Gardenerella Vaginalis And Candida Albicans Pathogens Causing Vagonosis/Vaginitis, Possible Causes Of Acute Diffuse Purulent Peritonitis

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Abstract

Although there are reports on infection of pelvic peritonitis due to pathogens causing vaginosis/vaginitis, little has been reported on diffuse purulent peritonitis due to Gardnerella vaginalis, an anaerobic bacteria, one of the major causative pathogen in vaginitis/vaginosis. This is a classic case of vaginitis/vaginosis caused by G. vaginalis and Candida albicans not treated initially, which resulted in diffuse purulent peritonitis, but treatment was almost perfect, with a fast remission of the disease in a 23 years old patient.

INTRODUCTION

A 23 years old female was admitted in the gynaecological department for severe lower abdominal pain, supposed to be due to pelvic peritonitis at 7:30 in the morning hours. Although she gave a history of an earlier epigastric pain two-three days ago, the hypogastric region was more tender at the time of admission. The patient was clinically stable, with a blood pressure of 120/80, Pulse 100, breathing rate 22u/min. She had no fever initially, there was no vomiting too, she had even taken some food by mouth before midday.

History revealed that she had a spontaneous abortion on the 8th week of her first pregnancy, that was about a month earlier before she was admitted. After the spontaneous abortion, she had reported to one of the private clinics in the area where she lived, and received an intravenous fluid only, without further treatments. On examination, the lower abdomen was very tender, but she refused a per vaginam examination, though she allowed the gynecologist to take swab for culture, who however, noticed plenty of thick milky-cheesy like substance with a foul odour discharge from the vagina. She admitted noticing that discharge since after that abortion, but could not say if the problem had been there before pregnancy. Swab of the discharge was then obtained for culture.

The initial diagnosis was then: Status post spontaneous abortion, vaginosis/vaginitis? Pelvic peritonitis. Acute appendicitis?

As time passed by, the abdominal pain became worse, more severe, involving all parts of the abdominal cavity. The consulting surgeon of the hospital was summoned at about 12pm. Indeed, a severe abdominal pain, with the abdomen distended, diffusely tender with rebound tenderness sign and a positive Blumberg's sign, and muscular guarding. There was a weak bowel sound of the intestine, but was still flatus. At this time she had fever noted at 39ºC. Her tongue was coated, the eyes somewhat sunken, the blood pressure was 132/72, pulse rate 110u/min, respiratory rate 26u/min. An order was given to stop food by mouth, an IVF 3000ml, containing (1500ml 5% NDS and Ringer 1500ml) An empirical antibiotic therapy was begun, including Ceftriaxone 2x per 1.0g iv, Metronidazole 3x per 500mg iv, Omeprazole 1x 40mg iv, Pethidine 25mg iv/3-4hrs, Diclofenac 3x per 50mg i.m.

Urinary out put was not collected because the patient refused to accept the installation of a urinary catheter initially. The patient was told about her condition and the need for an emergency surgery due to the possibility of a perforated appendix from inflammation and a consequent acute generalized peritonitis.

The patient was counseled and persuaded to sign a consent for the said operation, but she refused. At 16.15pm. her clinical status was fast deteriorating, but she still refused to

succumb to operation. In fact, there was a lot of confusion caused more so by her family members, arguing that, that was not the case, and that we wanted to lure them into unnecessary cost.(well in this part of the world such arguments exist amongst family members, relatives, friends and patients).

It was after much persuasion from the staff members that the patient agreed to sign for an operation, that was at about 18.30pm. However, before the operation, the patient had wished for a small incision and even opted for a cosmetic one. The surgeon agreed to her demands and promised to do his best so that she would have a “nice scar”

The operation was begun at 19.15pm, under a general anesthesia, with a McBurney's incision, (Gridiron). A laparotomy was performed. It was observed that there was plenty of purulent fluid, cloudy, of an average viscosity about 1.5 to 2 litres, no odour, all the intestines were reddened, hyperemic including the gonads, but the small intestine seemed more inflamed. An appendectomy was performed, though with no signs of perforations, and doubts about the origin of the pus, the incision was extended using the Pfannansteil's modification, and of course bearing in mind the wish or demand of the patient for a “cosmetic incision for a nice scar”.

The uterus and the ovaries were checked, the uterus in particular if there was an infiltration or perforations, which could have been the result of a criminal abortion eventually, but none was found. The organs however were acutely inflamed. There were no perforations of the hollow viscerals but the small intestines were hyperemic, and bleeding easily on manipulation. The fluid from the abdominal cavity was collected in a sterile tube, about 5ml and sent to the laboratory for culture. The pussy fluid was suctioned, and the abdominal cavity was lavaged, irrigated several times with a warm 0,9% saline solution until a clear fluid was obtained. Metronidazole solution, 100ml, 500mg was poured into the abdominal cavity. A drainage tube, size 32 was installed and left in the cavity of Douglas (A closed drainage), and through a separate stab wound. The wound was closed in layers, with peritonisation first, a PDS II nr. 0 was used for fascia suture in a continuous fashion. A sterile packing and wound dressing was accomplished and the appendix sent for a histopathological investigation.

Surgical treatment: Laparotomy, (with the McBurney's then modification of the Pfannansteil's incision). Appendectomy, peritoneal lavage, irrigation, and drainage of the peritoneal cavity.

Diagnosis: Acute diffuse purulent peritonitis. Acute appendicitis. After the operation, on the first post operative day, the patient was clinically stable, in fact with an improvement. She was a febrile, with body temperature noted at 36.2ºCelsius, pulse rate 96u/min, respiratory rate 22u/min, and blood pressure 130/70. The urinary output was normal.

On the second post operative day, there was a bilious vomiting in large amounts, with a distended painful abdomen, for the patient had refused the installation of a naso-gastric tube even though she was persuaded to accept that. Intestinal sounds were heard however. The urinary output was 1850ml/24hrs and 250ml, clear serous drainage fluid was also obtained in the first 24 hrs.

On the third post operative day, the patient was in a stable clinical condition, with no fever, the pulse and blood pressure were normal with the body temperature of 36 ºC., pulse 76u/min, respiratory rate 20u/min, RR 139/87. Her breathing was not laboured, the peristalstic sounds were normal, she thus, passed flatus, the urinary catheter was removed, food was started by mouth, because she had even demanded for food earlier.

Water and sugared tea in sips for hourly dosage of 200ml, tolerance was good and after 6hrs. a soft diet was started with good tolerance. In the evening hours 18pm. IVF was stopped and drainage removed.

On the fourth post operative day, the patient was in a good clinical status, with no fever still, no vomiting, food by mouth was well tolerated, she became friendly and jovial and was discharged on the fifth day post operation. She continued however, the oral antibiotic, Metronidazole 3x per 500mg. After consultation by the gynaecologist, a vaginal Nystatin globules was to be recommended for 3-5days.

On the 14th day post operation period, the patient reported to the surgeon's office for suture removal, even though she was asked for an earlier follow-up date. The wound had healed, without complications, she was smiling and joking, and told the surgeon that she had no more discharge from the vagina and asked when she could make love to her husband, the surgeon replied in 10 days time, but only after her husband has been consulted by the urologist.

LABORATORY RESULTS

a)Laboratory test revealed the following levels of Beta.HCG;

Beta HCG 30.06mlU/ml. in this patient, in comparison with the mean value which is less than 2.0 for non-pregnant women and less than 6.0 on the average. During pregnancy; 4weeks of gestation=40-4480,

5weeks of gestation=270-28700, 6weeks of gestation=3700-84900.

7weeks.o.g.=9700-120,000, 8weeks. o. g.=31100-184000, 9weeks.o.g. 61,200-152,000, 10weeks.o.g.=22,000-143,00, 14weeks.o.g.=14300-758,000.

b)Whole blood count:

Blood group O Rh(D)positive. Glucose random 5.09mmol/l

Erythrocytes 6.42x10-6/ul, Hgb.14.5g/dl/ hematocrit 44.6%, MCV69.5FL. MCH 22.6pg, MCHC 32.5g/dl, PLT 326x10-3/uL

Leucocytes 28.5x10-3/Ul (3.5-10.0000), Neutrophils 90% (40-70)

Basophiles 00%(0-2), Monocytes 00%(1-10), Eosinophils 0.2%(1-6),

Lymphocytes 8% (20-50).

c)Routine urine test:

Colour-Deep yellow

Appearance-Turbid

Chemical examination:

pH-8

Specific gravity-1020

Nitrites-Positive

Proteins-present

Glucose–normal

Ketones-negative

Urobilinogen-(+)

Bilirubin-(Negative)

Microscopy:

Leucocytes- numerous/HPF

Epithelial cells, 20-25/HPF

RBC, 4-6/HPF

Casts-nil

Others: Bacteria(+++)

d)TRIPLE (AFM) Vaginal Swab Test with BD AFFIRM- , VP III. Microbiological Identification Test;

GARDNERELLA VAGINALIS, (+++) positive.

CANDIDA ALBICANS, isolated in moderate growth after 48hrs incubation period.

TRICHOMONAS VAGINALIS (---) Negative.

e)Radiological investigations:

1)Ultrasonograph of the pelvis;

a)The patient refused a transvaginal USG imaging.

b)The urinary bladder was of a normal size, shape and capacity with neither stones nor any filling defect or in wall thickness seen.

c)The uterus had a size of 6.55/3.46cm. and of a normal shape and homogeneous echo texture, neither local nor diffused lesions were seen. The endometrial thickness was 0.6cm.

d)The left ovary was of the size 3.4/2.56cm with one follicle of size 1.6/1.4cm. The right ovary could not be seen.

e)No free fluid was detected in the CUL-DE-SAC
f)Radiological report on the abdominal cavity:

The liver was mildly enlarged 14.8cm kkd, with homogeneous structure with no visible focal or diffused lesions in the parenchyma, the intrahepatic ramificattions of the portal, hepatic veins were within normal size , the pv and cbd and intrahepatic channels were of normal size.

The gall bladder was of a normal size and shape, and contained neither stones nor thickened wall, the pancreas was of a normal size, the head and body were not enlarged, there were gases in the loops of the bowel and in the epigatrium, the spleen was of a normal size and echo presentation of both kidneys were of normal sizes, shapes and location, the pelvi-calyceal system was not dilated, and

they contained neither stones nor local or difused lesions. There were no stones or any pathological filling defects in the lumen.

The urinary bladder was of a normal capacity, well distended and with smooth wall.

g)Clinical data: Acute appendicitis, diffuse purulent peritonitis.

1)Histopathology and Cytology report: Gross findings, appendix 7cm long, 0.5cm in diameter, congested serosa. Mcroscopic findings: Sections show three cross-sections through the wall of the appendix, including the tip. Mucosal lining and muscular layers show no signs of inflammation. Heavy neutrophil infiltration of the appendiceal serosa with blood vessels dilation and congestion. Pathological diagnosis: Acute periappendicitis. Remarks: Primary site of the inflamation is probably located outside the appendix.

Microbiology report:The peritoneal fluid was cultured on the following culture media and the results were as the following: a)Blood agar, a moderate growth of Gardnerella vaginalis after 72 hours of culture, sensitive to Metronidazole and Trimethoprim. b)McConkey agar, a scanty growth of Gardnerella vaginalis after 96 hrs was cultured, sensitive to Metronidazole and Trimethoprim. c)Nothing grew from Chocolate agar nor from the Sabonron agar.

DISCUSSION

Vaginitis, one of the most common problems in clinical medicine acounts for more than 10 million office visits each year.($_1$) The three main categories of vaginitis are bacterial vaginosis(BV), yeast vaginitis (candidiasis) and T.vaginitis (Trichomoniasis). BV is the most common vaginal infection, and accounts for 15 to 50% of vaginitis/vaginosis depending upon the patient population.($_2$,$_3$). While Gardnerella vaginalis is no longer thought to be the only etiologic agent of BV, it is considered to be one of the major bacteria contributing to the infection which involves an increase in anaerobic bacteria and reduction in the normal lactobacillus flora.

The complications of BV can be especially significant in pregnant women, resulting in increased risk of adverse pregnancy outcome($_4$,$_5$) including pre-term labour($_6$) and birth($_7$,$_8$). In addition, recent data suggest BV-associated bacteria in the endometrium maybe etiologic agents of endometritis and pelvic inflammatory pelvic disease independent of Neisseria gonorrhoeae and Chlamydia trachomatis infection.($_9$) BV is also factor for the development of post-hysterectomy cuff cellulites($_{10}$)Vaginal candidiasis is the second form of vaginal infection seen in varied clinical settings($_3$). Three quarters of all adult women will experience at least one episode of vaginal candidiasis during lifetime, with 40 to 50% experiencing a second episode. Approximately 5% of the adult female population suffers from recurrent, often intractable yeast infection.($_3$).Laboratory methods for the identification of these organisms include microscopic evaluation, amine test, Gram stain, pH and culture.

Gardenerella vaginitis: G. vaginalis, previously classified as Haemophilus vaginalis, frequently found in asymptomatic women. It is assumed that it may be responsible for the development of an infection without the interaction of other bacteria. The infection becomes symptomatic when the vaginal pH rises to more than 4.5($_{13}$) The results should be interpreted with caution because the isolated bacterial agent is not always the etiological factor responsible for the clinical disease, culture should confirm the diagnosis. Although inflammatory lesions of the peritoneum are common they are usually diagnosed bacteriologically rather than histologically and only infrequently generate material for the pathologist..

Peritonitis is most commonly due to infectious and chemical causes. Infectious peritonitis is usually bacterial and may be primary or secondary. The primary form is rare and usually is caused by pneumococci or streptococci. Secondary bacterial peritonitis usually is due to perforation of the viscus usually within the gastrointestinal tract, eg.

Peptic ulcer, diverticulum, or tumour, and the resulting peritonitis is both bacterial and chemical. In women, a common cause of peritonitis is acute bacterial salpingitis. The same agents may involve the cervix and the cause acute and chronic lesions. The infection may be the result of direct invasion of the cervix, or spread of the infection from other parts of the genital tract and adjoining organs or blood borne contamination.

In case of pelvic-peritonitis, until culture results are available in patients with spontaneous bacterial peritonitis (SBP) broad coverage antibiotic should be directed against enteric organisms. Nephrotoxic drugs including Aminoglycosides should be avoided when ever possible. Cefotaxime 2g iv every 8hrs has emerged as the favoured agent for empirical treatment of SBP. Alternative agents

include: Ceftriaxone, Ceftazidime, Cefonicid, Ceftizoxime, and Ampicillin-Sulbactam. Meropernem, and Imipenem-Cilastatin as well as Fluoroquinolones (eg.Ciproflocacin, Levofloxacin, Galifoxacin and Moxifloxacin).

Traditionally, i.v. antibiotics have been administered for 10-14 days. However, 5days therapy appears to be effective, provided the patient is doing well clinically and that the ascetic fluid is sterile, with polymorphonuclear leucocytes count is below 250cell/mm³ before discontinuation of antibiotics.($_{11}$,$_{12}$).

Gardnerella vaginalis, although controversy remains over the role of G. vaginalis as a genitourinary pathogen, most who strictly define the clinical manifestations accept the organism as one major contributors to nonspecific vaginosis. These vaginal infections are limited to the production of a discharge with an offensive odour, resulting from the break down of proteinaceous products of parasitized degenerating squamous epithelial cells that are sloughed into the vaginal secretions. ($_{13}$). There is also a belief that G. vaginalis infection are sexually transmitted because the organism can be recovered in high concentrations from male sexual partners or females who incur re-infection after successful treatment of previous infection.($_{3}$). With improved culture methods, Gardnerella vaginalis is being recovered with increased frequency from various extra-genital sites such as blood, urine, male urethra, perinephric abscess, pharynx, and intra-abdominal fluid.

Josephson and Thomason focused on the role of Gardnerella vaginalis as a cause of urinary tract infection citing several reported cases in the literature. Greenwood has found that a rough correlation exists between symptoms and the number of organisms isolated in cultures. ($_{14}$,$_{15}$,$_{16}$)

CONCLUSION

Acute diffuse purulent peritonitis from Gardnerella vaginalis is rare in literature. This is however, a classic case of an uncooperative patient, who probably lost her first pregnancy from this disease. She may have had a psychological pit during this disease, with a history of chronic/acute infection of Gardnerella vaginalis and Candida albicans not treated early enough resulting in acute diffuse purulent peritonitis. Although not all patients cooperate readily with medical staff, however, with persuasion and appropriate treatment, recovery can be fast and without many complications. The sexual partners of such patients should be treated equally for infections to avoid future re-infections. Acute generalized peritonitis from pelvic-peritonitis could be caused by pathogens from vaginal infection, which in this case Gardnerella vaginalis is the causative pathogen.

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