAL	-	NORMAL	3+	INT		-	-	-	NF

S.NO	OP	AGE	SYMPTOM	MARITAL	PARITY	CONTRA	EDU	RISK	WET MOUNT	KOH	GRAM S	AMSEL'S	NUGENT'S	CULTURE	HIV	RPR	DM	DIAGNOSIS
23	32860	41	AVD	M	P3L3	TUBECT	HS	-	NORMAL	BYC,PH	PUS, BYC,PH	<3	NF	C.ALB	-	-	-	VVC
24	33686	18	IM	M	P0L0	NIL	HSEC	-	NORMAL	-	NORMAL	<3	INT		-	-	-	NF
25	33723	45	LAP	W	P2L2	TUBECT	ILL	-	NORMAL	BYC	PUS, BYC	<3	INT	C.GLAB	-	-	+	VVC
26	34106	19	AVD	M	P0L0	NIL	HSEC	-	NORMAL	BYC,PH	PUS, BYC,PH	<3	NF	C.TROP	-	-	-	VVC
27	34118	30	INF	M	P0L0	NIL	DEG	-	NORMAL	-	NORMAL	<3	INT		-	-	-	NF
28	34111	42	AVD	M	P3L2	TUBECT	MID	-	CLUE	BYC,PH	CLUE,PUS, BYC	3+	BV	C.PARA	-	-	-	VVC+BV
29	34188	26	AVD	M	P1L1	CONDOM	DEG	-	NORMAL	BYC	PUS, BYC	<3	NF	C.GLAB	-	-	-	VVC
30	35144	37	AVD	M	P2L2	TUBECT	PRI	-	NORMAL	-	NORMAL	<3	NF		-	-	-	NF
31	35171	19	INF	M	P0L0	NIL	HSEC	-	NORMAL	-	NORMAL	<3	INT		-	-	-	NF
32	35162	40	AVD	M	P2L2	TUBECT	PRI	-	NORMAL	BYC,PH	PUS, BYC,PH	<3	NF	C.TROP	-	-	-	VVC
33	35375	35	AVD	M	P2L2	TUBECT	MID	-	NORMAL	-	NORMAL	<3	NF		-	-	-	NF
34	35452	21	INF	M	P0L0	NIL	DEG	-	NORMAL	-	NORMAL	3+	INT		-	-	-	NF
35	35365	30	AVD	M	P2L2	IUCD	HS	-	NORMAL	-	NORMAL	<3	INT		-	-	-	NF
36	35428	20	AVD	M	P1L1	OCP	HSEC	-	NORMAL	BYC,PH	PUS, BYC,PH	<3	NF	C.ALB	-	-	-	VVC
37	35479	35	IM	M	P2L2	TUBECT	DEG	-	CLUE	-	CLUE	3+	BV		-	-	-	BV
38	35468	23	LAP	M	P1L1	IUCD	HSEC	-	NORMAL	-	NORMAL	<3	NF		-	-	-	NF
39	35945	39	AVD	M	P2L2	OCP	ILL	-	NORMAL	BYC,PH	NORMAL	<3	NF	C.PARA	-	-	-	VVC
40	35969	26	AVD	M	P1L1	CONDOM	DEG	-	CLUE	-	CLUE	3+	BV		-	-	-	BV
41	35981	37	AVD	M	P2L2	CONDOM	MID	-	NORMAL	-	NORMAL	<3	NF		-	-	-	NF
42	35993	40	IM	M	P4L3	TUBECT	HS	-	NORMAL	-	NORMAL	<3	NF		-	-	-	NF
43	35942	32	INF	M	P0L0	NIL	HSEC	-	NORMAL	BYC	PUS, BYC,PH	3+	INT	C.GLAB	-	-	+	VVC
44	35615	30	AVD	M	P2L2	IUCD	MID	-	NORMAL	-	NORMAL	<3	NF		-	-	-	NF
45	35598	21	AVD	M	P0L0	NIL	DEG	-	NORMAL	-	NORMAL	<3	INT		-	-	-	NF

S.NO	OP	AGE	SYMPTOM	MARITAL	PARITY	CONTRA	EDU	RISK	WET MOUNT	KOH	GRAM S	AMSEL'S	NUGENT'S	CULTURE	HIV	RPR	DM	DIAGNOSIS
46	37079	25	AVD	M	P1L1	OCP	HSEC	-	NORMAL	-	NORMAL	<3	NF		-	-	-	NF
47	37140	37	AVD	M	P3L3	TUBECT	HS	-	CLUE	BYC,PH	CLUE,PUS, BYC	3+	BV	C.ALB	-	-	+	VVC+BV
48	37135	40	AVD	M	P2L2	TUBECT	PRI	-	NORMAL	-	NORMAL	<3	NF		-	-	-	NF
49	37307	38	AVD	M	P3L3	TUBECT	HS	-	NORMAL	-	NORMAL	3+	INT		-	-	-	NF
50	37323	37	IM	M	P2L2	TUBECT	HSEC	-	NORMAL	-	NORMAL	<3	INT		-	-	-	NF
51	1594	30	AVD	M	P2L2	TUBECT	HS	-	NORMAL	BYC,PH	PUS, BYC,PH	<3	NF	C.TROP	-	-	-	VVC
52	1593	42	IM	M	P0L0	NIL	PRI	-	NORMAL	-	NORMAL	<3	NF		-	-	+	NF
53	1607	29	AVD	M	P2L2	IUCD	DEG	-	CLUE	BYC,PH	CLUE	3+	BV	C.PARA	-	-	-	VVC+BV
54	1802	31	INF	M	P1L1	NIL	HS	-	NORMAL	BYC	PUS, BYC	<3	NF	C.KRU	-	-	-	VVC
55	1734	26	LAP	M	P1L1	IUCD	DEG	-	NORMAL	-	NORMAL	<3	INT		-	-	-	NF
56	1793	34	AVD	S	P2L2	CONDOM	HS	-	NORMAL	-	NORMAL	<3	INT		-	-	-	NF
57	1792	29	AVD	M	P1L1	OCP	HSEC	-	NORMAL	BYC,PH	PUS, BYC,PH	<3	NF	C.ALB	-	-	-	VVC
58	2005	32	AVD	M	P2L2	TUBECT	MID	-	CLUE	-	CLUE	3+	BV		-	-	-	BV
59	2043	28	AVD	M	P1L1	OCP	HS	-	NORMAL	BYC,PH	PUS, BYC,PH	<3	NF	C.TROP	-	-	-	VVC
60	2037	18	AVD	M	P0L0	NIL	HSEC	-	NORMAL	-	NORMAL	<3	NF		-	-	-	NF
61	2039	38	IM	M	P1L1	IUCD	DEG	-	CLUE	-	CLUE	3+	BV		-	-	-	BV
62	2042	40	AVD	M	P3L3	TUBECT	HS	-	NORMAL	-	NORMAL	<3	NF		-	-	+	NF
63	2045	24	AVD	M	P1L1	OCP	DEG	-	NORMAL	BYC,PH	PUS, BYC,PH	<3	INT	C.TROP	-	-	-	VVC
64	2048	37	AVD	M	P2L2	TUBECT	HS	-	NORMAL	-	NORMAL	<3	NF		-	-	+	NF
65	2053	23	IM	M	P0L0	NIL	HSEC	-	NORMAL	-	NORMAL	<3	NF		-	-	-	NF
66	2047	29	AVD	M	P2L2	TUBECT	DEG	-	CLUE	-	CLUE	3+	BV		-	-	-	BV
67	2059	23	INF	M	P0L0	NIL	DEG	-	NORMAL	BYC	PUS, BYC	<3	INT	C.GLAB	-	-	-	VVC
68	2062	20	AVD	M	P0L0	NIL	HSEC	-	NORMAL	-	NORMAL	<3	INT		-	-	-	NF

S.NO	OP	AGE	SYMPTOM	MARITAL	PARITY	CONTRA	EDU	RISK	WET MOUNT	KOH	GRAM S	AMSEL'S	NUGENT'S	CULTURE	HIV	RPR	DM	DIAGNOSIS
69	2063	31	AVD	M	P2L2	TUBECT	MID	-	CLUE	BYC,PH	CLUE,BYC,PH	3+	BV	C.ALB	-	-	-	VVC+BV
70	2078	19	AVD	M	P0L0	NIL	HSEC	-	NORMAL	-	NORMAL	<3	NF		-	-	-	NF
71	2076	45	IM	M	P2L2	TUBECT	ILL	-	NORMAL	-	NORMAL	<3	NF		-	-	-	NF
72	2014	20	AVD	M	P0L0	NIL	HSEC	-	CLUE	-	CLUE	3+	BV		-	-	-	BV
73	2089	30	AVD	M	P2L2	TUBECT	DEG	-	NORMAL	-	NORMAL	<3	NF		-	-	-	NF
74	2095	29	IM	M	P2L2	CONDOM	PRI	-	NORMAL	-	NORMAL	<3	INT		-	-	-	NF
75	2101	35	LAP	M	P3L2	TUBECT	MID	-	NORMAL	BYC,PH	PUS, BYC,PH	3+	INT	C.ALB	-	-	+	VVC
76	2145	27	AVD	M	P1L1	IUCD	DEG	-	NORMAL	-	NORMAL	<3	INT		-	-	-	NF
77	2149	37	AVD	M	P2L2	TUBECT	HS	-	NORMAL	-	NORMAL	<3	NF		-	-	-	NF
78	2188	40	AVD	S	P3L3	TUBECT	PRI	-	CLUE	BYC,PH	CLUE,BYC,PH	3+	BV	C.TROP	-	-	-	VVC+BV
79	2189	28	INF	M	P0L0	NIL	DEG	-	NORMAL	-	NORMAL	<3	NF		-	-	-	NF
80	2191	21	AVD	M	P0L0	NIL	HSEC	-	NORMAL	-	NORMAL	<3	NF		-	-	-	NF
81	2264	41	AVD	M	P2L2	TUBECT	MID	-	NORMAL	BYC,PH	PUS, BYC,PH	<3	INT	C.ALB	-	-	+	VVC
82	2269	28	AVD	M	P1L1	OCP	HS	-	CLUE	BYC,PH	CLUE,BYC,PH	3+	BV	C.ALB	-	-	-	VVC+BV
83	2276	31	AVD	M	P1L1	IUCD	MID	-	BYC,PH	BYC,PH	BYC,PH	<3	NF	C.TROP	-	-	-	VVC
84	2277	42	IM	M	P2L2	TUBECT	PRI	-	NORMAL	-	NORMAL	<3	NF		-	-	+	NF
85	2279	30	AVD	M	P1L1	OCP	MID	-	NORMAL	BYC,PH	PUS, BYC,PH	<3	INT	C.KRU	-	-	-	VVC
86	2283	27	LAP	M	P1L1	CONDOM	HSEC	-	NORMAL	-	NORMAL	<3	NF		-	-	-	NF
87	2291	35	AVD	M	P2L2	TUBECT	HS	-	CLUE	BYC,PH	CLUE	3+	BV	C.PARA	-	-	-	VVC+BV
88	2297	43	AVD	M	P3L3	TUBECT	PRI	-	NORMAL	-	NORMAL	3+	NF		-	-	-	NF
89	2299	26	AVD	M	P1L1	OCP	DEG	-	NORMAL	BYC,PH	PUS, BYC,PH	<3	INT	C.ALB	-	-	-	VVC
90	2371	25	AVD	M	P1L1	IUCD	HSEC	-	NORMAL	-	NORMAL	<3	NF		-	-	-	NF
91	2374	41	AVD	M	P2L2	TUBECT	PRI	-	CLUE	-	CLUE	3+	BV		-	-	-	BV

S.NO	OP	AGE	SYMPTOM	MARITAL	PARITY	CONTRA	EDU	RISK	WET MOUNT	KOH	GRAM S	AMSEL'S	NUGENT'S	CULTURE	HIV	RPR	DM	DIAGNOSIS
92	2379	22	INF	M	P0L0	NIL	DEG	-	NORMAL	-	NORMAL	<3	INT		-	-	-	NF
93	2364	42	AVD	M	P2L2	TUBECT	HS	-	NORMAL	BYC,PH	PUS, BYC,PH	<3	NF	C.ALB	-	-	+	VVC
94	2387	40	AVD	M	P2L2	TUBECT	PRI	-	NORMAL	-	NORMAL	3+	INT		-	-	-	NF
95	2389	36	AVD	S	P2L2	TUBECT	HS	-	CLUE	-	CLUE	3+	BV		-	-	-	BV
96	2395	28	IM	M	P1L1	OCP	HSEC	-	NORMAL	BYC,PH	PUS, BYC,PH	<3	NF	C.TROP	-	-	-	VVC
97	2398	30	AVD	M	P2L2	IUCD	HS	-	NORMAL	BYC,PH	PUS, BYC,PH	<3	INT	C.ALB	-	-	-	VVC
98	2399	21	AVD	M	P0L0	NIL	DEG	-	NORMAL	-	NORMAL	<3	NF		-	-	-	NF
99	2411	30	INF	M	P0L0	NIL	HS	-	NORMAL	-	NORMAL	<3	INT		-	-	-	NF
100	2412	21	INF	M	P0L0	NIL	HSEC	-	NORMAL	-	NORMAL	<3	INT		-	-	-	NF
101	2420	37	AVD	M	P2L2	TUBECT	HS	-	NORMAL	BYC,PH	PUS, BYC,PH	<3	NF	C.ALB	-	-	-	VVC
102	2456	19	AVD	M	P0L0	NIL	HSEC	-	NORMAL	-	NORMAL	<3	INT		-	-	-	NF
103	2445	44	IM	M	P3L3	TUBECT	ILL	-	NORMAL	-	NORMAL	<3	NF		-	-	-	NF
104	2500	38	AVD	S	P2L2	NIL	PRI	-	NORMAL	BYC,PH	PUS, BYC,PH	<3	NF	C.TROP	-	-	-	VVC
105	2512	26	AVD	M	P1L1	OCP	HSEC	-	NORMAL	-	NORMAL	<3	INT		-	-	-	NF
106	2510	30	IM	M	P2L2	TUBECT	HS	-	NORMAL	BYC	PUS, BYC	<3	NF	C.GLAB	-	-	-	VVC
107	2528	43	AVD	M	P2L2	TUBECT	PRI	-	NORMAL	-	NORMAL	<3	NF		-	-	+	NF
108	2588	21	AVD	M	P0L0	NIL	HSEC	-	CLUE	-	CLUE	3+	BV		-	-	-	BV
109	2589	28	LAP	M	P1L1	OCP	HS	-	NORMAL	-	NORMAL	<3	NF		-	-	-	NF
110	2594	39	AVD	S	P2L2	TUBECT	HS	-	CLUE	BYC,PH	CLUE,BYC	3+	BV	C.ALB	-	-	-	VVC+BV
111	2599	42	AVD	M	P3L3	TUBECT	PRI	-	NORMAL	-	NORMAL	<3	NF		-	-	-	NF
112	2597	31	AVD	M	P2L2	TUBECT	HS	-	NORMAL	-	NORMAL	3+	INT		-	-	-	NF
113	2613	25	AVD	M	P1L1	OCP	HSEC	-	NORMAL	-	NORMAL	<3	NF		-	-	-	NF
114	2617	30	INF	M	P0L0	NIL	HS	-	NORMAL	-	NORMAL	<3	INT		-	-	-	NF

S.NO	OP	AGE	SYMPTOM	MARITAL	PARITY	CONTRA	EDU	RISK	WET MOUNT	KOH	GRAM S	AMSEL'S	NUGENT'S	CULTURE	HIV	RPR	DM	DIAGNOSIS
115	2627	41	AVD	M	P2L2	TUBECT	HS	-	NORMAL	BYC,PH	PUS, BYC,PH	<3	INT	C.ALB	-	-	+	VVC
116	2625	26	AVD	M	P2L2	IUCD	HSEC	-	NORMAL	-	NORMAL	<3	INT		-	-	-	NF
117	2629	24	AVD	M	P1L1	NIL	HS	-	NORMAL	BYC	PUS, BYC	<3	NF	C.GLAB	-	-	-	VVC
118	2633	30	AVD	M	P2L2	TUBECT	HS	-	NORMAL	-	NORMAL	<3	NF		-	-	-	NF
119	2638	35	AVD	M	P2L2	TUBECT	PRI	-	NORMAL	-	NORMAL	<3	NF		-	-	-	NF
120	2645	40	AVD	M	P2L2	TUBECT	MID	-	CLUE	-	CLUE	3+	BV		-	-	-	BV
121	2699	32	LAP	M	P2L2	OCP	HSEC	-	NORMAL	-	NORMAL	<3	NF		-	-	-	NF
122	2714	22	AVD	M	P0L0	NIL	HS	-	NORMAL	BYC,PH	PUS, BYC,PH	<3	NF	C.ALB	-	-	-	VVC
123	2708	37	AVD	M	P2L2	TUBECT	PRI	-	CLUE	-	CLUE	3+	BV		-	-	-	BV
124	2719	34	AVD	M	P2L2	TUBECT	PRI	-	CLUE	-	CLUE	3+	BV		-	-	-	BV
125	2725	31	INF	M	P0L0	NIL	HSEC	-	NORMAL	BYC	PUS, BYC	<3	NF	C.GLAB	-	-	-	VVC
126	2726	22	AVD	M	P0L0	NIL	HSEC	-	NORMAL	-	NORMAL	<3	INT		-	-	-	NF
127	2728	30	AVD	M	P2L2	TUBECT	HS	-	NORMAL	BYC,PH	PUS, BYC,PH	<3	INT	C.KRU	-	-	+	VVC
128	2745	25	LAP	M	P1L1	CONDOM	HS	-	NORMAL	-	NORMAL	<3	NF		-	-	-	NF
129	2611	30	LAP	M	P2L2	IUCD	MID	-	NORMAL	BYC,PH	PUS, BYC,PH	<3	INT	C.KRU	-	-	-	VVC
130	2751	25	AVD	M	P1L1	OCP	HSEC	-	NORMAL	-	NORMAL	<3	NF		-	-	-	NF
131	2822	36	IM	M	P2L2	TUBECT	PRI	-	NORMAL	BYC,PH	PUS, BYC,PH	<3	NF	C.ALB	-	-	+	VVC
132	2826	24	AVD	M	P1L1	CONDOM	HS	-	NORMAL	-	NORMAL	<3	INT		-	-	-	NF
133	2831	37	LAP	S	P2L2	TUBECT	HS	-	NORMAL	-	NORMAL	<3	NF		-	-	-	NF
134	2839	38	AVD	M	P2L2	TUBECT	PRI	-	NORMAL	BYC,PH	PUS, BYC,PH	<3	NF	C.KRU	-	-	-	VVC
135	2841	27	AVD	M	P2L2	IUCD	HS	-	CLUE	-	CLUE	3+	BV		-	-	-	BV
136	2844	37	AVD	M	P1L1	IUCD	PRI	-	NORMAL	-	NORMAL	<3	NF		-	-	-	NF
137	2846	24	AVD	M	P2L2	IUCD	HS	-	NORMAL	-	NORMAL	<3	NF		-	-	-	NF

S.NO	OP	AGE	SYMPTOM	MARITAL	PARITY	CONTRA	EDU	RISK	WET MOUNT	KOH	GRAM S	AMSEL'S	NUGENT'S	CULTURE	HIV	RPR	DM	DIAGNOSIS
138	2852	30	IM	M	P2L2	TUBECT	MID	-	NORMAL	BYC	BYC	<3	NF	C.GLAB	-	-	-	VVC
139	3381	21	AVD	M	P1L1	IUCD	HS	-	NORMAL	-	NORMAL	<3	NF		-	-	-	NF
140	3401	31	AVD	M	P2L2	TUBECT	HS	-	NORMAL	-	NORMAL	<3	INT		-	-	-	NF
141	3514	19	AVD	M	P1L1	NIL	HS	-	NORMAL	-	NORMAL	<3	NF		-	-	-	NF
142	3557	30	IM	M	P3L2	IUCD	HS	-	NORMAL	BYC,PH	PUS, BYC,PH	<3	NF	C.ALB	-	-	-	VVC
143	3761	30	INF	M	P0L0	NIL	HS	-	NORMAL	-	NORMAL	<3	NF		-	-	-	NF
144	3773	29	AVD	M	P2L2	TUBECT	HS	-	NORMAL	-	NORMAL	<3	NF		-	-	-	NF
145	3792	35	LAP	M	P2L2	TUBECT	MID	-	CLUE	-	CLUE	3+	BV		-	-	-	BV
146	3729	19	AVD	M	P1L1	NIL	HS	-	NORMAL	-	NORMAL	<3	NF		-	-	-	NF
147	3812	39	AVD	W	P1L1	IUCD	PRI	-	NORMAL	BYC,PH	PUS, BYC	<3	NF	C.ALB	-	-	+	VVC
148	1739	32	AVD	M	P1L1	OCP	PRI	HRP	NORMAL	BYC,PH	PUS, BYC,PH	3+	INT	C.TROP	-	-	-	VVC
149	1747	41	AVD	W	P3L3	TUBECT	MID	MSP	TV	-	TV	<3	NF	TV	-	-	-	TV
150	1756	28	AVD	M	P1L1	IUCD	HSEC	MSP	CLUE	-	CLUE	3+	BV		-	-	-	BV
151	1744	40	LAP	M	P2L2	TUBECT	MID	PVD	NORMAL	-	NORMAL	<3	NF		-	-	-	NF
152	1765	34	AVD	M	P2L2	CONDOM	PRI	VD	NORMAL	BYC.PH	PUS, BYC	<3	INT	C.KRU	-	+	-	VVC
153	1761	27	AVD	M	P1L1	IUCD	DEG	MSP	NORMAL	-	NORMAL	<3	NF		-	-	-	NF
154	1779	40	AVD	S	P2L2	CONDOM	ILL	FSW	CLUE	BYC.PH	CLUE,PUS, BYC	3+	BV	C.PARA	+	-	-	VVC+BV
155	1755	36	AVD	S	P2L2	CONDOM	PRI	FSW	NORMAL	-	NORMAL	<3	NF	TV	-	-	-	TV
156	1279	26	LAP	M	P1L1	NIL	HSEC	HRP	NORMAL	-	NORMAL	<3	INT		-	-	-	NF
157	1287	40	AVD	S	P3L3	TUBECT	MID	FSW	CLUE	BYC.PH	CLUE,PUS, BYC	3+	BV	C.TROP	-	-	+	VVC+BV
158	1282	23	LAP	UN	P0L0	OCP	HS	FSW	NORMAL	BYC	PUS, BYC	<3	INT	C.GLAB	-	-	-	VVC
159	1318	30	AVD	W	P1L1	IUCD	MID	VD	CLUE	-	CLUE	3+	BV		+	+	-	BV
160	1151	41	AVD	M	P2L2	TUBECT	MID	MSP	NORMAL	-	NORMAL	<3	INT		-	-	+	NF

S.NO	OP	AGE	SYMPTOM	MARITAL	PARITY	CONTRA	EDU	RISK	WET MOUNT	KOH	GRAM S	AMSEL'S	NUGENT'S	CULTURE	HIV	RPR	DM	DIAGNOSIS
161	1322	38	AVD	S	P2L2	TUBECT	ILL	FSW	CLUE	BYC.PH	CLUE,PUS, BYC	3+	BV	C.KRU	-	-	-	VVC+BV
162	1323	25	AVD	UN	P0L0	OCP	PRI	FSW	TV	-	TV	<3	NF	TV	-	-	-	TV
163	1359	18	AVD	M	P0L0	OCP	HSEC	HRP	NORMAL	-	NORMAL	3+	INT		-	-	-	NF
164	2120	40	AVD	S	P2L2	CONDOM	ILL	FSW	NORMAL	BYC.PH	PUS, BYC,PH	<3	NF	C.ALB	-	+	+	VVC
165	2579	24	AVD	M	P0L0	OCP	HS	MSP	CLUE	-	CLUE	3+	BV		-	-	-	BV
166	2612	23	AVD	M	P0L0	OCP	HSEC	MSP	NORMAL	-	NORMAL	<3	INT		-	-	-	NF
167	317	34	AVD	M	P2L2	TUBECT	MID	PVD	NORMAL	-	NORMAL	<3	NF		-	-	-	NF
168	321	37	AVD	S	P2L2	CONDOM	MID	VD	NORMAL	BYC.PH	PUS, BYC,PH	<3	INT	C.ALB	-	-	-	VVC
169	349	41	LAP	W	P2L2	CONDOM	PRI	FSW	CLUE, TV	-	CLUE	3+	BV	TV	-	-	-	TV+BV
170	357	31	AVD	M	P1L1	IUCD	PRI	MSP	NORMAL	BYC.PH	PUS, BYC,PH	<3	NF	C.ALB	-	-	-	VVC
171	360	26	AVD	M	P0L0	NIL	HS	MSP	NORMAL	-	NORMAL	<3	INT		-	-	-	NF
172	367	30	AVD	S	P1L1	IUCD	PRI	FSW	CLUE	-	CLUE	3+	BV		-	-	-	NF
173	411	27	AVD	M	P2L2	CONDOM	HS	MSP	NORMAL	-	NORMAL	<3	NF		-	-	-	NF
174	427	43	AVD	M	P2L2	CONDOM	PRI	VD	NORMAL	BYC.PH	PUS, BYC,PH	<3	INT	C.KRU	-	+	+	VVC
175	444	32	AVD	M	P2L2	TUBECT	HSEC	HRP	NORMAL	BYC.PH	PUS, BYC,PH	<3	NF	C.ALB	-	-	-	VVC
176	378	28	AVD	UN	P0L0	OCP	HS	MSP	TV	-	TV	3+	INT	TV	-	-	-	TV
177	507	36	AVD	W	P3L3	CONDOM	MID	FSW	CLUE	BYC.PH	CLUE,PUS, BYC	3+	BV	C.ALB	-	-	-	VVC+BV
178	521	34	AVD	M	P2L2	CONDOM	HS	HRP	CLUE	-	CLUE	3+	BV		-	-	-	BV
179	571	32	LAP	M	P1L1	IUCD	PRI	HRP	TV	-	TV	<3	NF	TV	-	-	-	TV
180	589	21	AVD	UN	P0L0	OCP	HS	MSP	NORMAL	BYC,PH	PUS, BYC,PH	<3	INT	C.TROP	-	-	-	VVC
181	601	39	AVD	W	P2L2	TUBECT	MID	FSW	NORMAL	BYC.PH	PUS, BYC,PH	<3	NF	C.ALB	-	-	-	VVC
182	606	24	LAP	M	P0L0	OCP	HS	MSP	CLUE	-	CLUE	3+	BV		-	-	-	BV
183	791	24	AVD	S	P0L0	CONDOM	HS	MSP	NORMAL	-	NORMAL	<3	NF		-	-	-	NF

S.NO	OP	AGE	SYMPTOM	MARITAL	PARITY	CONTRA	EDU	RISK	WET MOUNT	KOH	GRAM S	AMSEL'S	NUGENT'S	CULTURE	HIV	RPR	DM	DIAGNOSIS
184	826	38	LAP	S	P0L0	CONDOM	MID	PVD	NORMAL	BYC.PH	PUS, BYC,PH	<3	INT	C.ALB	-	-	-	VVC
185	829	28	AVD	M	P2L2	NIL	HS	HRP	NORMAL	-	NORMAL	<3	NF		-	-	-	NF
186	864	37	AVD	M	P2L2	TUBECT	MID	FSW	CLUE	BYC.PH	CLUE,PUS, BYC	3+	BV	C.ALB	-	-	-	VVC+BV
187	870	23	AVD	UN	P0L0	CONDOM	HS	MSP	NORMAL	-	NORMAL	<3	INT		-	-	-	NF
188	872	38	LAP	S	P2L2	TUBECT	MID	VD	NORMAL	BYC	PUS, BYC	<3	NF	C.GLAB	-	+	-	VVC
189	881	39	AVD	S	P3L3	NIL	ILL	FSW	NORMAL	BYC.PH	PUS, BYC,PH	<3	INT	C.ALB	-	-	-	VVC
190	892	22	AVD	M	P0L0	CONDOM	HSEC	MSP	CLUE	-	CLUE	3+	BV		-	-	-	BV
191	893	35	AVD	S	P2L2	CONDOM	MID	FSW	NORMAL	-	NORMAL	<3	NF		-	-	-	NF
192	910	36	AVD	W	P2L2	TUBECT	MID	FSW	NORMAL	BYC.PH	PUS, BYC,PH	<3	INT	C.TROP	-	-	-	VVC
193	922	34	AVD	S	P2L2	CONDOM	MID	MSP	NORMAL	-	NORMAL	<3	NF		-	-	-	NF
194	927	30	AVD	M	P2L2	CONDOM	PRI	HRP	CLUE	-	CLUE	3+	BV		-	-	-	BV
195	934	37	AVD	S	P2L2	TUBECT	ILL	FSW	BYC,PH	BYC.PH	PUS, BYC,PH	<3	NF	C.ALB	-	-	-	VVC
196	937	36	AVD	M	P3L2	TUBECT	MID	HRP	NORMAL	-	NORMAL	<3	INT		-	-	-	NF
197	618	41	LAP	W	P2L2	CONDOM	MID	FSW	NORMAL	BYC	PUS, BYC	<3	NF	C.GLAB	-	-	-	VVC
198	952	39	AVD	S	P2L2	CONDOM	PRI	FSW	NORMAL	BYC.PH	PUS, BYC,PH	<3	INT	C.PARA	-	-	-	VVC
199	973	30	AVD	M	P0L0	CONDOM	PRI	PVD	NORMAL	-	NORMAL	<3	INT		-	-	-	NF
200	985	37	AVD	S	P2L2	TUBECT	PRI	MSP	CLUE	BYC.PH	CLUE,PUS, BYC	3+	BV	C.ALB	-	-	-	VVC+BV