Bacterial vaginosis and *Candida albicans* vaginitis among women in Ramadi City

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Abstract

Patients and methods: A prospective study was performed involving 65 women who consecutively attended Gynecological ward in Maternity and Child Hospital in Ramadi for complaints of genital malodour and/or abnormal vaginal discharge. Bacterial vaginosis was diagnosed in 30 women on the basis of four diagnostic criteria: vaginal pH> 4.7, homogenous vaginal discharge, a positive amine test and presence clue cells. This diagnosis was further strengthened by microscopy of wet preparation and stained smears and culture of vaginal discharge under aerobic, facultative anaerobic, and anaerobic cultural condition.

Results: A total of 65 high vaginal specimens, bacterial vaginosis was diagnosed in 30 (46.2%) women, vulvovaginal yeast fungi infection in 12 (18.5%) women, other aetiology in 16 (24.6%) and in Seven (10.8%) women showing sterile vaginal discharge. The sensitivity for *in vitro* bacterial vaginosis detecting criteria was > 90% with the exception of homogenous discharge (73.3%) and their specificity was > 90% except vaginal pH > 4.5 (56%). ***Gardenerella vaginalis*** 27 (90%) was the predominant bacteria isolated in women suffering from bacterial vaginosis followed by ***Staphylococcus epidermidis*** (63.3%) and ***Streptococcus faecalis*** 13 (43.3%). ***Candida albicans*** 11 (91.6%) was the major cause of vulvovaginal yeast fungi infections followed by ***Saccharomyces cerevisiae*** 1 (8.3%).

Conclusions: The four diagnostic criteria for bacterial vaginosis supported by gram staining and facultative anaerobic culture should be introduce into laboratory routine procedures to differentiate symptomatic carrier (true infection) caused largely by ***Gardenerella vaginalis*** from asymptomatic carrier (colonization).Also, there was a strong association between diagnosis of bacterial vaginosis and the occurrence of ***Gardenerella vaginalis***. Further, there was no difference between women with or without bacterial vaginosis in regards to contraceptive methods used (except for use of an intrauterine device). Further more, Diabetes mellitus, hygienic condition were the most predominant risk factors associated with highly frequently fungal isolates, ***Candida albicans***.

Key words:-Bacterial Vaginosis, *Candida albicans* vaginitis

Introduction:

Abnormal vaginal discharge is a common complaint in primary health care and bacterial vaginosis is the most common condition seen. It is represent a complex change in vaginal flora characterized by a reduction in the prevalence and concentration of hydrogen peroxide-producing lactobacilli[1]. and an increase in the prevalence and concentration of the following:

Gardnerella vaginalis; Mobiluncus species; Mycoplasma hominis; anaerobic gram-negative rods belonging to the genera Prevotella , Porphyromonas , and Bacteroides;and Peptostreptococcus species[2].

Ever since Gardner and Duke in 1955regarded Gardenerella (originally Haemophilus vaginalis) as the aetiologic agent of bacterial vaginosis, this concept has been challenged. The role of Gardenerella vaginalis in bacterial vaginosis has been questioned, because of the high carrier rate in asymptomatic women3.

Also, Much of the controversy surrounding bacterial vaginosis can partly be related to the emphasis placed on the mere presence of Gardenerella vaginalis in the vagina rather than on the entire disease entity, including the change in flora in such women4. Thus, this study has been designed based on in vitro detection criteria to differentiate the pathological presence of Gardenerella vaginalis as the causative agent of bacterial vaginosis from their nature colonization, to study the vaginal flora of women with bacterial vaginosis and their effectiveness by contraception methods including intra uterine devices. Further, to isolate mycological causes of yeast fungi vaginitis and its correlation with their risk factors.

Patients and Methods:

Sixty-five women consecutively attending Maternity and Child Hospital in Ramadi during the period from August, 2007 to February, 2008. All patients complained of genital malodour and/or abnormal vaginal discharge. Out of 65 women, 30 (46.2%) were diagnosed as having bacterial vaginosis if at least three of the following criteria laid down by Amsel, et al., 1983[3] and published by National guidline for the management of bacterial vaginosis5 were present : a vaginal pH > 4.7, a positive whiff-amine test, defined as the presence of a fishy odor when 10 percent KOH is added to vaginal discharge samples, the presence of clue cells in wet mount preparations, and a characteristic homogenous adherent vaginal discharge. This diagnosis was further strengthened by microscopy of stained smears and culture of vaginal discharge.

Vaginal discharge from the posterior fornix was spread on two glass slides which were air dried, fixed and stained with Gram stain. For cultures of anaerobic, aerobic, facultative anaerobic and yeast fungi, samples were collected from the posterior vaginal fornix, using sterile cotton swabs and cultures for anaerobic bacteria were made on anaerobic blood agar plates and incubated for 72 hr. at $37^{o}C$ in an anaerobic gas Pack system. Cultures for aerobic and facultative anaerobic bacteria were made on nutrient agar, blood agar and MacKoncy agar and blood agar in an atmosphere of 10% CO_2. Yeast fungi were cultured on Sabouraud's agar, incubated for 72 hr. at $37^{o}C$.

Results:

A total of 65 high vaginal specimens, bacterial vaginosis was diagnosed in 30 (46.2%) women, vulvovaginal yeast fungi infection 12 (18.5%), other aetiology in 16 (24.6%) patients and in Seven (10.8%) women showing no growth of pathogenic microbes on the culture plates. The sensitivity for in vitro bacterial vaginosis detecting criteria was > 90% with the exception of homogenous discharge (73.3%) and their specificity was > 90% except vaginal pH > 4.5 (56%). Gardenerella vaginalis 27 (90%) was the predominant bacteria isolated from women suffering from bacterial vaginosis followed by Staphylococcus epidermidis 19 (63.3%) and Streptococcus faecalis 13 (43.3%). Candida albicans 11 (91.6%) was the major cause of vulvovaginal yeast fungi infections followed by Saccharomyces cerevisiae in one patient (8.3%)(table 1).

Table 1: Microbes isolated in women with bacterial vaginosis, or yeast fungi infection and women with abnormal discharge of other aetiology.

Microbes	Bacterial vaginosis (n=30)	Yeast fungi infections (n=12)
Gardenerella vaginalis	27	3
Staph.epidermidis	19	2
Strep.faecalis	13	-
Candida albicans	-	11
***Klebsiellae* spp.**	10	-
Peptostreptococcus	7	-
Escherichia coli	5	-
***Bcateroides* spp.**	4	-
Staph. aureus	4	-
Pseudomonas aeruginosa	2	-
Lactobacilli	1	8
Saccharmyces cerevisiae	-	1

Table 2: Number of patients with risk factors for vulvovaginal yeast fungi infection.

Risk factors	Yeast fungi vaginitis (n=12)
Antibiotic use	2(16.7%)
Pregnancy	3(25%)
Oral contraceptive	3(25%)
Diabetes mellitus	7(58.3%)
Corticosteroides	0(0.0)
Hygienic condition	6(50%)

Discussion:

Much of the controversy surrounding bacterial vaginosis can partly be related to the emphasis placed on the mere presence of *Gardenerella vaginalis* in the vagina rather than on the entire disease entity, including the change in flora in such women [4]. In the present study, the diagnosis of bacterial vaginosis was based on the appearance of discharge at speculum examination, vaginal pH measurement, microscopic examination of wet mount preparations of vaginal secretion and amine

tests. Results of these tests corresponded in all to the microbiological findings in stained smears and cultures. Thus, if three of four test parameters were positive, the abnormal vaginal discharge presumed to be due to bacterial vaginosis.

While the cultures for *Gardenerella vaginalis* are positive in almost all cases, in which the organism may be detected in 50 to 60 percent of healthy asymptomatic women. Thus, examination the presence of three of the following four diagnostic criteria confirmed latterly by the cultural techniques is necessary to differentiate true infection from colonization : 1) homogeneous, grayish-white discharge, 2) vaginal pH greater than 4.5, 3) positive whiff-amine test, which is confirmed by the presence of a fishy odor when 10 percent KOH is added to vaginal discharge samples, 4) presence of Clue cells on saline wet mount (or gram stain).

Although the first three criteria are sometimes present in patients with trichomoniasis. The presence of clue cells is the single most reliable predictor of bacterial vaginosis [6]. Clue cells are vaginal epithelial cells studded with adherent coccobacilli that are best appreciated at the edge of the cell. Gram staining of vaginal secretions is more reliable than wet mount for the diagnosis of bacterial vaginosis, with a sensitivity and specificity of 93 and 70 percent, respectively [7].

Candida albicans is responsible for 80 to 92 percent of episodes of vulvovaginal candidiasis [8]. Some but not all investigators have reported an increased frequency of other candida species, particularly *C. glabrata* [9], possibly due to widespread use of over-the-counter drugs, long-term use of suppressive azoles, and the use of short courses

of antifungal drugs. The mechanism by which *Candida* species cause symptomatic disease is complex, including host inflammatory response to invasion and yeast virulence factors (eg, elaboration of proteases).

The study result showed that 7 (58.3%) patients with vulvovaginal yeast fungi infection were suffering from diabetes mellitus (table 2). It is well known that diabetes mellitus is the most common underlying disease associated with this condition and this disease is believed to increase the risk of vulvovaginal candidiasis by promoting vulvovestibular colonization with candida in women, encouraging fungal growth due to glycosuria, and by impairing the ability of neutrophils and monocytes to phagocytize

Candida then kill the organism using myeloperoxidase, hydrogen peroxide, and superoxide anion (10).

Data showed that 3 (25%) pregnant women out of 12 patients were suffering from vulvovaginal yeast fungi infection. This may be due to change in cell-mediated immunity (decreased T cell activity), altered glucose metabolism, and provision glycogen-rich vaginal epithelium (11).

Two (16.7%) out of 12 patients with vulvovaginal yeast fungi infection were under broad-spectrum antibiotics treatment of third generation cephalosporins or ciprofloxacin. It is well known that antibiotics suppress the endogenous bacterial flora and allow *Candida* colonization of the gastrointestinal tract from 30% in normal adults to nearly 100% in antibiotic-treated individuals ([10,12]. A minority of women are prone to vulvovaginal candidiasis while taking antibiotics; inhibition of normal bacterial flora by broad-spectrum antibiotics favors yeast growth. Vulvocandidiasis also has been reported in a significant number of women treated with oral metronidazole or vaginal antimicrobials for bacterial vaginosis [13].

The present finding also showed that 3 (25%) women out of 12 patients with vulvovaginal yeast fungi infection were use oral contraceptive. This result is in agreement with the result observed by Foxman[12] who demonstrated that the risk of vulvovaginal candidiasis may be higher in women who use oral contraceptives containing high levels of estrogen.

Microscopic observations may be negative in up to 50 percent of patients with confirmed vulvovaginal candidiasis [13]. Although, empiric therapy is often considered in women with typical clinical features, a normal vaginal pH, and no other pathogens visible on microscopy, every effort should be made to confirm the diagnosis and avoid empiric therapy. Culture should be performed in patients with persistent or recurrent symptoms.

On the other hand, there is no evidence that women with recurrent vulvovaginal candidiasis have a vaginal flora deficient in lactobacilli [14]. One study suggested that ingestion of yogurt containing live *Lactobacillus acidophilus* decreased the rate of candidal colonization and symptomatic relapse[15].

It has generally been claimed that the presence of a foreign body can facilitate the establishment of endogenous anaerobic infections. 9 (30%) out of 30 of the women with the bacterial vaginosis studied were IUD users, the same finding mentioned by other researcher [3]. Recent reports indicate that a women using an IUD has a threefold increase in risk for bacterial vaginosis. It is believed that only sexual active women get it. Perhaps semen alkalizes the vagina and predispose for it[16].

The study concluded that the four diagnostic criteria for bacterial vaginosis supported by gram staining and facultative anaerobic culture should be introduce into laboratory routine procedures to differentiate symptomatic carrier (true infection) caused largely by *Gardenerella vaginalis* from asymptomatic carrier (colonization). Also, there was a strong association between diagnosis of bacterial vaginosis and the occurrence of *Gardenerella vaginalis.* Further, there was no difference between women with or without bacterial vaginosis in regards to contraceptive methods used (except for the use of an intrauterine device). Further more, Diabetes mellitus, hygienic condition were the most predominant risk factors associated with highly frequently fungal isolates, *Candida albicans*.

References:

1.Eschenbach DA, Davick PR, Williams BL, et al. Prevalence of hydrogen peroxide producing *Lactobacillus* spp. in normal women and women with bacterial vaginosis. J Clin Microbiol 1989; 27:251.

2.Hill GB. The Microbiology of bacterial vaginosis. Am J Obstet Gynecol 1993; 169: 54.

3.Amsel R, Totten PA, Spigel A, Chen KC. Non-specific vaginitis. Diagnostic criteria and microbial and epidemiological associations. Am J Med 1983; 74: 14-22.
4.Holst E, et al. Bacterial Vaginosis: Microbiological and clinical finding. Eur J Clin Microbiol 1987; 6: 536-41.
5.National guideline for the management of bacterial vaginosis. Clinical Effectiveness Group Association for Genitourinary Medicine and the Medical Society for the study of Veneral Disease 2001.
6.Eschenbach DA, Hillier Sl, Critchlow C, et al. Diagnosis and clinical manifestations of bacterial vaginosis. Am j Obstet Gynecol. 1988; 158: 819.
7.Nurgent RP, Krohn MA, Hillier Sl. Reliability of diagnosis bacterial vaginosis is improved by a standardized method of Gram-stain interpretation. J Clin Microbiol 1991; 29: 297.
8.Odds Fc. Candidiasis of the genitalia. In: Odds Fc. Candida and candidiasis: A review and bibliography, London: Bailliere Tindall 1988; 2nd ed: p 124.
9.Horowitz BJ, Giaquinta D, I to S. Evoloing pathogens in vulvovaginal candidiasis: implication for patient care. J Clin Pharmacol 1992; 32-248.
10. Mitchell TG. Medical Mycology. In: Jawetez, Melnick and Adelberg's Medical Microbiology. By: Brooks GF, Butel JS, Morse SA. California: Appleton and Lange 2004, 23 ed. P530.
11. Fleury FJ, Recurrent candida vulvovaginitis. chemotherapy 1982; 28 suppl 1: 48.
12. Foxman B. The epidemiology of vulvovaginal candidiasis: Risk Factors. Am J Public Health 1990; 80: 329.
13. Sobel JD. Epidemiology and pathogenesis of recurrent vulvovaginal candidiasis. Am J Obstet Gynecol 1985; 152: 924.
14. Spbel JD, Chaim W. Vaginal microbiology of women with acute recurrent vulvovaginal candidiasis. J Clin Microbiol 1996; 34: 2497.
15. Hilton E, Isenberg HD, Alperstein P, et al. Ingestion of yogurt containing Lactobacillus acidophilus as prophylaxis for candidal vaginitis. ANN Intern Med 1992; 116: 353.
16. Holmes KK. Non specific vaginosis. Scand J Infect Dis 1985.