MENINGITIS SEPSIS AFTER IUD INSERTION, A CASE PRESENTATION

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MENINGITIS SEPSIS AFTER IUD INSERTION, A CASE PRESENTATION (Abstract): Neisseria meningitidis is a normal commensal of human mucous membranes that is no longer considered to be restricted to the nasopharynx. Due to the practice of oral sex, the mucous membranes of the cervix, urethra or anus have become a potential infection site for this bacterium. Inserting an intrauterine device (IUD), can alter the protective barrier of the endocervical mucosa, allowing for bacterial infection and systemic spread. We present a case report of a 40-year old woman who presented with abdominal pain, spotting and fever after inserting an IUD and developed a fulminant septical shock. Blood cultures and cultures from ascites showed the presence of Neisseria meningitidis group Y. From our knowledge, there are a few cases presented in the literature of toxic shock syndrome after IUD insertion, caused by Staphylococcus aureus or Streptococcus group A, but this is the first case of meningococcal sepsis after IUD insertion described. So, even though IUDs rarely cause significant infection, physicians should consider this device as a possible source in reproductive-aged women with the clinical features of sepsis. Keywords: MENINGOCOCCUS, IUD, SEPSIS

Neisseria meningitidis, an encapsulated, gram-negative diplococcus, is a normal commensal of human mucous membranes. It is no longer considered to be restricted to the nasopharynx, although this is the most frequent site identified as its reservoir (1). Meningococci are spread through respiratory droplets, normally requiring close and prolonged exposure or direct contact with saliva and respiratory secretions. Spread can occur from an asymptomatic carrier or from a patient who is ill. Occasionally, shortly after the onset of colonization, N. meningitidis strains might penetrate the mucosal membrane and gain access to the bloodstream. Bacteria that are able to survive and multiply in the circulation may then cross the meninges and gain access to the cerebrospinal fluid. Of the various forms of meningococcal disease, the most common ones are meningitis and severe sepsis with often a rapid fatal outcome (2).

Due to the practice of oral sex, the mucous membranes of the cervix, urethra or anus have become a potential infection site for this bacterium, and by inserting an IUD, the protective barrier of the endocervical mucosa can be altered, allowing for bacte-
Primary infection and systemic spread.

Genital infection caused by *Neisseria meningitidis* (*N. meningitidis*) is not a common infection met in a gynecological practice, even if over the years there have been described cases when *N. meningitidis* was isolated from the urogenital tract, mostly as associated with subclinical infection (3-5) but also with cervicitis and even with salpingitis and peritonitis (1, 6). From our knowledge no case of sepsis related to a gynecological meningitis infection after IUD insertion was described until now.

Intrauterine devices (IUD) are a common used contraceptive method and even if it is known their association with a slightly increased risk for pelvic inflammatory diseases (cervicitis, endometritis, salpingitis, peritonitis), it is considered that the benefits of IUD insertion generally outweigh the risks (7).

In this case report we present a young woman that suffered a fulminant meningococcal sepsis presumably after an IUD insertion.

**CASE REPORT**

A 40-year-old woman, in a stable relation for one and a half years, zero-gravid, presented in November 2012 to the Obstetrics and Gynecological hospital, Malmö, Sweden, with lower abdominal pain and spotting after inserting an IUD. Anamnestic, she was suffering from Sjögren’s syndrome and in 1997 had been operated for thyroid cancer, now on substitution medication.

For two weeks back, inserted a Copper-IUD and have had since then low-moderate abdominal pain and vaginal spotting. She presents to the gynecological emergency department with abdominal pain and fever. Her triage vital signs were blood pressure (BP) 112/63 mm Hg, pulse 97 beats/min, and temperature 39.3°C.

Initial examination showed tenderness of the lower abdomen without muscular defense. There was a brownish discharge from the cervix at speculum examination and the patient reported severe pain on dislocation of the cervix and uterus. No adnexal masses were palpated. A wet smear of the vaginal discharge showed leukocytosis and clue-cells. Cervical specimens were taken for culture of pathogenic *Neisseria gonorrhea* and *Chlamydia trachomatis*. Vaginal ultrasound showed a normal uterus with the IUD lying in the endometrial cavity and normal ovaries; no fluid in the Douglas pouch was noted at that time. Venous blood proves showed a normal level reactive protein C at 1.7 mg/ml, WBC count 3.6/mm³ and normal renal, hepatic and electrolytes values.

The diagnosis of endometritis is set and the patient is sent home with oral treatment with Doxycillin, Metronidazol and anti-inflammatory. Seven hours later, she wakes up with abdominal pain, diarrhea, vomiting and rash and comes by ambulance to the emergency hospital with the initial suspicion of an allergic reaction to medication. The examination shows intense abdominal pain, an increased liver, peripheral cyanosis, hypotonia, petechiae, but no neck stiffness and normal lungs auscultation. Afebrile at incoming, in the next hour the temperature rises to 40°C, triage vital signs are now BP 90/60 mmHg, pulse 120 beats/min. Laboratory results show WBC count 0.8/mm³, platelets 11/mm³, creatinine 165 mg/dl, reactive protein C 67 mg/dl, base excess - 10 mmol/l and lactic acid 8 mmol/l. Deteriorates fast, needing intubation. Cultures are taken from blood, urine, throat,
Meningitis sepsis after iud insertion, a case presentation

nose and an initial dose of Meropenem and Tobramycin is administered. A CT thorax and abdomen without contrast, shows a lot of fluid in the abdomen, an increased liver with edema around the intrahepatic veins and bilateral infiltrates at the basis of the lungs with pleural fluid. With a fulminant progress of sepsis, the patient becomes anuric, with distended abdomen and marmorated extremities and coagulopathy. The IUD is removed and diagnostic laparoscopy is performed next morning but only via the camera port because of the patient’s increased abdominal pressure. The image (of poor quality) shows normal appearance of the bowels that flow in serous fluid. The genital organs are not seen, but there are no signs of peritonitis or abscess. Culture from the ascites is taken.

In a matter of hours she develops dry gangrene in fingers and toes, probably after septic embolism and aggressive vasopressor treatment.

At this time the culture results become available and they are positive for Neisseria meningitidis group Y, in blood and the peritoneal fluid.

Under broad spectrum antibiotic treatment, respiratory support and continuous renal replacement therapy, the patient starts to recover, but ten days after admission, her evolution becomes unstable again with intense abdominal pain and hypotension, and requires laparotomy because of suspicion of internal bleeding. Under operation spleen rupture is found, presumably after spleen infarct as secondary complication to sepsis and a splenectomy is performed, the internal genitalia are also inspected but except some secondary fibrin deposits on the uterus, no other pathology is seen.

After another complication in form of candida sepsis with retinitis, she finally slowly recovers and after nine weeks of hospitalization, is discharged with planned follow ups at orthopedic, hand surgery, endocrinology and ophthalmology clinics.

DISCUSSION

Sepsis after insertion of an IUD is a rare condition. The few cases found described in literature referred to toxic shock syndrome after IUD insertion caused by Streptococcus group A (8, 9) and Staphylococcus aureus (10). We haven’t found any case reporting meningococcal sepsis after an IUD insertion. In this case presentation, there was no available culture from cervix or from the removed IUD, but beside the positive blood culture, the only other positive culture for N. meningococcus was in the peritoneal fluid. Since the patients symptoms started together with the IUD insertion, and her only complain was gynecological, this was considered the primary focus.

There are at least 13 serogroups of N. meningitidis, with the most important being serogroups A, B, C, Y and W-135 (11). Meningococcemia is defined as dissemination of meningococci into the bloodstream. The fundamental pathologic change in meningococcemia is widespread vascular injury characterized by endothelial necrosis, intraluminal thrombosis, and perivascular hemorrhage. Endotoxin, cytokines, and free radicals damage the vascular endothelium, producing platelet deposition and vasculitis. The deleterious effects of cytokines play a major role in the pathogenesis of meningococcemia by causing severe hypotension, reduced cardiac output, and increased endothelial permeability (12). Management should focus on the treatment of the urgent clinical issues in addition to the administration of specific antibiotic
therapy. Empirical antibiotic therapy for suspected meningococcal disease consists of a third-generation cephalosporin to cover the various other (potentially penicillin-resistant) bacteria that may produce an indistinguishable clinical syndrome (13).

As isolation of Neisseria gonorrhea has been reported from pharynx (14), findings of N. meningitidis in urogenital sites has been also described over the years, most probably due to changes in sexual behavior and habits during the last few decades, where the majority of cases of infection occur via the urogenital contact, with the nasopharynx acting as the major reservoir in healthy carriers (3).

There have been found healthy carriers of meningococci in urethra, cervix and rectum in both males and females, where meningococci were isolated from pharynx of partners (15,16). There are also cases described of urethritis, anitis cervicitis, salpingitis (1, 3, 6, 16) associated with the presence of N. meningitidis.

CONCLUSIONS

Even though IUDs rarely cause significant infection, emergency physicians should consider this device as a possible source in reproductive-aged women with the clinical features of sepsis. And since the isolation of Neisseria meningitidis has been reported from urogenital sites, it is recommended in cases of suspected sepsis related to an IUD insertion to take also in consideration Neisseria meningitidis as a potential pathogen, along Streptococcus group A and Staphylococcus aureus.

REFERENCES

Meningitis sepsis after iud insertion, a case presentation


MESOTHELIOMA NOT ASSOCIATED WITH ASBESTOS EXPOSURE

Despite asbestos being identified as the single most important cause of malignant mesothelioma, the tumor is known to occur in only 10% to 20% of heavily exposed individuals. In addition, about 20% of the patients have no history of asbestos exposure even after detailed assessment. It should be realized that although there is a strong link between the development of malignant mesothelioma (MM) and asbestos exposure, not all cases are etiologically related to asbestos exposure. The proportion of cases attributable to asbestos exposure varies between the sexes and country, according to occupation and use of amphibole asbestos. A number of other agents have been implicated in the causation of MM but the best evidence appears to relate to another mineral fiber erionite and to irradiation. Erionite has emerged as the most important example of non asbestos-mediated cause of mesothelioma in regions such as Turkey where exposure to this type of fiber is highly prevalent. Ionizing radiation is a recognized human carcinogen and an established risk factor for several different types of cancers including hematologic malignancies and solid tumors. Cases of MM of the pleura, peritoneum, and pericardium have been reported in humans after therapeutic irradiation and thorium dioxide administration. Also, there is a strong experimental, epidemiologic and molecular evidence to suggest a possible carcinogenic or cocarcinogenic role of viruses such as SV40, a monkey polyoma virus, in the induction of MM. Although the polyoma virus SV40 has been unexpectedly discovered as an effective cocarcinogen of asbestos in the causation of animal mesothelioma, its potential role in human mesothelioma remains unproven. (Bharat Jasani, Allen Gibbs. Mesothelioma Not Associated With Asbestos Exposure. Arch Pathol Lab Med. 2012;136:262–267).

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