Chapter 4

Management of Abnormal Vaginal Discharge in Pregnancy

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Abstract

Abnormal vaginal discharge in a pregnant woman causes discomfort and increases risk of complications. Management of such patient is difficult as the physician will need to distinguish leucorrhoea of pregnancy from pathological vaginal discharge and also to decide on the drugs to prescribe that are not contraindicated in pregnancy.

The objective of the study is to discuss the prevalence, causes and treatment of abnormal vaginal discharge in pregnant women.

Searches from PubMed and using other scientific search engines were performed. The chapter was supported with findings from the authors' previous study on the same topic. In the study, high vaginal and endocervical swab samples were collected from 400 pregnant women with complaints of abnormal vaginal discharge and another 400 controls.

The result showed that the prevalence of abnormal vaginal discharge in pregnancy was 31.5%. Vulval pruritus, 200 (75%), was a significant feature ($\chi^2 = 1.011$, $P < 0.001$), and *Candida albicans*, 160 (40%), was the commonest cause.

Although antibiotic sensitivity testing was not done for *Candida albicans*, *all the microorganisms were sensitive to Augmentin®*

The prevalence of abnormal vaginal discharge in pregnancy was high and *C. albicans* was the commonest cause. Assessment of pregnant woman complaining of vaginal discharge for aetiology is necessary in order to give an appropriate treatment.

Keywords: Vaginal discharge, Pregnancy, Antibiotic treatment, Maiduguri, Nigeria

1. Introduction

Most pregnant women have vaginal discharges that are either physiologic or pathologic. The challenge to the clinician is to separate the vaginal infections with potentially serious input for pregnancy from annoying but not serious secretions, irritation and pruritus [1]. Infectious vaginitis is usually caused by yeast, such as *Trichomonas vaginalis*, bacterial vaginosis, gonorrhoea, *Chlamydia trachomatis*, *Mycoplasma*, Group B streptococcus or herpes [1]. Normal vaginal secretions consist of water, electrolytes, epithelial cells, microbial organisms, fatty acid and carbohydrate compounds [1, 2].The concentration of anaerobic bacteria is usually five times than that of aerobic organisms. The most prevalent organisms in the vagina are lactobacilli, *Streptococci*, *Staphylococcus epidermidis*, *Gadnerella vaginalis* and *Escherichia coli*. Anaerobic species that are frequently isolated include *Peptostreptococci*, anaerobic *lactobacilli* and *bacteroides* [3].

Vaginal pH, glycogen content and amount of secretion influence the quantity and type of organisms present in the vagina. Lactobacilli restrict the growth of other organisms by producing lactic acid, thus maintaining a low pH. These organisms also produce hydrogen peroxide, which is toxic to anaerobes. The normal vaginal bacterial population assists in inhibiting the growth of pathologic vaginal organisms. If the normal vaginal ecosystem is altered, there is a greater chance of proliferation of pathogenic organisms. The challenge of treating vaginitis in pregnancy is the necessity of making accurate diagnosis and treating correctly [2]. True infections (some of which can have dangerous effect on gestation) must be separated and distinguished from the exaggeration of physiologic discharge by pregnancy. Infection with bacterial vaginosis, *Chlamydia trichomonas* or Group B Streptococcus has been associated with septic abortion, premature rupture of membranes and premature delivery [2, 4].

2. Management of common causes of abnormal vaginal discharge in pregnancy

2.1. Vulvovaginal candidiasis

Vulvovaginal candidiasis (VVC) is a common cause of vaginal discharge worldwide [5, 6]. It is estimated that approximately 75% of women will experience an episode of VVC [7]. Candidiasis is caused by the fungus, *Candida* species. *Candida* species include *Candida albicans*, *Candida tropicalis, Candida pseudotropicalis, Candida krusei* and *Candida stellaloidea*. Other strains are *torulopsis, glabrata* and *rhodotromla*. *C. albicans* accounts for 60–80% of vaginal fungal infection [5–7], *Candida glabrata* accounts for 20% and *C. tropicalis* accounts for 6–23% [5]. Predisposing factors to VVC include pregnancy, diabetes mellitus, immunosuppressive therapy (cytotoxic drugs, steroids, etc.), antibiotics, oral contraceptives, immunodeficient conditions (HIV, cancer, chronic illness) and tight fitting and nylon undergarments [5]. Heat and moisture favour the growth of *Candida* species [8]. VVC can be sexually transmitted, and several studies reported an association between candidiasis and orogenital sex [7, 9].

C. albicans can be identified by culture from the vagina during pregnancy in approximately 25% of women [10]. While the prevalence of VVC in pregnancy in Zabrze of Poland is 42% [11], few studies in Africa reported the prevalence of 65% [12], 42% [13] and 23% [14] in Benin City of Nigeria, Addis Ababa of Ethiopia and Papua of New Guinea, respectively. Similarly, studies in India reported incidences ranging from 45% to 61% [15, 16].

Most patients with VVC will complain of vaginal discharge [5]. Dyspareunia, vulval pruritus and burning are the main symptoms [17]. Patients commonly complain of pruritus and burning after intercourse or upon urination. Erythema and oedema of the labia majora and minora and rashes on the perineum and thighs may be seen on physical examination, and a whitish, thick and curd-like vaginal discharge is usually present [17]. Recurrence requiring repeated treatment during pregnancy is likely [18].

The diagnosis is made on both clinical examination and laboratory identification of *Candida* by positive wet-mount test or potassium hydroxide (KOH) preparation [17]. In the wet-mount test, the spores and *Candida* are seen when vaginal discharge or scrapings from vulval lesions are mixed with normal saline and viewed under high-power magnifications. The presence of yeast blastospores or pseudohyphae can be detected in approximately 30–50% of patients with symptomatic VVC [17]. The addition of 10% KOH to the solution lyses white blood cells, red blood cells and vaginal epithelial cells, making the alkali-resistant branching budding hyphae of *Candida* easier to see[17]. Because vaginal pH usually remains normal in VVC, positive results from these two tests in combination with a normal vaginal pH are helpful in confirming the diagnosis. Most studies demonstrate that most of vaginal isolates are *C. albicans* [17]. Therefore, fungal cultures have not been used by most clinicians as part of the initial evaluation [17].

Various drug formulations are effective in treating both uncomplicated and complicated infections [10]. Both intravaginal and oral agents are available [10, 19]. Uncomplicated VVC includes sporadic or infrequent VVC, mild-to-moderate VVC, VVC with likely infecting agent being *C. albicans*, and non-immunocompromised patient. Complicated VVC is recurrent candidal infection or severe infection or non-albicans candidiasis (*C. tropicalis, C. glabrata*, etc.), VVC in uncontrolled diabetes mellitus, VVC with associated immunosuppression, VVC with debilitation or VVC in pregnancy [10]. Women who have four or more candidal infections during a year are classified as having complicated disease [9]. Intravaginal agents used in the treatment of VVC are 5 g of 2% butoconazole cream intravaginally for 3 days or 5 g (sustained-release) once; 5 g of 1% clotrimazole cream for 7–14 days or 100 mg tablet intravaginally for 7 days; 5 g of 2% miconazole cream intravaginally for 7 days or 100 mg suppository intravaginally for 7 days or 200 mg suppository for 3 days or 1200 mg suppository once; Nystatin 100,000 U tablet intravaginally for 14 days; 5 g of 6.5% tioconazole ointment intravaginally once; 5 g of 0.4% terconazole cream intravaginally for 7 days or 0.8% cream 5 g intravaginally for 3 days or 80 mg suppository intravaginally for 3 days. Oral agent is fluconazole 150 mg oral tablet once.

Prolonged local intravaginal therapy regimens and addition of oral fluconazole may be required to treat non-albicans VVC [7]. Fluconazole, 100–200 mg weekly for 6 months, is also

the drug for prevention of recurrent VVC, whereas 600 mg boric acid gelatine capsule intravaginally daily for 2 weeks is useful in the management of non-albicans recurrent VVC [7].

2.2. Anaerobic bacterial infection and bacterial vaginosis

Vaginal flora of a normal asymptomatic reproductive-aged woman includes multiple aerobic or facultative species as well as obligate anaerobic species [9]. Of these, anaerobes are predominant and outnumber aerobic species approximately 10–1 [20]. These anaerobes include gram-negative organisms such as *Prevotella*, *Bacteroides*, *Fusobacterium* species, and *Veillonella* species and gram-positive bacilli such as *Propionibacterium* species, *Eubacterium* species and *Bifidobacterium* species [9, 20]. These anaerobic bacteria cause non-specific vaginitis [5].

Bacterial vaginosis is characterised by a shift from normal vaginal population of lactobacilli to anaerobes such as *G. vaginalis, Prevotella, Bacteroides* and *Mobiluncus* species and other bacteria such as *Mycoplasma* and *Ureaplasma* species [20]. It is one of the most frequent conditions encountered in reproductive health clinics throughout the world [20]. The condition had been previously called *Haemophilus vaginalis* vaginitis, non-specific vaginitis and *G. vaginalis* vaginitis [5, 21].

Bacterial vaginosis has been strongly associated with poor pregnancy outcomes such as preterm delivery and low birth weight infants, and several studies have now established the associations between bacterial vaginosis, human immunodeficiency virus and puerperal sepsis [20, 22].

Bacterial vaginosis appears to be particularly common in Sub-Saharan Africa where several studies have reported high prevalence rates, ranging from 20–49% among women presenting to STD clinics with vaginal discharge to 21–52% among pregnant women attending antenatal clinic [20]. These are very much higher than the rates reported from industrialised countries with 13% in the United Kingdom [23], 11% in London [20] and 15–30% in the United States [24].

Bacterial vaginosis usually occurs in sexually active patients. Some of the other risk factors include multiple sexual partners, low socioeconomic status, lesbians, presence of intrauterine device and prior STD [5]. It is still debatable whether it is sexually transmitted; however, supporting this is the recovery of *G. vaginalis* in the urethral of male partners [5].

Bacterial vaginosis is characterised by a malodorous, profuse, thin, homogenous yellow, white or grey discharge that is adherent to the anterior and lateral vaginal walls. Typically, the patient may complain of a fishy odour during or shortly after coitus and also during menses. The alkaline nature of blood or semen (pH > 7) brings about a transient increase in the vaginal pH, and this causes the release of amines, which the patient perceives as fishy odour. This typical discharge may be found on examination in some patients who in fact have not complained of a vaginal discharge [25]. The fishy smell of the discharge is the main problem and is often responsible for sexual disharmony between partners. Vulvitis and pruritus are very minimal or totally absent. Nearly half of patients with BV have no symptoms. Obstetric complications include premature rupture of foetal membranes, late miscarriage and postpartum endometritis, whereas pelvic inflammatory disease, post-hysterectomy cuff infection and postabortal sepsis are some of the gynaecological complications [25].

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Diagnosis of BV can be based on the Amsel's clinical criteria or the microbiological Nugent's scoring technique [26, 27]. In Amsel's criteria, three of the following are required to diagnose BV: (1) homogenous vaginal discharge; (2) vaginal pH greater than 4.5; (3) positive Whiff test and (4) presence of clue cells on microscopy. The Nugent's method relies on the identification of categories of vaginal microflora based on quantitative assessment of a vaginal gram-stained smear. The Nugent's method has been extensively validated in industrialised countries where assessment of vaginal microflora is an important step in understanding the pattern of flora association with BV. Culture is the least accurate in making a diagnosis of BV as there is overgrowth of many vaginal organisms in this condition [5]. Though virtually, all patients with BV have *G. vaginalis* isolated on culture, it must also be noted that the organism can also be cultured in 40–50% of women with normal flora [5].

In pregnancy, BV should be treated with metronidazole 250 mg three times a day (alternatives; metronidazole 2 g single dose, clindamycin 300 mg twice a day; or metronidazole gel) [5]. The standard treatment of BV in non-pregnant women is oral metronidazole 500 mg twice daily for 7 days; clindamycin cream 2% on applicator ful (5 g) intravaginally at bedtime for 7 days or metronidazole gel one applicator ful (5 g) intravaginally once or twice a day for 5 days [5, 10, 28].

2.3. Trichomoniasis

Trichomoniasis is the commonest sexually transmitted disease worldwide [5]. It was originally thought to be innocuous but has now been found to be associated with preterm labour, premature rupture of membranes, increased perinatal loss and pelvic inflammatory disease (PID) [5, 29]. *T. vaginalis* can be identified during perinatal examination in as many as 20% of women [29].

T. vaginitis is caused by the trichomonas organism, which is a small, flagellated, motile and anaerobic protozoan. The particular trichomonad responsible for vaginitis is *T. vaginalis*, which is the type found in the vagina. Other trichomonads, which include *Trichomonas buccalis* found in the mouth and *Trichomonas hominis* found in the anal canal and rectum, are known but do not cause vaginal discharge because they cannot survive in the vagina. *T. vaginalis* has been demonstrated in the male urethra and prostate gland.

T. vaginalis is usually transmitted sexually. The organism may survive for several hours in urine, wet towels and even on toilet seats. The possibility of transmission by these routes had been suggested but not completely proven [5]. Incubation period is 4–20 days with an average of 7 days. Males are usually asymptomatic, but they can easily infect treated female.

The prevalence of trichomoniasis in pregnancy has been found to be 7.5–19% [14, 30]. A prevalence of 10.1% has been reported from the Gambia, West Africa [20].

The vaginal discharge of trichomoniasis is malodorous, frothy and profuse, thin creamy or slightly greenish and may cause itching. The classic yellow–green discharge is found in 20–50% of patients [5]; more often, the discharge is grey or white. The patient may also complain of dyspareunia, postcoital bleeding, pruritus vulvae, frequency of micturition and dysuria.

Characteristically, vulvitis is minimal or absent compared with candidiasis. On speculum exam, apart from the discharge, a cervical erosion may be seen, and in severe cases, multiple, small punctuate haemorrhages and swollen papillae may be found on the cervix ("straw berry" cervix) and vagina [17].

The vaginal pH is usually 5–5.5 in trichomonas infection [17]. Applying litmus to the unlubricated speculum after it has been withdrawn from the vagina easily tests the pH. A saline wet mount of the swab taken from the vagina or cervix will show motile flagellated protozoa and leucocytes. Wet mount alone detects 64% infection in asymptomatic women, 75% of those with clinical vaginitis and 80% of those with characteristic symptoms [5]. The use of culture (Feinberg-Whittington or Diamond culture) gives a sensitivity of 86–97% [5]. Pap smear has a detection rate of about 50–86% [5]. Monoclonal antibody staining is also used. It is sensitive and is reported to detect 77% of those missed on wet mount [5].

Metronidazole is effective in eradicating *T. vaginalis* administered orally in a single 2 g dose [5, 10]. Ootrimazole, which is both a fungicide and trichomonacide, can be used intravaginally usually in pregnancy in the same dosage regime as in candidiasis [5]. In persistent infection, it is best to treat the patient and her male sexual partner simultaneously.

2.4. Gonorrhoea and chlamydial infection

Chlamydia and gonorrhoea can both cause vaginal discharge in pregnancy and a major cause of morbidity among women in developing countries [31]. Both infections have been associated with pregnancy-related complications [32]. These two conditions are prevalent worldwide particularly in Africa [20]. They are a major cause of acute pelvic inflammatory disease, infertility and adverse pregnancy outcomes [20].

The prevalence of *Chlamydia* and gonorrhoea among pregnant women in Africa, in several studies, is between 6–13% [4, 33] and 2–8% [4, 34], respectively. According to the WHO, globally new cases of *C. trachomatis* infection have been estimated as 92 million, including 19 million in Sub-Saharan Africa [35–37]. In Maiduguri, North-eastern Nigeria, Amin et al. reported a prevalence of 9% [38].

Chlamydia is characteristically asymptomatic [39, 40]. About one-third of patients may have symptoms including mucopurulent vaginal discharge [5]. The role of *Chlamydia* in infertility is well documented [39–42]. Tubal pathology in *Chlamydia* infection is the cause of infertility in 10–30% of couples in developed countries and in up to 85% in developing countries [36, 37, 43, 44]. The main cause of tubal pathology is PID. Several different methods to diagnose chlamydial infection are available. Great studies have been performed in the areas of reliable methods of diagnosis [40, 45]. *Chlamydia* culture is considered as the gold standard because it has near 100% specificity [40, 45]. Because only viable infectious chlamydial elementary bodies are detected by culture, this is the method of choice for medico-legal issues. The disadvantages of culture include its low sensitivity and is that it depends on the laboratory inter-personal experience [46]. Non-culture methods include enzyme immunoassay (EIA), direct fluorescent staining with monoclonal antibodies (DFA), nucleic acid amplification tests (NAATs) and

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nucleic acid hybridisation techniques. In the management of chlamydial infection, both the patient and her infected sexual partner must be treated. It is, therefore, important to screen the sexual partners and treat those who are infected. With the advent of single-dose therapy, patients are now diagnosed and treated with the highest convenience and reliability [47]. Antimicrobial groups effective against *C. trachomatis* include the tetracyclines, macrolides, quinolones and penicillins [47].

Most women with gonorrhoea are asymptomatic [48]. When symptoms occur, they are localised to the lower genitourinary tract and include vaginal discharge, urinary frequency or dysuria and rectal discomfort. The incubation period is only 3–5 days [48]. The vulva, vagina, cervix and urethra may be inflamed and may itch or burn. Specimens of discharge from the cervix, urethra and anus should be taken for culture from the symptomatic patients. A stain of purulent urethra exudates may demonstrate gram-negative diplococci in leucocytes. Similar findings in a purulent cervical discharge are less conclusively diagnostic of *Neisseria gonorrhoea*. Gram-negative diplococci that are oxidase positive and obtained from selective media (Thayer-Martin or Transgrow) usually signify *N. gonorrhoea*. Carbohydrate fermentation tests may be performed, but in addition to being time consuming and expensive, they occasionally yield other species of Neisseria. Therefore, cultures are reported as presumptive for *N. gonorrhoea*. In addition, other techniques for detecting gonorrhoea include EIA for cervical swab or urine specimens, DNA probes for endocervical swabs and NAATs for endocervical swabs, liquid Papanicolaou specimens, vaginal swabs and urine specimens. Any patient with gonorrhoea must be suspected of having other STDs and managed accordingly. Treatment should cover *N. gonorrhoea*, *C. trachomatis* and incubating syphilis. Dual therapy has contributed greatly to the declining prevalence of *Chlamydia* infection. Therefore, if chlamydial infection is not ruled out, doxycycline (for non-pregnant women) should be added to ceftriaxone or azithromycin.

3. Outcome of study on abnormal vaginal discharge among pregnant women conducted in Maiduguri, Borno State in North-eastern Nigeria

3.1. Goal and objectives

The general objective of the study is to detect the clinical features associated with abnormal vaginal discharge and antibiotic sensitivity pattern of the causative microorganisms in pregnant women to improve the early diagnosis and prompt treatment. The specific objectives were as follows:

1. To determine the prevalence of abnormal vaginal discharge as a presenting complaint in pregnancy
2. To determine the frequency of bacterial causes of abnormal vaginal discharge in pregnancy and symptoms associated with it
3. To evaluate the sensitivity of microbial isolates from the vaginal discharge to antibiotics

3.2. Methodology

Borno State lies between latitude 10° and 14° north and longitude 14° and 45° east. It is located in the north-eastern part of Nigeria. Maiduguri is the capital city. The University of Maiduguri Teaching Hospital (UMTH) is a tertiary health institution and is the only functional teaching hospital in the north-eastern zone of Nigeria. The 2006 Nigerian provisional census puts the population of Borno State at 4,151,193 with 1,990,036 females [49].

It was a cross-sectional analytical study. The study population consisted of pregnant women presenting to the antenatal clinic with complaint of abnormal vaginal discharge while pregnant women without complaints of abnormal vaginal discharge attending the antenatal clinic of the hospital served as controls. A sample size of 800, consisting of 400 cases and 400 controls, was obtained using Taylor's and Kish's formulas [50]. Information on sexual and reproductive risk factors and symptoms was obtained. Vaginal examination was performed, and discharge was assessed. Endocervical and high vaginal swabs were collected and immediately processed in accordance with microbiological standard. Infection with *Candida* species was diagnosed by microscopy of a saline mount, which showed a highly refractile, round or oval budding yeast cells, and gram-stained smear of material from the vagina showed gram-positive pseudohyphae with budding yeast cells; *T. vaginalis* was diagnosed by microscopy of a saline mount for actively motile, spear-shaped flagellates, whereas bacterial vaginosis was diagnosed using Amsel's criteria [26]. *N. gonorrhoea* was identified by typical colonial morphology, reactions to gram stain, positive oxidase test and sugar fermentation. The antibiotic sensitivity of isolates was tested by the agar diffusion method on chocolate agar plates using oxoid multi discs with standard antibiotic concentration.

The computer program SPSS V 20.0 (2010) Inc., Illinois, United States was used to analyse the results; the association between organisms and studied variables was compared using chi-square (χ^2) and Fisher's exact tests while P value <0.05 was considered significant at 95% confidence level.

3.3. Results

During the period of study, 1280 pregnant women were seen at the antenatal booking clinic among which 800 satisfied the inclusion criteria. Four hundred of the pregnant women complained of abnormal vaginal discharge (cases), whereas 400 had no complaint of vaginal discharge, giving a prevalence of abnormal vaginal discharge in pregnancy of 31.5%.

Table 1 shows the clinical features associated with vaginal discharge in the study group. Vulval pruritus was present in 266 patients, and 200 (75%) of them complained of vaginal discharge, whereas 66 (25%) were in the control group. There was a significant association between pruritus and vaginal discharge ($\chi^2 = 1.011$, $P < 0.001$). As much as 63% of those without itching were in the control group. Dysuria showed statistically significant association with vaginal discharge ($\chi^2 = 44.008$, $P < 0.000$) with 74 (83%) of the 89 patients who complained of dysuria having vaginal discharge. There was no statistically significant association between dyspareunia and vaginal discharge ($\chi^2 = 2.082$, $P = 0.149$). The only patient that had vulval wart

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complained of vaginal discharge, there was, however, no statistically significant association between vulval warts and abnormal vaginal discharge.

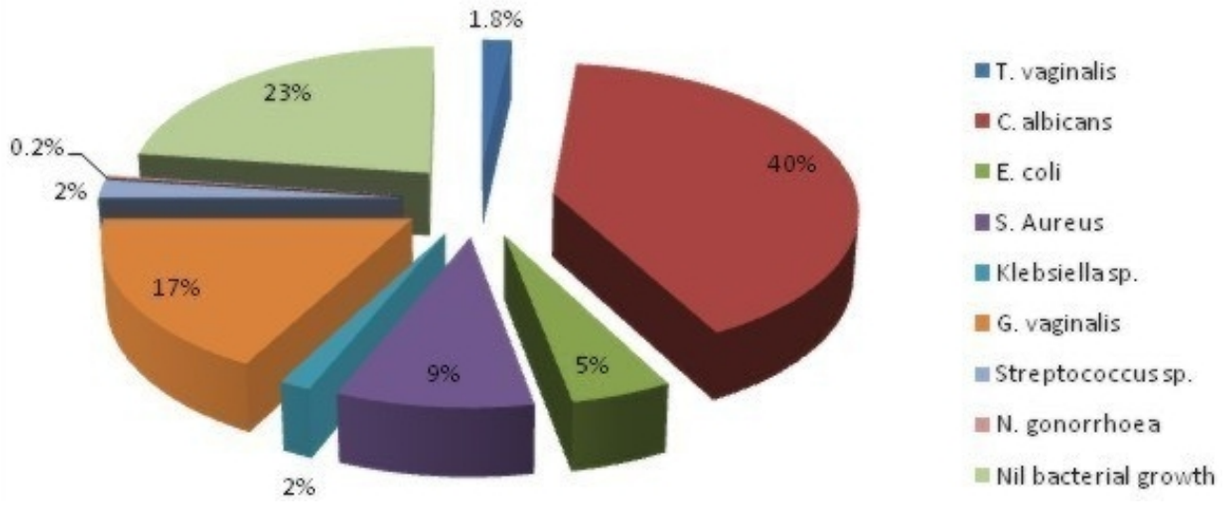

Figure 1. (Findings from culture of ECS/HVS from pregnant women with abnormal vaginal discharge (N=400)) shows outcome of culture and microscopy from vaginal discharge specimens collected from women with complaint of vaginal discharge. The prevalence of positive culture was 77% (308) among the cases and 21.3% (85) among the control group. Of the 400 patients with abnormal vaginal discharge, the commonest microorganism found was *C. albicans*, 160 (40%), whereas *N. gonorrhoea* infection was the least, 1(0.2%). *E. coli* was isolated in 20 (5%), *T. vaginalis* in 7 (1.8%), *Staphylococcus aureus* in 36 (9%), *Klebsiella* species in 8 (2%), *G. vaginalis* in 68 (17%) and *Streptococcus* species in 8 (2%) of the pregnant women. Samples from 92 (23%) patients had negative culture.

Feature		Case	Control	Total
1. Vulval itching	Yes	200 (75 %)	66 (25 %)	266
	No	200 (37 %)	334 (63 %)	534
$x^2 = 1.011$, $P < 0.001$				
2. Dysuria	Yes	74 (83 %)	15 (17 %)	89
	No	326 (46 %)	385 (54 %)	711
$x^2 = 44.008$, $P = 0.000$				
3. Dyspareunia	Yes	25 (61 %)	16 (39 %)	41
	No	375 (49 %)	384 (51 %)	759
$x^2 = 2.082$, $P = 0.149$				
4. LAT[a]	Yes	24 (60 %)	16 (40 %)	40
	No	376(49 %)	384 (51 %)	760
$x^2 = 1.684$, $P = 0.194$				
5. Vulval warts	Yes	1 (100 %)	0 (0 %)	1
	No	399 (49.9 %)	400 (50.1 %)	799
$x^2 = 1.001$, $P = 0.317$				

[a]Lower abdominal tenderness.

Table 1. Clinical features associated with vaginal discharge in the study group ($N = 800$).

Table 2 shows the association between the bacterial isolates and their antibiotic sensitivity patterns. *G. vaginalis* was sensitive to augmentin and ofloxacin in 64% of cases. *Streptococcus* sp. was most sensitive to augmentin and erythromycin in 92% (12/13) and 84% (11/13) of cases, respectively. *N. gonorrhoea* was sensitive to augmentin and ofloxacin in 100% of cases but was 100% resistant to other antibiotics. *E. coli* was sensitive to cefuroxime and gentamicin in 62% and 65% of cases, respectively. Most of the microbial isolates were resistant to ampicillin and norbactam. Only augmentin had greater than 60% sensitivity rate to all the isolated microorganisms.

Antibiotics	***S. aureus***	***Klebsiella sp.***	***E. coli***	***G. vaginalis***	***Streptococcus sp.***	***N. Gonorrhoea***
Amoxicillin	16.9	10.5	14.2	35.9	0	0
Augmentin[a]	86	61.5	75	64	92	100
Ofloxacin	75	55	12.5	64	25	100
Ciprofloxacin	64	51	25	9.2	22.5	0
Erythromycin	45	1.2	25	72	84	0
Cefuroxime	50	50	62	26	50	0
Gentamicin	61	64	65	21	30	0
Ampicillin	25	45.5	0	7.2	0	0
Norbactam	17.5	25	4.2	0	0	0

[a]Amoxicillin-clavulanic acid.

Table 2. Antibiotic sensitivity rate (%) of isolated bacteria.

4. Conclusion

Vaginal discharge in pregnancy is common, but distinguishing abnormal vaginal discharge from normal leucorrhoea of pregnancy is challenging. Since findings have showed that the trio of vaginal candidiasis, trichomoniasis and bacterial vaginosis are common causes of abnormal vaginal discharge in pregnancy; efforts must be made to exclude these conditions in pregnant patients presenting with vaginal discharge so that appropriate treatment can be instituted timely. Finally, gonococcal infection must also be excluded since though it is less prevalent than others, it is a major cause of morbidity in women in developing countries.

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