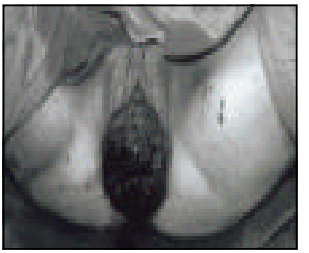

Figure 1 - The uterus and cervix that is edematous, desiccated, dark blue-red and covered with bloody secretions.

clindamycin 600 mg intravenously 4 times daily and gentamycin 80 mg intravenously 3 times daily. Tetanus vaccine and immunoglobulin were also administered. Her prolapsed uterus was manually reduced to its normal position and then 1% oxytocin solution infusion was started to promote uterine contractility. She was placed at Trendelenburg position with strict bed rest. On the second day, oxytocin was stopped and methylergobasine 0.125 mg 3 times daily was started and continued for 3 days, per orally. She was discharged on the 8th postpartum day and no prolapse of the uterus was noted during the follow up period of 6 months.

The cause of uterine prolapse is unclear, but in several predisposing factors (such as, childbirth trauma, congenital and developmental weakness, and the influence of menopause), failure of the supportive ligaments leading to prolapse of the uterus and vaginal vault is thought to be the most important factor. Although the diagnosis of uterine prolapse is usually based on clinical signs and symptoms, it is clinically important to differentiate it from acute uterine inversion. The classical signs of acute total uterine inversion are: shock incompatible with the quantity of blood loss, absence of uterine fundus on abdominal examination and no visualization of the cervix.[3] Our case was hemodynamically stable and the cervix was visible, which confirmed uterine prolapse. Delayed treatment leads to impaired lymphatic and venous drainage, resulting in acute edema of the protruding uterus and cervix, thus, its reduction is difficult without general anesthesia. Delayed treatment also result in mechanical trauma that causes ulceration and infection of the edematous cervix and even severe urinary tract infection due to acute urinary retention.[4] Considering that the woman had delivered the baby under non-sterile condition, prophylactic antibiotics and tetanus immunization were used, without delay in diagnosis.

Differently from acute uterine inversion, manual manipulation without general anesthesia before the development of excessive edema, slight Trendelenburg position with bed rest is the treatment modality in successful reduction of prolapse, and this will protect the patient from above discussed complications. Agents such as oxytocin or methylergobasine are then given to produce uterine contraction to prevent a second prolapse. Since perineal descent on straining is almost always evident immediately after vaginal delivery and returns to normal position during the subsequent 2 months,[5] a control examination 2 months after vaginal delivery was recommended, reminding that on this case, there is a possibility of recurrence on the next pregnancies.

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Spontaneous bacterial peritonitis due to *Hafnia alvei* in a patient with peritoneal mesothelioma

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Spontaneous bacterial peritonitis (SBP) is a frequent and severe complication of cirrhotic patients with ascites. It has also been reported in patients with chronic active hepatitis, acute viral hepatitis, congestive heart failure, metastatic malignant disease, systemic lupus erythematosus, lymphedema, and rarely without any underlying disease.[1] Most patients with SBP have symptoms

and signs clearly suggestive of peritoneal infection, especially abdominal pain, fever and alterations in gastrointestinal motility. However, SBP may be asymptomatic or there may be only minor symptoms in some patients.[2] Peritoneal mesothelioma is a rare neoplasia usually associated with exposure to asbestos. The incidence in the population not in contact with asbestos is of one per million per year. The disease is most common in males over the age of 40, with signs and symptoms of neoplasic disease together with abdominal pain and ascites with or without a palpable abdominal mass.[3]

A 60-year-old man was admitted to a hospital for abdominal distention, indigestion and dry mouth. The laboratory findings were not suggestive of a liver disorder. He was hospitalized for etiological investigation. Although, no abdominal mass was palpated ascites was found on physical examination. Paracenthesis fluid was cloudy and chemically exudative but not hemorrhagic. White blood cell was 4600/mm^3 with 86% neutrophils and no organism was isolated in the material. Ceftriaxone (2x1 gram intravenously) treatment was initiated empirically. Since hyperplasic mesothelial cells were detected in cytological examination of the ascites material, diagnostic laparoscopy was performed in this afebrile patient with a relatively well general status. During laparoscopy, approximately 4 liters of dark yellow, sticky mucous ascites material was drained. Lipoid, irregular multiple mass lesions were detected on visceral and parietal peritoneum. *Hafnia alvei (H. alvei)* was isolated in biopsy specimens that were obtained from parietal peritoneal lesions. It was susceptible to ciprofloxacin, imipenem, and aminoglycosides such as netilmisin, amikacin. The patient was reported as having malignant mesethelioma after pathological evaluation and then was referred to the oncology department. Microorganisms, presumably of enteric origin, account for up to 75% of the pathogens in SBP. *Escherichia coli* is the most frequently recovered pathogen, followed by *Klebsiella pneumoniae*, *Streptococcus pneumoniae*, and other streptococcal species, including enterococci.[1,4] In literature, we observed some uncommon bacteria as a cause of SBP, such as *Pasturella multocida*, *Listeria monocytogenes* and *Brucella melitensis*. All those patients were cirrhotic due to chronic liver diseases caused by alcohol or hepatitis C virus. *Hafnia alvei* is an extremely uncommon cause of peritonitis since there is only one literature about peritonitis caused by *H. alvei*.[5]

Hafnia alvei, is a Gram-negative aerobic bacillus in the family *Enterobacteriaceae* that may occur as a gastrointestinal commensal. It is not frequently involved with infection. It is found in sewage, soil and the large intestines of humans.[6] Infections due to *H. alvei* are acquired nosocomially or occurs in patients with chronic underlying illnesses, including chronic obstructive pulmonary disease, diabetes, chronic renal failure and malignancy. Many different infections due to *H. alvei* have rarely been described such as lung infections, and diarrheal diseases. Extra intestinal invasive infections caused by the organism usually occur in patient with chronic debilitating disorders and they are frequently isolated after antibiotic treatment. In the patient, there is a history of using third-generation cephalosporin before isolation of *H. alvei.* Despite most isolates of *H. alvei* reported in the literature are susceptible to third-generation cephalosporins, antibiotic susceptibilities that appears to be similar to those of the Enterobacter group.[6] In addition, our patient's isolate was resistant to all penicillins and cephalosporins and their combination with beta-lactamase inhibitors. It was susceptible to ciprofloxacin, imipenem, and aminoglycosides such as netilmisin, amikacin. Even though most patients with SBP have symptoms and signs suggestive of peritoneal infection, the clinical manifestations may be atypical in some patients. There were no signs and findings of a typical peritonitis in our patient. The diagnosis of peritonitis can usually be established by paracentesis. If Gram-negative organisms, a mixed flora, or no organisms are obtained, full exploratory laparotomy/laparoscopy is indicated to rule out possible intra abdominal sources of continuing peritoneal contamination.[1]

In conclusion, we believe that if no microbiological evidence is found in peritoneal fluid of patients with unexplained ascites, peritoneal biopsy for cytopathological and microbiological examination should be considered.

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Antibiotic resistance patterns of *Acinetobacter* species isolated in King Hussein Medical Center, Jordan

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Acinetobacter species is aerobic, Gram-negative coccobacilli, oxidase-negative, catalase positive, nonfermenting bacteria. Though widely prevalent in nature and generally regarded as commensals of human skin, respiratory and genitourinary tracts colonize the skin up to 25% and up to 7% in the pharynx of healthy adults.[1]

In 1986, taxonomy of the genus *Acinetobacter* was changed extensively by Bouvet and Grimont,[2] who outlined 12 different species by DNA-DNA-hybridization, including the named species *Acinetobacter Baumanii* (*A. Baumanii*), *Acinetobacter Calcoaceticus* (*A. Calcoaceticus*), *Acinetobacter Haemolyticus* (*A. Haemolyticus*), *Acinetobacter Johnsonii* (*A. Johnsonii*), *Acinetobacter Junii* (*A. Junii*) and *Acinetobacter Lwoffii* (*A. Lwoffii*) and 6 unnamed genomic species. Most of *A. Baumannii* and all *Acinetobacter* species strains 3 and 10 represent organisms that were formerly classified as *Acinetobacter Anitratus* (*A. Anitratus),* whereas all *A. Junii, A. Lwoffii* and *Acinetobacter* species strains 11 were formerly classified as *A. Lwoffii*.[3] These species are often multiresistant to antibiotics, meaning that therapy and infection control are complicated.[4] *Acinetobacter* species now known to be responsible for a wide range of nosocomial infections, including bacteremia, secondary meningitis, urinary tract infections, pneumonia, tracheobronchitis, endocarditis, wound infections and surgical site infections. *Acinetobacter* infections are most frequently associated with the use of a ventilator, urinary tract catheter or other invasive device. In the United States of America, among the intensive care unit (ICU) patients, during the period 1987-1996, reported cases of nosocomial *Acinetobacter* infections were 3447, the average rate of infection being significantly higher during summer than in winter.[4]

The aim of this study was to determine antibiotic resistance rates of *Acinetobacter* species strains isolated from patients in order to give information to clinicians when empiric therapy is necessary. A total of 133 consecutive, non duplicate *Acinetobacter* species isolates were studied over a period of 18 months, between July 2000 and December 2001 from various clinical materials at King Hussein Medical Center, Amman, Jordan, which is a 800 bed hospital. Duplicated isolates from the same infective episode in the same patients were excluded. According to the instructions provided by the manufacture, Vitek-1 system (Bio Merieux, France) were used for identification and studying the susceptibility of isolates, using V 1306 vitek GNI (Gram-negative identification) card for identification of the isolates, and V 4313 vitek GNS-528 (Gram-negative susceptibility) cards were used for studying the susceptibility of *Acinetobacter* isolates, and for the susceptibility testing of the isolates from urine using V 4525 Vitek GNS-203. *Echerichia coli* ATTCC 25922, *Staphylococcus aureus* ATTCC 25923 and *Pseudomonas aeruginosa* ATTCC 27853 were used as quality control organisms.

During the period of 18 months, a total of 133 *Acinetobacter* species isolated from clinical specimens were tested for antimicrobial susceptibility, among these isolates were 38 (28.57%) were from urine, 31 (23.31%) from blood, 54 (40.60%) from wounds, 7 (5.26%) from sputum, and 3 (2.55%) from other specimens. *Acinetobacter Calcoaceticus Biotype Anitratus* was the most common *Acinetobacter* species isolated with 130 isolates (97.74%), followed by *A. Lowffii* with 2 isolates (1.50%) and *A. Calcoaceticus-Bumanii* complex with 1 isolate (0.75%). The results of activities of antimicrobial agents against the *Acinetobacter* isolates are shown in **Table 1**. The most active antimicrobials for the isolates from urine was minocycline, while for the isolates from other specimens were the imipenem, ticarcillin/clavulanate, ceftazidime and netilmicin. An important feature of *Acinetobacter* species, is their intrinsic resistance to multiple antibiotics. Recently, reported surveys have demonstrated high rates of resistance to aminoglycosides, cephalosporins, quinolones, penicillins, monobactams, and imipenem, often in excess of 50%, among clinical isolates of *Acinetobacter*. Antimicrobial treatment of the infections due to highly resistant *Acinetobacter* strains can lead to treatment failure, and is associated with an increased risk of death.[5]