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Case report

LATE PUERPERAL SEPSIS, CASE REPORT AND LITERATURE REVIEW

Palabras clave: Salpingitis; Peritonitis; Infección puerperal; Puerperio.
Keywords: Salpingitis; Peritonitis; Puerperal Infection; Postpartum.

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SUMMARY

A case of extremely rare puerperal sepsis is presented in this paper. Postpartum infection is an entity given in between 0.1% and 10% of postpartum patients and has a mortality rate ranging from 2% to 11%.

In this case report, a primigravida patient, age 19, presented hypogastric pain, emesis and fever five days after delivery. Postpartum endometritis and retained products of conception were diagnosed; uterine curettage was performed and antibiotic treatment was formulated with satisfactory outcome. The patient was discharged on the fourth day.

The patient was readmitted 27 days after delivery with hypogastric persistent pain and fever, vomiting, hypotension and pulmonary dysfunction; gynecological examination showed findings consistent with salpingitis and a laparotomy was performed to confirm the diagnosis, finding salpingitis along with pelvic peritonitis. An intravenous antibiotic treatment, laparotomy and peritoneal washings were provided with satisfactory evolution.

The literature on puerperal sepsis, myometritis and postpartum salpingitis is reviewed because, in order to improve morbidity and mortality, timely diagnosis and treatment are determining.

INTRODUCTION

The issue presented in this case report refers to late puerperal sepsis, more than eight days after partum, which is a rare entity and can be avoided through clinical management and appropriate antimicrobial therapy. Given these circumstances, the presentation of this case is relevant for the obstetric medical community.

CLINICAL CASE

A female patient, age 19, common-law marriage, technician, primigravida, gestation of 39.9 weeks, seven prenatal checkups and no history of importance, attended consultation due to onset of labor; she also reported persistent episodes of liquid stools —four a day— and negated other symptoms.

The physical examination found her weight was 70 kg, with body mass index of 27.3, normal vital signs, pain on widespread palpation of the abdomen, no signs of peritoneal irritation and uterine height of 32 cm. A longitudinal single fetus, cephalic and fetal heart rate of 125 beats/min, was observed. A speculoscopy was performed and homogeneous white discharge without amniorrhea was found, as well as a closed, short cervix and complete membranes. She was admitted with a full-term pregnancy diagnosis, and antepartum and induction of labor was prescribed.

The patient went into normal labor and gave birth a newborn weighing 2950 gr, 53 cm, good APGAR, uncomplicated birth by Schultz mechanism, complete and normal placenta and bleeding of 300 cm^3. Postpartum evolution was satisfactory with adequate uterine involution, therefore, the patient was discharged.

After four days, the patient entered again into the emergency room due to abdominal pain of half an hour of evolution with predominance in the lower abdomen and right iliac fossa, associated with an episode of emesis and unquantified fever. The patient was found normotensive, tachycardic, afebrile, with congestive secreting breasts, soft abdomen painful on palpation of the right iliac fossa, with doubtful Blumberg and a 7 cm infraumbilical tonic uterus. Bimanual examination showed a short, two fingerbreadth ac-

cessible cervix with clots in the cervical canal and scant non-fetid hematologic lochia palpated. Patient was admitted with a diagnostic impression of abdominal pain for study, postpartum endometritis *vs.* hematometra *vs.* acute appendicitis and breast engorgement.

Paraclinical tests included a full blood count that showed 9980/mm^3 of leukocyte, neutrophils of 73%, Hb of 14mg/dL, Hct of 41%, platelet count of 293,000/mm^3, PCR of 37 and normal urinalysis. A transvaginal ultrasound showed a 13 cm long uterus and a thickened hyperechoic endometrium of 24 mm.

Lochiometra without superinfection by thickening of the endometrial cavity was diagnosed and obstetric curettage was performed, in which a moderate amount of ovular tissue along with a fetid placental cotyledon were found. Through these findings, endometritis was diagnosed and antibiotic treatment with clindamycin and gentamicin was delivered for three and four days respectively, showing an adequate clinical evolution, so the patient was discharged.

Nine days after curettage, the patient attended a control consultation reporting occasional pain in the right flank, but with a normal physical examination. 27 days after partum, the patient visited the emergency room due to "low pain" and referred a clinical profile of one day of evolution with unquantified spiking fever and chills; she also presented non-fetid genital bleeding, a sharp pain in both breasts and constipation for two days. The patient denied engaging in postpartum sexual activity.

A physical examination was performed finding good general conditions: blood pressure of 93/62 mmHg, heart rate of 108/min, respiratory rate of 18/min and temperature of 36.7 °C; symmetrical soft breasts, without signs of infection, slightly painful to the touch and galactorrhea; pain in lower abdomen; hyperthermic vagina, with pain on palpation of annexes and cervical mobilization, without bleeding, and fetid uterine secretion. Patient was admitted under an obstetrical origin sepsis diagnostic impression, late puerperal infection and tubo-ovarian abscess *vs.* salpingitis. Antibiotic treatment with clindamycin and gentamicin was delivered.

Paraclinical tests included a urine analysis with no suggestion of infection and contamination, PCR of 16, blood count of 13,130 leukocytes/mm^3, neutrophils of 69%, Hb 13.9 mg/dl, Hct of 39.8% and platelets of 209000/ mm^3.

Given the antibiotic scheme received in the previous hospitalization, medication was changed the following day to piperacillin-tazobactam and azithromycin was added to cover *Chlamydia trachomatis*, and puerperal sepsis was considered due to the presence of tachycardia, tachypnea and leukocytosis; paraclinical tests were requested to complement the sepsis profile, along with blood cultures and transvaginal ultrasound, and thromboprophylaxis was begun.

Arterial blood gases showed pH of 7.45, PO_2 of 50.2%, PCO_2 of 25.9, HCO_3 of 18.2, BE of 12 mEq/L, delta hydrogen ions of +4.57, saturation of 87.7% with FiO_2 of 0.21. The doctor who assessed the patient interpreted this data as an acid-base balance, with hypoxemia, tissue hypoperfusion —lactate 1.2, cut-off point 1.0— and mild pulmonary dysfunction (PaFi: 238) with normal coagulation tests.

Transvaginal ultrasound showed a normal uterus, a normal endometrium, no adnexal masses, posterior fornix and right ovarian fossa with fluid collection of 7 cm^3; chest X - rays showed multiple interstitial infiltrates

without signs of consolidation, and blood cultures were negative.

Severe sepsis due to mild pulmonary dysfunction, PAFI 238, without involvement of other organs was diagnosed. Tubo-ovarian abscess was discarded based on sonographic findings.

On the second day of hospitalization, the patient complained of severe pain in the right iliac fossa, was afebrile, with sustained arterial hypotension —mean arterial pressure (MAP) of 62 mmHg— associated with tachycardia, tachypnea and absence of diuresis. She was transferred to the intermediate care unit for resuscitation and diagnosis of puerperal myometritis was suspected.

The pain was persistent on palpation of lower abdomen and right iliac fossa without peritoneal irritation, and bimanual examination showed a 10 cm with marked retroverted uterus, one fingerbreadth cervix, intense pain in the lower abdomen and a feeling of ballooning on the bottom side of the right fornix with an area of about 4 cm. Control arterial blood gases showed compensated metabolic acidosis; a probable diagnosis of pelvic collection was done and a laparotomy was decided, with prior informed consent about the possibility of a hysterectomy and residual infertility. Passage of a central venous catheter for possible vasopressor support was indicated.

During the exploratory laparotomy, an uterus approximately 8 cm long, pink surface, well perfused, normal looking ovaries, slightly edematous and erythematous tubes with fibrinopurulent membranes in fimbriae and iliac fossae, cloudy fluid in posterior fornix and scarce ascitic fibrinopurulent membranes, normal appendix and normal gallbladder were found. Two samples were taken for cultures and a pelvic peritonitis secondary to puerperal salpingitis was diagnosed. A cavity wash was performed and a hysterectomy was not performed given the normal appearance of the uterus; a laparostomy with Bogota bag was used.

Postoperatively, the patient presented MAP of 55 mmHg, central venous pressure CVP of 3 cm of H_2O, tachycardia, tachypnea, mucocutaneous pallor, hypoactive bowel sounds, Bogota bag without active bleeding, no signs of peritoneal irritation, genitourinary without active bleeding and a normal physical examination. Paraclinical tests showed PCR of 108, blood count without leukocytosis or neutrophilia, and normal renal and liver function.

Because the patient persisted with average blood pressure values below 65 mmHg, despite adequate fluid resuscitation, noradrenaline 0.05 µg/kg/min was administered. After seven hours, the patient continued with low mean blood pressure, so the drip of noradrenaline was increased to 0.15 µg /kg/min. It was considered that the patient presented multiple organ dysfunction caused by vascular dysfunction —septic shock— and pulmonary dysfunction with a SOFA score of 4.

The next day, the patient reported episodes of emesis at multiple times and breast engorgement; the attending physician found her tachycardic and afebrile with normal mean arterial pressure, thus the optimization of fluid resuscitation through a nasogastric tube and nutrition assessment was ordered.

During the second surgical wash, at 24 hours, Fallopian tubes with improved edema and erythema, scarce fibrinopurulent membranes in fimbriae and iliac fossae, normal pelvic infundibula and serohematic liquid in posterior fornix, from which a sample was taken to culture, were found. Puncturing of the uterus was performed, which allowed obtaining non-fetid hemorrhagic endometrial materials that were sent to culture; suture

material and Bogota bag were removed and the laparostomy was closed.

On the fifth day of hospitalization, recovery of gastrointestinal function was obtained and progressive vasopressor weaning and decreased water intake was initiated with adequate tolerance, and serial control paraclinical tests showed progressive improvement; blood cultures were negative at 48 hours.

On the seventh day of hospitalization, the peritoneal fluid culture report was received showing little growth of yeast at 48 hours of incubation; the patient continued with clinical improvement, tolerated full vasopressor weaning, started standing position, had adequate tolerance to soft diet, normal gastrointestinal function, marked reduction of abdominal pain and complete cessation of vaginal bleeding, which led to medical floor transfer.

On the ninth day, the peritoneal fluid culture results were received reporting *multi susceptible Candida spp.* Contamination was considered taking into account the satisfactory clinical and paraclinical evolution of the patient, who completed an antibiotic regimen of ten days without complications and was discharged.

DISCUSSION AND LITERATURE REVIEW

Based on the diagnoses of the patient, a systematic literature search was performed in the Medline database via PubMed with the following terms: ("Salpingitis" [Mesh] OR "Peritonitis" [Mesh] OR "Pelvic Inflammatory Disease "[Mesh] OR" Puerperal Infection "[Mesh]) AND "Postpartum Period" [Mesh]. 363 articles were obtained and filter "Human" was used, so the number decreased to 167. These articles were manually reviewed, finding 24 of them suitable for the review of this case. A search of book chapters related to the subject was also performed.

According to the World Health Organization **(1)**, uterine puerperal sepsis is defined as the infection occurred between the rupture of membranes and the first 42 days postpartum, with at least two of the following conditions: pelvic pain, fever —oral temperature equal to or higher than 38.5 °C— and purulent, cloudy or fetid vaginal discharge or delayed uterine involution.

The incidence of puerperal sepsis in developing countries is estimated to range between 0.1% and 10%, although the wide disparity of the estimates may be caused probably due to the difference in diagnostic criteria between different sources of study. It is estimated that puerperal sepsis causes at least 75000 maternal deaths each year, mainly in low-income countries (2). Mortality of puerperal sepsis, by region, is estimated at 11.7% in Asia, 9.7% in Africa, 7.7% in Latin America and the Caribbean, which is relatively high compared to 2.1% in developed countries (3, 4). Studies in high-income countries report an incidence of infectious disease due to sepsis from 0.1 to 0.6 per 1000 births **(2)**.

Identified causes of puerperal fever are associated with genital infection as in the case of endomyometritis, chorioamnionitis, pelvic abscess, septic pelvic thrombophlebitis, peritonitis, episiotomy and operative site infection, and other causes not associated with the genital tract such as urinary tract infections, mastitis, deep vein thrombosis, venipuncture site infection, cholecystitis, appendicitis, respiratory tract infections, rheumatic endocarditis, myocarditis, other infectious diseases — malaria, tuberculosis, HIV— malignant neoplastic diseases or drug induced fever **(5)**.

Although there is no clear definition, the time of onset of the infection and sepsis in the postpartum period is related to the stages of normal puerperium, with immediate events occurred within the first 24 hours, mediate events between the second and seventh day after delivery and late events from the second to the sixth week postpartum (42 days) **(6)**.

The most common risk factors for puerperal infection are caesarean section, prolonged labor, rupture of membranes with several hours of evolution, prior chorioamnionitis, repeated vaginal examinations, vaginal infections before delivery or caesarean section and internal fetal monitoring **(7)**. Acosta *et al.* **(8)**, in a study of cases and controls in a Scottish population, also found other risk factors such as obesity, operative vaginal delivery, being under 25 years old, multiparity, anemia, delivery induction and preterm birth. The single most important risk factor is the cesarean section, so prophylactic antibiotics administration during this procedure substantially reduce the risk of infection **(2)**.

In the diagnosis of puerperal infections that threaten the life of the patient, early detection is very important to reduce maternal mortality. Some alarming signs that should be taken into consideration are: fever higher than 38.9 °C and heart rate above 120 beats/min. Hypotension with systolic blood pressure below 90 mmHg or a basal reduction of 40 mmHg suggests severe sepsis and septic shock. Tachypnea with a respiratory rate above 20/min may be a sign of metabolic acidosis and the clinician should consider examining arterial blood gas for evaluating the patient **(9)**. Puerperal infections progress rapidly, therefore, a continuous assessment of the evolution of the patient must be done. Puerperal uterine infection can be topographically classified according to the compromised site **(9)** as shown in Table 1.

Table 1. Topographic classification of puerperal uterine infection.

I. Uterus engagement a. Endometritis b. Myometritis
II. Annexes and parametria engagement a. Salpingitis b. Tubo-ovarian abscesses c. Parametritis, pelvic cellulitis and septic pelvic thrombophlebitis
III. Peritoneum engagement a. Pelviperitonitis c. Peritonitis

Source: Own elaboration based on Angel-Müller & Gaitán-Duarte **(9)**.

Physical examination can be confusing because many women with symptoms of puerperal sepsis may have discrete local findings that suggest a less severe infection **(10)**.

Peritonitis secondary to puerperal uterine infection is a rare event; after reviewing the literature, it was found that Pańczyk *et al.* **(11)** made a review of a period of 10 years and found 2238 patients with caesarean section and eight of them had peritonitis, for a frequency of 0.36%. These patients underwent partial or total hysterectomy between four and seven days postpartum.

Krafft *et al.* **(12)** described the cases of six patients with puerperal peritonitis, noting that the main source of infection was the rise of pathogenic microorganisms from the vagina and reported the following as major risk factors: unknown vaginal microflora, the surgical technique used during the cesarean delivery and the premature rupture of membranes. In this study, laparotomy and removal of the uterus were also performed as part of patient treatment.

Rivlin **(13)** conducted a retrospective review between 1972 and 1976 in 176 women who had surgery due to diffuse peritonitis secondary to pelvic infections. Fifteen of these patients presented puerperal infection and their mortality rate was 6.7% (one out of 15). Factors associated with mortality found in this study included surgery after 24 hours and the lack of use of antibiotics with antianaerobic coverage. This series, despite dating back several years, emphasized the importance of early surgery and the inclusion of an antibiotic with antianaerobic coverage. This recommendation also appeared recently in sepsis management guidelines of the American Society of Critical Care Medicine **(14)**.

The etiologic agents of puerperal sepsis may include sexually transmitted bacteria, bacteria of microbiota endogenous to the patient or associations of both. Among sexually transmitted bacteria, *Chlamydia trachomatis*, *Neisseria gonorrhoeae* and less frequently *Mycoplasma hominis* and *Mycoplasma genitalium* are included. Among the bacteria of endogenous microbiota, *Enterobacteriaceae*, gram-positive cocci such as *Streptococcus spp.* and *Enterococcus faecalis,* and strict anaerobic bacteria are found. These infections are usually polymicrobial **(6, 9)**.

Historically, since the first half of the twentieth century, the Group A *streptococcus* (GAS) was acknowledged as one of the main causes of puerperal sepsis **(14-15)**, despite the fact that its incidence decreased significantly by the end of the century. However, the incidence and severity of this infection has recently increased for unknown reasons.

Risk factors that contribute to GAS infection include the mode of delivery, the site of attention, exposure to GAS carriers, the altered immune state associated with pregnancy, genetic susceptibility of the host, the virulence of the infecting strain and immune response specialized in the female genital tract organs **(15-16)**. Their importance lies on the high mortality rates secondary to complications such as toxic shock syndrome (TSS), necrotizing myometritis —known like this due to its similarity to the necrotizing fasciitis— pneumonia, septic arthritis and meningitis **(16-18)**.

GAS infection is characterized by its early onset in the postpartum period and its fulminant course, but cases of puerperal sepsis of late onset that may cause peritonitis and multiple organ dysfunction have been reported and an apparent focus is not always found **(19 -22)**.

Agalactiae Streptococcus, or Group B, also causes puerperal uterine infection and has been found as a single pathogen in between 2% and 14% of cases or in combination with other germs. Redondo -Aguilar **(23)** described a case of puerperal pyometra — pus accumulation in the uterine cavity— in which this bacterium was isolated.

Regarding other etiologic agents, no literature related to the late onset of puerperal infection is found, possibly because of the difficulty for finding the causative agent; for example, in a study conducted in Sudan between 2011 and 2012 to detect pathogens in patients with puerperal sepsis, blood samples were cultured and 72.9% positive samples were found (124 of 170), isolating *Staphylococcus aureus* (39.5%), *Clostridium perfringens* (27.4%), *Listeria monocytogenes* (16.9%), *Enterobacter cloacae* (10.5%) and *Staphylococcus epidermidis* (5.6%) **(22, 24)**.

In puerperal endometritis, routine culture is not advised because its etiology is polymicrobial, because it is difficult to obtain a sample that is not contaminated with vaginal microflora and because of the delay for obtaining results. Cultivation is useful in patients

with complications or that have not responded to initial treatment **(24)**.

Some pathogens associated with puerperal infections may require special techniques or media for collection, classification and culture, so the causative agent cannot always be identified. *Chlamydial trachomatis* and *Neisseria gonorrhoeae* are examples of these germs that require media such as Mac Coy and Tayher Martin cells, respectively, for cultivation. By the late 80s, a study found a prevalence of 20% of upper genital tract infections presented at around the seventh day of puerperium caused by these two agents, and even 30 days later in a much smaller proportion, causing endometritis or peritonitis **(23, 25)**. With respect to microorganisms other than bacterial agents such as Candida sp infections or other fungal infections, there are no reports of cases related to puerperal infection.

Compared with the general population, women infected with HIV are not related to an increased risk of puerperal infection, except in those cases of infections secondary to cesarean operations; nevertheless, it is unclear whether the risk of puerperal infection in these patients is directly attributable to pregnancy or indirectly to HIV infection **(24, 26)**.

In the case of this patient, who presented a pelviperitonitis secondary to a bilateral salpingitis on the 28th day postpartum, the fact that she received antibiotic treatment with clindamycin and gentamicin prior to hospitalization due to severe sepsis must be taken into account; it is worth noting that by not having sexual intercourse and in the absence of other risk factors, a sexually transmitted infection recently acquired as causing pelviperitonitis was discarded. Moreover, it is also worth noting that in the last hospitalization, more than 24 hours of antibiotic treatment at the time of sampling for culture had passed, which might explain why the cultures were negative for bacteria. Finally, considering the good immunological status of the patient and in the absence of conditions or immunodeficiencies that predispose to infections, the isolation of Candida in the culture was considered to be the result of peritoneal fluid sample contamination.

In the literature reviewed, no case reports of peritonitis secondary to postpartum tubal infection were found, so the strong point of this case is the proper management of the patient and the final lesson that uterine infection can affect belatedly fallopian tubes and be complicated due to peritonitis.

The handling of these cases must include, in addition to an extensive antibiotic coverage scheme, surgery to control the source of infection. The weak point of this case is the lack of identification of microorganisms causing this infection because cultures were taken after starting antibiotic treatment.

This case also shows that it is important to accurately assess sepsis and organ dysfunction in patients with puerperal infection and the topographic location of the site of infection through clinical, as well as the support of images to adequately determine the need for surgical intervention and control the source of infection

CONCLUSIONS

Peritonitis is a serious complication, but is rare in the postpartum period, and is usually secondary to myometritis. Salpingitis postpartum is a rare entity, and it is even less frequent to find a complicated salpingitis with peritonitis in a late postpartum period.

In the diagnosis of uterine infections, it is important to monitor systemic signs of infec-

tion and to evaluate the function of different organs and systems to detect severe sepsis or septic shock, which are indicative of severe infections that may require surgical management. Improving the care of severe sepsis in medical services, as promoted by the Surviving Sepsis Campaign **(14)**, may reduce the overall risks of maternal mortality and morbidity sepsis in low and high income countries.

In puerperal endometritis, cultures of the endometrium are not initially recommended; nonetheless, when there is no improvement with the first antibiotic scheme or complications arise, it is important to perform cultures to discover which microorganisms are causing the infection and to optimize antibiotic use.

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