

# THE CHALLENGES TO HOMEOSTASIS IN THE VAGINAL ECOSYSTEM

The essence of healthy existence for every living being is homeostasis within itself and the surrounding environment. Sadly, the human being, despite being at the highest echelons of the animal kingdom, is the only living organism who continuously defies this ideology resulting in the calamitous state of affairs we are experiencing both in our environment and in our personal health, both physical and psychological.

Vaginal ailments, even though usually not so devastating, are a clear example of how loss of ecological balance can affect our quality of life. The most common of these is vaginitis, and more specifically, bacterial vaginosis.


Homeostasis in the post-pubertal human vagina is maintained by the colonization of commensal bacteria, mainly hydrogen peroxide-producing lactobacilli<sup>1</sup>. Other bacteria are also present, namely streptococcal species, gram-negative bacteria and other anaerobic species, including *Gardnerella vaginalis*. *Candida albicans* is also present in 10 to 25% of asymptomatic women<sup>2</sup>.

Estrogen increases the production of glycogen in vaginal epithelial cells and its metabolism by the lactobacilli to lactic acid. This renders the vaginal ecosystem mildly acidic (pH 3.8-4.5) and inhibits the adherence of bacteria to vaginal cells; the hydrogen peroxide which is produced by the lactobacilli inhibits the growth of bacteria and has also been found to kill HIV in vitro<sup>2</sup>. This mutualism between lactobacilli and vaginal epithelium, together with innate and acquired immunity, protects the vagina from colonisation by pathogenic bacteria, thus preventing ascending or systemic infection<sup>3</sup>.

The most common organisms causing vaginitis are bacteria, fungi and protozoa. Bacterial vaginosis is not caused by one agent<sup>4</sup>, as is commonly believed but is rather a polymicrobial syndrome usually caused by *Gardnerella vaginalis*, *Mobilincus*, *Bacteroides saprophytes* and *Mycobacterium Hominus*; *Chlamydia trachomatis* and *Neisseria gonorrhoea* are less commonly implicated with bacterial vaginosis. Fungal vaginitis is usually caused by *Candida albicans*. The epidemiology varies according to the geographical location - bacterial vaginosis is the most common vaginal infection followed by vulvo-vaginal candidiasis in the US; in Europe however, the fungal form is the most prevalent<sup>1</sup>.

Bacterial and fungal vaginosis usually occur following the exposure to an external agent that alters the vaginal homeostasis either by altering the pH or by disturbing the vaginal commensal flora. This could include biological factors like pregnancy and menopause or external agents such as vaginal douches, chemotherapy (direct inhibition or cell lysis<sup>5</sup>), sexual intercourse (multiple partners) and contraception (IUD). Trichomoniasis, on the other hand, is a sexually transmitted disease caused by the protozoan *Trichomonas vaginalis*. Vaginosis is easily curable with adequate treatments; however all types can be associated with recurrent relapses and adverse outcomes, including increased risk of HIV or other forms of upper genital infections including pelvic inflammatory disease, premature rupture of membranes or abortion<sup>1,2</sup>.

Despite there being a few lactobacillus species constituting the vaginal flora, there are various species- and strain-specific



differences which account for the wide variability in their ability to maintain the stability of the vaginal ecosystem. Thus, the vaginal immunity is one of the factors - apart from the effect of endocrinal changes on vaginal physiology, and external challenges - in determining both the liability to infection and recurrence, as well as the propensity to respond to treatment<sup>3</sup>.

Diagnosis hinges on the symptomatology described by the patient and the naked eye examination of the vaginal discharge. The clinical findings can be very similar especially in the bacterial and fungal cases which can render diagnosis difficult. Microscopical assessment of the discharge is the mainstay of diagnosis; unfortunately it is usually resorted to on therapeutic failure of the usual repeated courses of empirical therapy; this not only leads to a financial and social burden resulting in non-compliance on the part of the patients but also contributes to the overall emergence of resistance<sup>1</sup>.

The hallmark of the management of vaginitis rests on the triad of prevention, education and antimicrobial treatment, all of which lie in the remit of the general practitioner. Education so far is mostly carried out through face-to-face interventions when the patient turns up for treatment of one or more of the unpleasant symptoms, or for the insertion of a contraceptive device. The success of prevention is directly proportional to how much the educational messages manage to get driven home and how much the patient truly comprehends and appreciates their importance especially considering the embarrassment associated with such disturbances.

Antimicrobial treatment comes mostly in the form of topical agents - creams or pessaries - or oral medication. Creams or pessaries can deliver either one antimicrobial agent or contain a synergistic combination of two or more active ingredients to increase the likelihood of remission and decrease the risk of recurrence.

Unfortunately, recurrence of vaginal infections is common. In most cases this is the result of wrong diagnosis, self-treatment

with over-the-counter medication, poor compliance to treatment by the female, failure to treat the male partner, resistance to treatment and/or non-adherence to the preventive measures advised. The rest of the recurrences occur due to an inherent predisposition of the female, either as a result of a particular physiological state e.g. pregnancy and menopause, the presence of an IUD, insufficient numbers of peroxide-producing vaginal lactobacilli or altered host immune response<sup>3</sup>.

The best way to avoid this state of affairs is to take a structured and systematic approach to the management of vaginitis. A good history from the patient regarding symptomatology, past medical history and sexual/gynae history is an important start. This should be ideally followed by pelvic examination including determination of vaginal pH, visual assessment and microscopic evaluation of the discharge<sup>2</sup>. It is only through correct diagnosis that recurrence of disease and misuse of medication can be avoided. In this day and age, where vaginal and sexually transmitted diseases are exponentially increasing due to our 'boundary-less' way of living, the author strongly believes that prevention, especially through 'out there' education, should be given more importance and validity. The path to achieving a balance is simple... the difficulty lies in choosing it. ❄️

#### REFERENCES

1. Khan Shazia A et al. Evaluation of Common Organisms causing Vaginal Discharge. J Ayub Med Coll Abbottabad 2009; 21(2):90-3.
2. Eckert Linda O. Acute Vulvovaginitis. The New England Journal of Medicine 2006; 1244- 1252.
3. Verstraelen, H. Cutting Edge: the vaginal microflora and bacterial vaginosis. Verh K Acad Geneesk Belg 2008; 70(3):147-74.
4. Hill GB, Eschenbach DA, Holmes KK. Bacteriology of the Vagina. Scand J Urol Nephrol Suppl. 1984;86:23-39.
5. Martin R, Soberon N, Escobedo S, Suarez JE. Bacteriophage induction versus vaginal homeostasis: role of H<sub>2</sub>O<sub>2</sub> in the selection of Lactobacillus defective prophages. Internation Microbiology 2009; 12:131-6.