Clinico-Microbiological Study of Symptomatic Vaginal Discharge

Sneha Kukanur* and Ashish Bajaj

1Department of Microbiology, PES Institute of Medical Sciences and Research, Kuppam, AP, India
2Department of Microbiology, ESI Medical College, Faridabad, Haryana, India
*Corresponding author

A B S T R A C T

Abnormal vaginal discharge is the most common gynaecological symptom in reproductive age group (15-45 years). It’s prevalence in India is 30%. Aetiological agent of vaginitis can be bacterial, fungal or parasitic. It causes significant morbidity like pelvic inflammatory disease (PID), infertility, pregnancy loss, preterm labour, increased risk of other STDs to mention a few. Hence, the present study was undertaken to unravel the specific etiological agents and their sensitivity pattern. Totally, 200 high vaginal swabs from clinically suspected vaginitis cases were subjected to saline-wet mount, pH-estimation, Gram stain, Methyl-violet stain, KOH-mount and whiff/amine test. Samples were cultured aerobically. Organisms were identified with their antibiotic sensitivity pattern. Most common cause was Aerobic-vaginitis (32%). Commonest organisms were E.coli, P.aeruginosa and S.aureus. Many were sensitive to amikacin except for P.aeruginosa which were sensitive to imipenem and colistin. Vulvovaginal-candidiasis, Bacterial-vaginosis and Trichomoniasis were seen in 14.5%, 14% and 6.5% of our cases respectively. All vaginal discharge cases should be considered for rapid microbiological examination like Saline-wet mount, Gram stain, KOH-mountain and Whiff test. The diagnosis should be confirmed by identification and it’s susceptibility pattern for specific treatment to avoid above complications.

Introduction

Symptomatic vaginal discharge due to vaginitis is the frequent complaint encountered every other day both by gynaecologists and general practitioners. 1-14% of women in the reproductive age group suffer from vaginitis and it accounts for 5-10 million OPD visits per year all over the world. In India, among the females of reproductive age group its prevalence is estimated to be 30% (Rekha et al., 2010).

Vaginitis is a diagnosis based on the presence of symptoms of abnormal discharge, vulvovaginal discomfort or both. Small amount of discharge flows from the vagina daily as the body’s way of maintaining a normal healthy environment. Normal discharge is usually clear with no malodour. Change in amount, colour, odour, irritation, itching or burning could be due to an imbalance of healthy bacteria in the vagina, leading to vaginitis (Gor et al., 2013).
Few morbid conditions like PID, infertility, endometriosis, cuff-cellulitis, urethral syndrome, pregnancy loss, preterm labor can be predisposed by abnormal vaginal discharge (Rekha et al., 2010).

Results from many different studies performed in order to establish the frequency of the infectious agents for vaginitis have varied widely. The ranges found for Bacterial-vaginosis have varied between 8% & 75%; Aerobic-vaginitis between 7.9% (Donders et al., 2002) & 51% (Jahic et al., 2013); Vulvovaginal-Candidiasis has presented rates between 2.2% & 30% & Trichomoniasis between zero & 34%(Adad et al., 2001); yet 7-72% of women with vaginitis may remain undiagnosed. Precise diagnosis may be difficult to identify, but care must be taken to differentiate these conditions from other infectious and non-infectious causes (Gor et al., 2013).

Successful management of symptomatic vaginal discharge lies in the diagnostic approach. In most situations, a presumptive diagnosis is made based on nature of discharge (clinical diagnosis), which is often incomplete. Thus, elimination of laboratory component (microbiological diagnosis) has led to treatment mismanagement, giving rise to over or under treatment, increase in recurrence rates, and increase in resistant strains of the etiological agents as well. Conventional approach for the diagnosis is through microbiological diagnosis of the aetiological agent(s). This is vitally necessary for proper management of the condition (Rekha et al., 2010).

Hence, the present study was conducted to identify specific aetiological agents causing vaginitis, by simple & rapid methods which further leads to appropriate treatment of the condition.

Materials and Methods

It’s a prospective study, where high vaginal swabs from 200 clinically suspected vaginitis cases were processed for the identification of causative organism(s) over a period of one year, attending the Gynaecology clinic of rural tertiary care hospital, South India, Karnataka.

Institutional ethical committee clearance was taken. Samples were collected after obtaining written informed consent from the patients.

Inclusion Criteria: Patients in reproductive age group with symptomatic vaginal discharge.

Exclusion Criteria: Patients in whom per speculum and pelvic examination was not possible, who were menstruating, who were on antimicrobials/antifungals(topical/oral), pregnant women, postmenopausal patients, post hysterectomy patients and who were in post-partum period.

A complete medical history was taken which included age, address, presenting complaints(type of discharge, odour, associated pain etc.), history of present illness, predisposing factors, previous history of treatment, past history, obstetric history and occupational history.

The vaginal discharge was collected from the posterior fornix and lateral vaginal wall with cotton tipped sterile swabs. These specimens were further subjected to various tests for identification of the organisms causing vaginitis (Collee et al., 2007). Oedema, inflammation of vaginal wall, vaginal ulcerations and nature of the vaginal discharge (i.e. colour, odour, consistency) were observed (Money et al., 2005).
The various tests done were, pH estimation using pH (Qualigens) paper, saline-wet mount, KOH preparation(Amine/Whiff test & KOH mount). Gram stain & Methyl violet stain was done. Sample cultured on Blood Agar, MacConkey’s Agar and Sabouraud dextrose agar for aerobic bacterial and fungal cultures (El-Din et al., Khamees et al., 2012) Aerobic bacteria grown were identified by conventional methods (Collee et al., 2007).

Antibiotic sensitivity of aerobic bacterial isolates was performed on Mueller Hinton agar(MHA) plates by standardized Kirby Bauer disc diffusion technique as per the CLSI guidelines (CLSI, 2013).

**Statistical Analysis:** Pearson’s Chi square test and Fischers’ Exact test was used.

**Results and Discussion**

All our patients were from Out Patient Department of Gynaecology. Study group included women in the reproductive age group.

Maximum number of cases fell in the age group of 36-40years (55, 27.5%), followed by 31-35years (49, 24.5%), 26-30years (47, 23.5%), 21-25years (32, 16%) and 41-45years (14, 7%) The least number of cases were detected in the age group of 15-20years (3, 1.5%). Out of 200 cases, 197 were married.

Most of our cases 127(63.5%) had 2 children, followed by 1 child in 44(22%), 3 children in 20(10%), no children in 8(4%) and 5 children in 1(0.5%) case.

Among 200 cases studied most of them were housewives 74(37%), followed by agriculturists 54(27%), construction workers 45(22.5%), house maids 13(11.5%) and office workers 4(2%) by occupation.

Most common method of contraception practiced among the study group was tubal sterilization 63(31.5%), followed by Intra-uterine Contraceptive devices 52(26%), Oral contraceptive pills 46(23%) and barrier contraception 26(13%).

All our cases presented with vaginal discharge, 44(22%) of them presented with pruritus, 43(21.5%) pain in lower abdomen and 30(15%) foul smelling vaginal discharge.

68(34%) of them gave the history of recurrence, 19(9.5%) history of abortion and 15(7.5%) history of Diabetes mellitus.

pH strip examination of vaginal discharge showed 101(50.5%) cases with pH less than 4.5 and 99(49.5%) of them more than 4.5.

Saline wet mount showed fungal elements and motile trichomonads in 29(14.5%) and 13(6.5%) cases respectively.

KOH-mount showed fungal elements in 29(14.5%) cases where as Whiff test/ amine test was positive in 28(14%) cases.

Methyl-violet staining done to demonstrate Trichomonads showed positive results in 13(6.5%) cases.

Only 2 strains of *P.mirabilis* were isolated which were uniformly sensitive to almost all antibiotics tested.

Present study consists of females in reproductive age group. Mean age in our study was 32.49 years which is similar to previous studies (Cook RL et al., 1992; Jahic M et al., 2013; Thulkar J et al., 2010; Hemalatha et al., 2013; Jahic M et al., 2013)

The present study revealed that 101 cases were with median parity 2, of which around 50% had vaginitis. Similarly (Hemalatha et
al., 2013) has also revealed maximum cases with maternal parity 2.

37% of our cases were housewives, with primary education, around 61% were illiterate and only around 2% were literate and were office workers. This is comparable to the work done by (Rahman et al., 2013) where they also observed 60% illiteracy among the study group. As ours is a rural hospital, majority of them were illiterates. It is lack of education which makes them ignorant about the facilities available in hospital. Due to their busy schedule they fail to approach health care system on time resulting in complications. Thulkar et al., 2010 observed around 38.8% of tubal sterilization which is almost comparable to our study that is around 32%.

Once women are sterilized, they stop using barrier contraceptives and are prone to various STDs. Unhygienic factors may also be possible explanation for the increase in vaginal infection rates. Counselling about good hygiene and proper menstrual care may help in prevention of recurrence. In India, National AIDS Control Program launched in 1987, started promoting condom for prevention of AIDS and other STDs. But still condom use is more for prevention of pregnancy rather than prevention of STDs and recurrent vaginitis (Thulkar et al., 2010).

Table-9 shows occurrence predisposing factors like DM, abortion and recurrences in the study groups. DM can be considered as one of the predisposing factors which varied between 7 and 30%, whereas history of abortion and history of recurrence can be considered as complications of persistent vaginitis which varied between 9 to 60% and 5 to 50% respectively, and our results are within agreeable limit. The p value for all 3 parameters are <0.01, <0.001 and <0.01 which are highly significant.

Assessment of intra-vaginal pH is of great help, but often neglected procedure that can be used to evaluate vaginal health (Hemalatha et al., 2013). Vaginal pH plays a critical role in maintaining normal flora, hence even a slightest variation will reflect in the change of flora which finally results in various types of infections. We have observed 50% of pH less than 4.5 and rest more than 4.5 which is well correlated with other studies (Aggarwal et al., 2003; Rekha et al., 2010; Fule et al., 2012; Verma et al., 2013; Jahic et al., 2013; Hemalatha et al., 2013).

Saline wet mount is the most cost-effective diagnostic test (Garber et al., 2005) We observed 14.5% and 6.5% of fungal elements and motile Trichomonads respectively. Other studies observed Trichomonads in around 10 to 20% of the cases (Clay et al., 1998; Rekha et al., 2010; Fule et al., 2012; Rahman et al., 2013) In a study done by Rahman et al., 2013 they reported around 10% of fungal elements in the wet mount.

In 14.5% of our cases KOH mount was positive for fungal elements which was almost similar to the study done by Samia S Khamees et al., 2012.

Positive amine test was observed in 14% in our study, whereas it varied from 7 to 57% in various studies (Donders et al., 2002; Fule et al., 2012; Khamees et al., 2012; Rahman et al., 2013; Hemalatha et al., 2013; Jahic et al., 2013) Whiff test seems less practical and requires a good sense of smell. However, inclusion of whiff test along with pH value may improve the specificity of provisional diagnosis (Hemalatha et al., 2013). Presence of clue cells is very characteristic of BV which was observed in 14% of our cases (p value – <0.001, highly significant) whereas it was observed in
58.9% of cases in a study done by Hemalatha et al., Hemalatha et al., have quoted 95% sensitivity and 90% specificity for clue cells in the diagnosis whereas our study showed 100% specificity and sensitivity. Presence of clue cells was correlating with Nugent score and amine/whiff test results.

Analysing the Nugent score which is useful for rapid diagnosis of BV(Money et al., 2005), our study was comparable to other studies except in the range of 4-6. We have observed 40.5% cases in 0-3 score, 32% cases in 4-6 score and 14% cases in 7-10 score. Whereas other studies showed 56% in 0-3 score, 5 to 20% in 4-6 score and 14 to 24% cases in 7-10 score (Modak et al., 2011; Mohanty et al., 2010).

An attempt was made to observe Trichomonas under Gram stain and Methyl-violet stain (Fowler et al., 1952). We observed aerobic vaginitis in 50.5% of the cases, but its frequency varied between 28 to 90% in other studies (Khan et al., 2004; Khamees et al., 2012; Jahic et al., 2013) We observed 14.5% of culture positive VVC while in other studies there is an uniform occurrence of Candidal infection from about 11 to 35% (Khan et al., 2004, El-Din et al., 2009; Khamees et al., 2012; Jahic et al., 2013) Our results are well within this range.

Although a positive yeast culture may represent a woman who is asymptotically colonized, addition of vulvovaginal symptoms yields the correct diagnosis in approximately 90% of women (Nyirjesy et al., 2008).

In the present study, we observed 67% of pathogens, 32% of AV, 14.5% of VVC, 14% of BV and 6.5% of TV. 18.5%(37 cases) of CONS (non-pathogens) which are often considered as commensals (Lakshmi et al., 2012).

Table-10 shows various organisms isolated which include pathogens and commensals. We observed the occurrence of E.coli in 18.6%; it appears to be highest among all studies published varying between 13 and 21%. The role of E.coli in vaginitis is very controversial and is one of the main causes of neonatal sepsis and chorioamnionitis (Jahic et al., 2013).

The presence of K.pneumoniae in 9.3% of our cases of vaginitis can be attributed to taking of antibiotics by infected women (beta-lactam antibiotics), while Klebsiella isolates are considered the most common resistant bacteria to most antibiotics by producing extended spectrum beta lactamase. It can also be attributed to absence or decrease in numbers of lactobacilli due to over the counter drug usage and subsequently their defence factors (Razzak et al., 2011).

We observed around 12% of P.aeruginosa, its incidence varied from 2-9% and ours is on the higher side.

Among Gram positive pathogen, S.aureus is also the common pathogen encountered (10.2% cases). It has been reported by various workers from 2-46% of cases with AV. S.aureus is one of the most persistent pathogens of humans (Mumtaz et al., 2008).

Out of 11 S.aureus isolates 6 were MRSA tested by Cephoxitin disk test.

Our antibiotic sensitivity pattern is in agreement with the other study. With regards to notorious P.aeruginosa, all our 13 strains have slightly lower percentage of sensitivity as compared to other study (Mumtaz et al., 2008).

In the present study 75% of Acinetobacter spp. were sensitive to commonly used antibiotics. Among 2 P.mirabilis, both were
sensitive to commonly used antibiotics except one was resistant to Cotrimoxazole. Antibiotic sensitivity pattern for *Acinetobacter spp.* and *P. mirabilis* cannot be commented upon due to less number of isolates.

**Table 1** Distribution of Nugent score among cases studied.

| Score | No. of cases |
|-------|--------------|
| 0-3   | 81 (40.5%)   |
| 4-6   | 64 (32%)     |
| 7-10  | 28 (14%)     |
| Not applicable | 27 (13.5%) |

**Table 2** Organisms isolated

| Organism isolated          | Single | Mixed | Total |
|----------------------------|--------|-------|-------|
| *E. coli*                  | 13     | 7     | 20    |
| *P. aeruginosa*            | 9      | 4     | 13    |
| *S. aureus*                | 9      | 2     | 11    |
| *Enterococcus spp.*        | 6      | 4     | 10    |
| *K. pneumonia*             | 8      | 2     | 10    |
| *Acinetobacter spp.*       | 4      | -     | 4     |
| *P. mirabilis*             | 2      | -     | 2     |
| *Non albicans candida*     | 14     | 2     | 16    |
| *C. albicans*              | 10     | 3     | 13    |
| Coagulase Negative Staphylococci(CONS) | 37 | - | 37 |

**Table 3** Distribution of Mixed isolates.

| Organisms                          | No. of cases(18) |
|------------------------------------|------------------|
| *T. vaginalis & Enterococcus spp.* | 3                |
| *T. vaginalis & P. aeruginosa*     | 2                |
| *T. vaginalis & Bacterial vaginosis* | 1               |
| *T. vaginalis & C. albicans*       | 1                |
| *T. vaginalis & S. aureus*         | 1                |
| Bacterial-vaginosis & *E. coli*    | 3                |
| *C. albicans & E. coli*            | 1                |
| *C. albicans & P. aeruginosa*      | 1                |
| Non albicans Candida & *K. pneumoniae* | 1              |
| Non albicans Candida & *S. aureus* | 1                |
| *E. coli & P. aeruginosa*          | 1                |
| *E. coli & K. pneumonia*           | 1                |
| *E. coli & Enterococcus spp.*      | 1                |
| Total                               | 18               |
Table 4. Distribution - types of vaginal infection.

| Diagnosis                  | No. of cases |
|----------------------------|--------------|
| Aerobic vaginitis (AV)     | 51 (25.5%)   |
| Bacterial vaginosis (BV)   | 24 (12%)     |
| Vulvovaginal candidiasis (VVC) | 24 (12%) |
| Trichomoniasis (TV)        | 5 (2.5%)     |
| Mixed (AV & TV)            | 6 (3%)       |
| Mixed (AV & VVC)           | 4 (2%)       |
| Mixed (AV & AV)            | 3 (1.5%)     |
| Mixed (AV & BV)            | 3 (1.5%)     |
| Mixed (TV & BV)            | 1 (0.5%)     |
| Mixed (TV & VVC)           | 1 (0.5%)     |
| Normal                     | 78 (39%)     |

Table 5. Antibiotic sensitivity pattern of Gram positive cocci

| Antibiotics       | S.aureus (11) | CONS (37)       | Enterococcus spp (10) |
|-------------------|---------------|-----------------|-----------------------|
|                   | Sensitive     | Resistant       | Sensitive             | Resistant             | Sensitive | Resistant |
| Ampicillin (AMP)  | 4 (36.3%)     | 7 (63.6%)       | 21 (56.7%)            | 16 (43.2%)            | 8 (80%)   | 2 (20%)   |
| Cefoxitin (CX)    | 5 (45.4%)     | 6 (54.5%)       | 26 (70.2%)            | 11 (29.7%)            | -         | -         |
| Cefalexin (CN)    | 5 (45.4%)     | 6 (54.5%)       | 26 (70.2%)            | 11 (29.7%)            | -         | -         |
| Erythromycin (E)  | 8 (72.7%)     | 3 (27.2%)       | 29 (78.3%)            | 8 (21.6%)             | 5 (50%)   | 5 (50%)   |
| Clindamycin (CD)  | 8 (72.7%)     | 3 (27.2%)       | 28 (75.6%)            | 9 (24.3%)             | -         | -         |
| Gentamicin (GEN)  | 9 (81.8%)     | 2 (18.1%)       | 26 (70.2%)            | 11 (29.7%)            | -         | -         |
| Ciprofloxacin (CIP)| 8 (72.7%)  | 3 (27.2%)       | 24 (64.8%)            | 13 (35.1%)            | -         | -         |
| Amikacin (AK)     | 11 (100%)     | 0 (0%)          | 27 (72.9%)            | 10 (27.0%)            | 5 (50%)   | 5 (50%)   |
| Ceftriaxone (CTR) | -             | -               | -                     | 8 (80%)               | 2 (20%)   |           |
| Gatifloxacin (GAT)| -             | -               | -                     | 8 (80%)               | 2 (20%)   |           |
| Azithromycin (AZM)| -             | -               | -                     | 9 (90%)               | 1 (10%)   |           |
| High Level Gentamicin (HLG) | - | - | - | 10 (100%) | 0 (0%) |

Table 6. Antibiotic sensitivity pattern of Gram negative bacilli

| Antibiotics       | Acinetobacter spp (4) | E.coli (20) | K.pneumoniae (10) |
|-------------------|------------------------|-------------|-------------------|
|                   | Sensitive | Resistant | Sensitive | Resistant | Sensitive | Resistant |
| Amoxyclav (AMC)   | 1 (25%)  | 3 (75%)  | 10 (50%)  | 10 (50%)  | 5 (50%)   | 5 (50%)   |
| Gentamicin (GEN)  | 3 (75%)  | 1 (25%)  | 10 (50%)  | 10 (50%)  | 8 (80%)   | 2 (20%)   |
| Amikacin (AK)     | 3 (75%)  | 1 (25%)  | 18 (90%)  | 2 (10%)   | 10 (100%) | 0 (0%)    |
| Ceftriaxone (CTR) | 3 (75%)  | 1 (25%)  | 10 (50%)  | 10 (50%)  | 4 (40%)   | 6 (60%)   |
| Cotrimoxazole (COT)| 3 (75%) | 1 (25%)  | 4 (20%)   | 16 (80%)  | 8 (80%)   | 2 (20%)   |
| Sparfloxacin (SPX)| 2 (50%)  | 2 (50%)  | 5 (25%)   | 15 (75%)  | 7 (70%)   | 3 (30%)   |
| Cefotaxime (CTX)  | 3 (75%)  | 1 (25%)  | 9 (45%)   | 11 (55%)  | 5 (50%)   | 5 (50%)   |
Table 7 Antibiotic sensitivity pattern of *P. aeruginosa* (n=13).

| Antibiotics                  | Sensitive | Resistant |
|------------------------------|-----------|-----------|
| Amoxyclav(AMC)               | 7(53.8%)  | 6(46.1%)  |
| Gentamicin(GEN)              | 4(30.7%)  | 9(69.2%)  |
| Ciprofloxacin(CIP)           | 9(69.2%)  | 4(30.7%)  |
| Amikacin(AK)                 | 6(46.1%)  | 7(53.8%)  |
| Ceftriaxone(CTR)             | 4(30.7%)  | 9(69.2%)  |
| Cotrimoxazole(COT)           | 6(46.1%)  | 7(53.8%)  |
| Sparfloxacin(SPX)            | 6(46.1%)  | 7(53.8%)  |
| Cefoperazone/Subbactam(CFS)  | 7(53.8%)  | 6(46.1%)  |
| Piperacillin/Tazobactam(PIT) | 9(69.2%)  | 4(30.7%)  |
| Ceftazidime(CAZ)             | 7(53.8%)  | 6(46.1%)  |
| Meropenem(MRP)               | 4(30.7%)  | 9(69.2%)  |
| Ceftiraxone/Tazobactam(CIT)  | 6(46.1%)  | 7(53.8%)  |
| Tobramycin(TOB)              | 3(23.0%)  | 10(76.9%) |
| Imipenem(IMP)                | 12(92.3%) | 1(7.69%)  |
| Colistin(CL)                 | 13(100%)  | 0(0%)     |

Table 8 Comparison of various clinical features.

| Studies                              | Clinical features          |
|--------------------------------------|---------------------------|
|                                      | Pain abdomen | Pruritus | Foul odour |
| Gilbert G G Donders et al., 2002     | 6.2%         | 20%      | 8%         |
| A Aggarwal et al., 2003              | -            | -        | 52.5%      |
| S Rekha et al., 2010                 | -            | 20%      | -          |
| Varsha Chaudhary et al., 2012        | 54.3%        | 25%      | -          |
| Rita Elizabeth Moreira Mascarenhas et al., 2012 | 9.1%      | 19.1%   | -          |
| Aruna Verma et al., 2013             | 73%          | -        | 17%        |
| R Hemalatha et al., 2004             | -            | -        | 43.5%      |
| Mahira Jahic et al., 2013            | -            | 72.54%   | 29.94%     |
| Present study                        | 21.5%        | 22%      | 15%        |

Table 9 Comparison of predisposing factors in different studies.

| Studies                              | H/o Diabetes mellitus | H/o Abortion | H/o Recurrence |
|--------------------------------------|-----------------------|--------------|----------------|
| Marcia Edilaine Lopes Consolaro et al., 2004 | -                     | -            | 5.6%           |
| Al-Zahraa Karam El Din et al., 2009  | 28.9%                 | -            | -              |
| S Rekha et al., 2010                 | -                     | -            | 42%            |
| Varsha Chaudhary et al., 2012        | -                     | 19.2%        | -              |
| Rahman D et al., 2013                | -                     | 52%          | -              |
| Deepa Babin et al., 2013             | 8.6%                  | -            | -              |
| Aruna Verma et al., 2013             | -                     | 64.2%        | -              |
| Present study                        | 7.5%                  | 9.5%         | 34%            |
Table 10 Comparison of pathogens causing Aerobic vaginitis in various studies.

| Studies          | Khan et al., 2004 | Muntaz et al., 2008 | Razzaq et al., 2011 | Lakshmi et al., 2012 | Khames et al., 2012 | Present study |
|------------------|-------------------|---------------------|---------------------|----------------------|---------------------|---------------|
| E. coli          | 21%               | 13.67%              | 16.2%               | 15.2%                | 13.83%              | 18.6%         |
| P. aeruginosa    | 2%                | 7.25%               | 8.1%                | -                    | 9.57%               | 12.1%         |
| S. aureus        | 2%                | 46.1%               | 18.9%               | 8.7%                 | 21.28%              | 10.2%         |
| Enterococcus spp.| 31%               | 9%                  | -                   | 6.5%                 | 11.7%               | 9.3%          |
| K. pneumoniae    | 3%                | 10.5%               | 8.1%                | 0%                   | 13.48%              | 9.3%          |
| Acinetobacter spp.| -                | 1.36%               | 6.8%                | -                    | 1.06%               | 3.7%          |
| P. mirabilis     | -                 | 1.36%               | -                   | -                    | 5.32%               | 1.8%          |
| CONS             | 7%                | -                   | -                   | 21.7%                | -                   | 34.5%         |

Table 11 Comparison of Antibiotic sensitivity pattern of GPC

| Antibiotics | S. aureus | Enterococcus spp. |
|-------------|-----------|-------------------|
|             | Muntaz et al., 2008 | Present study | Muntaz et al., 2008 | Present study |
| Ampicillin  | 26.3%     | 36.3%             | 63.8%               | 80%            |
| Ciprofloxacin| 65.51%    | 72.7%             | -                   | -              |
| Gentamicin  | 67.6%     | 81.8%             | 43.5%               | 100%           |
| Amikacin    | 76.9%     | 100%              | -                   | -              |
| Methicillin/ Cephoxtin | 69.3% | 54.5%             | -                   | -              |

Table 12 Comparison of Antibiotic sensitivity pattern of GNB

| Antibiotics | E. coli | K. pneumoniae |
|-------------|---------|---------------|
|             | Muntaz et al., 2008 | Present study | Muntaz et al., 2008 | Present study |
| Amoxycalv   | 46.8%   | 50%           | 60.29%              | 50%            |
| Cephotaxime | 73.9%   | 45%           | 79.1%               | 50%            |
| Gentamicin  | 65.4%   | 50%           | 62.8%               | 80%            |
| Amikacin    | 81.3%   | 90%           | -                   | 100%           |
| Cotrimoxazole| 21.5%  | 20%           | 61.8%               | 80%            |

Summing up all the AV cases aerobic bacterial pathogens were isolated from 32% of the cases, but in various studies it ranged between 24 and 51% and our study is within the acceptable limits (Rekha et al., 2010; Jahic et al., 2013).

A total of 29 Candida species isolated in our study, of which 13(45%) were C. albicans and 16(55%) were Non albicans Candida. Other studies revealed 35.5 to 54.9% of C. albicans and 45.1 to 64.4% of Non albicans Candida spp. There is a gradual increasing trend of Non albicans Candida VVC. It is important to emphasize that in the past 3 decades there has been an increasing percentage of infections caused by non-albicans Candida spp., particularly C. tropicalis, C. glabrata and C. krusei and are resistant to conventional therapy (Babin et al., 2013; El-Din et al., 2009).
Summarizing the various pathogens observed in studies over 10 years, BV has varied from 6.6% to 75%, (Verma et al., 2013; Mohanty et al., 2010; Rekha et al., 2010; Modak et al., 2011; Khamees et al., 2012; Rahman et al., 2013; Jahic et al., 2013; Hemalatha et al., 2013) ours is around 14%. VVC is ranging between 4.8% and 39.5%, (Verma et al., 2013; Rekha et al., 2010; Khamees et al., 2012; Lakshmi et al., 2012; Rahman et al., 2013; Jahic et al., 2013) ours is 14.5% which is also well within the stated range. There is a uniform occurrence of TV from about 2% -10% in almost all the studies, (Verma et al., 2013; Rekha et al., 2010; Khamees et al., 2012; Lakshmi et al., 2012; Jahic et al., 2013) ours is around 6.5% which a significant number. With respect to AV, only 2 studies have quoted which varied from 24 to 51% (Rekha et al., 2010; Jahic et al., 2013) and ours is 32% which is acceptable.

Hence, for the confirmation of all provisional diagnosis, microbiological assistance is necessary. A proper diagnosis will lead to proper management and avoid complications like recurrence, resistance, abortion, PID, cuff cellulitis, chorioamnionitis, PROM etc. It is mandatory to diagnose all the cases of vaginal discharge with laboratory techniques and Antibiotic sensitivity testing.

**Acknowledgement**

We would like to acknowledge Dr Meera Meundi for her kind support and continuous encouragement.

**References**

Adad, S.J., Lima, R.V., Sawan, Z.T.E., Silva, M.L.G., Souza, M.A.H., Saldanha, J.C. et al. 2001. Frequency of *Trichomonas vaginalis*, *Candida sp* and *Gardenerella vaginalis* in cervical – vaginal smears in four different decades. *Sao Paulo Med. J. Rev. Paul Med.*, 119(6): 200-5.

Aggarwal, A., Devi, P., Jain, R. 2003. Anaerobes in Bacterial vaginosis. *Indian J. Med. Microbiol.*, 21(2): 124-6.

Babin, D., Kotigadde, S., Rao, P.S., Rao, T.V. 2013. Clinico- mycological profile of vaginal candidiasis in a tertiary care hospital in Kerala. 2013; *Int. J. Res. Biol. Sci.*, 3(1): 55-9.

Chaudhary, V., Kumar, R., Agarwal, V.K., Singh, A., Narula, R., Sharma, M. 2012. Prevalence and determination of Vaginal discharge among women of reproductive age group in tertiary care hospital of Northern India. *National J. Community Med.*, 3(4): 661-5.

Clay, J.C., Veeravahu, M., Smyth, R.W. 1998. Practical problems of diagnosing trichomoniases in women. *Genitourin Med.*, 64: 115-7.

CLSI. Clinical and Laboratory Standards Institute. 2013. Performance standards for antimicrobial susceptibility testing. Twenty-third informational supplement. Wayne, PA, USA: CLSI: M100-S23.

Collee, J.G., Duguid, J.P., Fraser, A.G., Marmion, B.P., Simmons, A. 2007. Laboratory strategies in the diagnosis of infective syndromes. In: Mackie and McCartney, Practical Medical Microbiology. Collee JG, Fraser AG, Marmion BP, Simmons A, editors. Chapter 4. 14th ed. Churchill Livingstone Inc; London: 53-94.

Collee, J.G., Duguid, J.P., Fraser, A.G., Marmion, B.P., Simmons, A. 2007. Tests for the identification of bacteria. In: Mackie and McCartney, Practical Medical Microbiology. Collee JG, Fraser AG, Marmion BP, Simmons A, editors. Chapter 7. 14th ed. Churchill Livingstone Inc; London:131-150.
Consolaro, M.E.L., Albertoni, T.A., Yoshida, C.S., Mazucheli, J., Peralta, R.M., Svidzinski, T.I.E. 2004. Correlation of *Candida* species and symptoms among patients with Vulvovaginal candidiasis in Maringa, Parana, Brazil. *Rev. Iberoam Micol.*, 21: 202-5.

Cook, R.L., Lopez, V.R., Schmit, C., Meriwether, C., Sobel, J.D. 1992. Clinical, Microbiological, and Biochemical Factors in Recurrent Bacterial Vaginosis. *J. Clin. Microbiol.*, 30(4): 870-7.

Donders, G.G.G., Vereecken, A., Bosmans, E., Dekeersmaecker, A., Salembier, G., Spitz, B. 2002. Definition of a type of abnormal vaginal flora that is distinct from bacterial vaginosis: aerobic vaginitis. *Br. J. Obstet. Gynaecol.*, 109: 34-43.

El-Din, A.K., Habib, F., Abd-Allah, N., Khorsheid, O. 2009. Mycotic VulvoVaginitis: Epidemiology, Pathogenesis and Profile of Antifungal Agents. *J. Taibah Univ. Med. Sci.*, 4(2): 123-36.

Fowler, W. 1952. Simple staining method for *Trichomonas vaginalis*. British J. Venereal Dis., 28: 144: 166-7.

Fule, S.R., Fule, R.P., Tankhiwale, N.S. 2012. Clinical and laboratory evidence of *Trichomonas vaginalis* infection among women of reproductive age in rural area. *Indian J. Med. Microbiol.*, 30(3): 314-6.

Garber, G.E. 2005. The laboratory diagnosis of *Trichomonas vaginalis*. *Can. J. Infect. Dis. Med. Microbiol.*, 16(1): 35-8.

Gor, H.B. Vaginitis [Internet].2013 [Updated: May 7, 2013].Available from: http://emedicine.medscape.com/article/257141-overview#a0104.

Hemalatha, R., Ramalaxmi, B.A., Swetha, E., Balakrishna, N., Mastromarino, P. 2013. Evaluation of vaginal pH for detection of bacterial vaginosis. *Indian J. Med. Res.*, 138: 354-9.

Jahic, M., Mulavdic, M., Hadzimehmedovic, A., Jahic, E. 2013. Association Between Aerobic Vaginitis, Bacterial Vaginosis and Squamous Intraepithelial Lesion of Low Grade. *Med. Arch.*, 67(2): 94-6.

Jahic, M., Mulavdic, M., Nurkic, J., Jahic, E., N. Midhat. 2013. Clinical Characteristics of Aerobic Vaginitis and its Association to Vaginal Candidiasis, Trichomonas vaginitis and Bacterial vaginosis. *Med. Arch.*, (6): 428-30.

Khamees, S.S. 2012. Characterization of vaginal discharge among women complaining of genital tract infection. *Int. J. Pharm & Sci.*, 3(10): 1997-2002.

Khan, I., Khan, U.A. 2004. A hospital based study of frequency of aerobic pathogens invaginal infections. *J. Rawal Med. Coll.*, 29(1): 22-5.

Lashmi, K., Chitralekha, S., Illamani, V., Menezes, G.A. 2012. Prevalence of Bacterial vaginosis infections in pre and postmenopausal women. *Int. J. Pharm. Bio Sci.*, 3(4): 949-56.

Mascarenhas, R.E.M., Machado, M.S.C., Silva, B.F.B.C., Pimentel, R.F.W., Ferreira, T.T., Leoni, F.M.S. *et al.* 2012. Prevalence and Risk Factors for Bacterial Vaginosis and Other Vulvovaginitis in a Population of Sexually Active Adolescents from Salvador, Bahia, Brazil. *Infect. Dis. Obstetrics and Gynecol.*, 378640-5.

Modak, T., Arora, P., Agnes, C., Ray, R., Goswami, S., Ghosh, P., Das, N.K. 2011. Diagnosis of bacterial vaginosis in cases of abnormal vaginal discharge: comparison of clinical and microbiological criteria. *J. Infect. Dev. Ctries.*, 5(5): 353-60.
Mohanty, S., Sood, S., Kapil, A., Mittal, S. 2010. Interobserver variation in the interpretation of Nugent scoring method for diagnosis of bacterial vaginosis. Indian J. Med. Res., 131: 88-91.

Money, D. 2005. The laboratory diagnosis of bacterial vaginosis. Can. J. Infect. Dis. Med. Microbiol., 16(2): 77-79.

Mumtaz, S., Ahmed, M., Aftab, I., Akhtar, N., UlHassan, M., Hamid, A. 2008. Aerobic vaginal pathogens and their sensitivity pattern. J. Ayub. Med. Coll. Abbottabad, 20(1): 113-7.

Nyirjesy, P. 2008. Vulvovaginal Candidiasis and Bacterial Vaginosis. Infect. Dis. Clin. N. Am., 22: 637-52.

Rahaman, D., Adhikary, A., Hussein, S. 2013. Aetiologies of Vaginal Discharge among Women Presented with Cervical Abnormalities: Experiences at a Tertiary Care Hospital. J. Shaheed Suhrawardy Med. Coll., 5(1): 31-4.

Razzak, M.S.A., Al-Charrakh, A.H., AL-Greitty, B.H. 2011. Relationship between lactobacilli and opportunistic bacterial pathogens associated with vaginitis. North Am. J. Med. Sci., 3: 185-192.

Rekha, S., Jyothi, S. 2010. Comparison of visual, clinical and microbiological diagnosis of symptomatic vaginal discharge in the reproductive age group. Int. J. Pharm. Biomed. Res., 1(4): 144-8.

Thulkar, J., Kriplani, A., Agarwal, N., Vishnubhatla, S. 2010. Aetiology & risk factors of recurrent vaginitis & its association with various contraception methods. Indian J. Med. Res., 131: 83-7.

Verma, A., Gupta, A., Goel, S., Garg, A. 2013. Clinicopathological correlation of infective vaginal discharges in non-pregnant sexually active women of reproductive age group in a tertiary care centre of Western UP. Int. J. Reprod. Contracept. Obstet. Gynecol., 2(3): 349-54.

How to cite this article:

Sneha Kukanur and Ashish Bajaj. 2016. Clinico-Microbiological Study of Symptomatic Vaginal Discharge. Int.J.Curr.Microbiol.App.Sci. 5(8): 293-304. doi: http://dx.doi.org/10.20546/ijcmas.2016.508.031