Idiopathic spontaneous rupture of the urinary bladder - A rare cause of death

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Abstract: Spontaneous rupture of the urinary bladder is a rare clinical condition, with a prevalence of about 1 in 126,000 hospitalizations. Idiopathic rupture is even more rarely encountered, constituting less than 1% of all cases. Given the low incidence, atypical symptoms and poor history data, the diagnosis is often established late and treatment is delayed, resulting in a high rate of mortality. Also, spontaneous rupture of the urinary bladder may be a surprise at the autopsy. In this paper, the authors present the case of a 67 years old woman who died due to idiopathic spontaneous rupture of the urinary bladder after ingestion of alcoholic beverages. The autopsy showed no signs of pathological or traumatic injury and the toxicological examination revealed a blood alcohol concentration of 2.05 g %. The case is analyzed in the context of existing literature data, taking into account the elements of anatomy, physiology and pathophysiology that can determine or favour the idiopathic spontaneous rupture of the urinary bladder.

Key Words: urinary bladder, idiopathic spontaneous rupture, alcohol.

The spontaneous rupture of the urinary bladder can occur intra or extra-peritoneal or in the pelvic tissue, with no history of trauma, having an incidence of approximately 1:126,000 hospitalized cases [1, 2]. Idiopathic spontaneous rupture is a very rare entity, representing about 1% of all the cases of bladder rupture. Acute alcohol intoxication is an important risk factor for idiopathic spontaneous rupture of the bladder. The diagnosis is often delayed and therefore treatment is delayed, resulting in increased morbidity and mortality. Sometimes the idiopathic spontaneous rupture of the bladder is not even diagnosed or suspected during the victim's life, being revealed during the autopsy.

CASE PRESENTATION

R.D., a 67 years old woman, was found dead few hours after ingestion of a large amount of alcoholic beverages. The victim's medical records showed that she was not suffering from any pathological condition. Investigation by police revealed no recent trauma suffered by the victim.

The autopsy was performed 24 hours after death, and the blood and organ samples were collected to perform toxicology and microscopic examinations.

External examination of the corpse found a stature of 165 cm, good nutritional status and signs of death completely expressed. No external traumatic lesions were observed.

Internal examination of the body revealed 1000 ml of liquid blood mixed with urine and blood clots into the peritoneal cavity (Fig. 1). On the rear wall of intraperitoneal urinary bladder it was discovered a transversal rupture of about 4 cm length and 3 cm width at maximum tensile, with irregular and hemorrhagic infiltrated edges. Inside the urinary bladder we observed about 700 ml of blood clots (Figs 2, 3). Further macroscopic examination of the urinary bladder

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revealed no other pathological or traumatic lesions. No other pathological or traumatic lesions were discovered during the internal examination.

During the autopsy we collected blood samples and samples of organs (stomach, liver, kidney) for toxicology examination and also samples of organs for microscopic examination.

Toxicology examination revealed a blood alcohol concentration of 2.05g ‰. No other toxic substances such as drugs, pesticides or drugs were detected.

**Microscopic examination**

The urinary bladder samples collected during the autopsy were preserved in formol 10% and processed by inclusion in paraffin. The sections were coloured with the standard method HE and the tricromic coloration Masson for a better differentiation of the tissue elements which are normally present in the wall of the urinary bladder. The sections were examined with the optic microscope Olympus Bx51, and the images were captured with the camera Olympus SP 350, QuickFoto program.

The microscopic examination revealed focal postmortem autolysis lesions in the urothelium and vascular hyperemia (Figs 4, 5) associated with focal hemorrhagic necrosis in the mucosal membrane and in the smooth muscle fibres of the muscular membrane (Figs. 6, 7) and also extensive hemorrhage in the entire wall of the urinary bladder (Fig. 8). The hemorrhage consisted of normal red cells and a few white cells, accompanied by a minimal inflammatory infiltration. The microscopic examination did not reveal any granulomatous inflammation suggestive for tuberculosis (Fig. 9).

**Figure 1.** Liquid blood and clots mixed with urine in the peritoneal cavity.

**Figure 2.** Transversal rupture of the rear wall of the urinary bladder; intracavitary blood clot.

**Figure 3.** Urinary bladder wall with hemorrhagic infiltration in the margins of the wall rupture.

Microscopic examination of the samples collected from the other organs did not reveal any pathological aspects which could determine or contribute to the rupture of the urinary bladder.

Summarizing, in this case, the medical records and autopsy showed no evidences of previous illnesses or traumatic injuries. The only macroscopic autopsy findings were: the rupture of the urinary bladder and an important break of urine mixed with blood in the peritoneal cavity. The microscopic examination of the samples of urinary bladder wall collected during autopsy revealed the hemorrhagic infiltration caused by the rupture of the wall without detecting any pathological or traumatic lesions of the bladder. Toxicological examination revealed a blood alcohol concentration of 2.05 g‰. Based on these findings, it was concluded that the victim died as a result of the spontaneous idiopathic bladder rupture due to over-distension in the context of the acute alcohol intoxication.
DISCUSSION

Rupture of the urinary bladder occurs mainly in the context of an abdominal-pelvic or iatrogenic trauma, or during obstetrical, urological and orthopedic maneuvers. Traumatic rupture of the bladder may occur intra-peritoneal, as a result of a contusion trauma of the abdomen or extra-peritoneal frequently associated with pelvic fractures.[15]

Spontaneous rupture occurs less frequently than the traumatic one, often against a background
of pre-existing pathology, at a rate of about 1/126,000 hospitalizations [2, 12, 15, 16]. Most cases occur in people aged 50 years or over who suffer from certain pathological diseases [3]. The first case of spontaneous rupture of the bladder bladder was reported by Bastable et al., in 1959 [16].

Spontaneous bladder rupture is often difficult to distinguish from other causes of acute abdomen [1, 2, 4, 5], representing a diagnostic challenge [2]. Due to diagnostic difficulties that often delay the treatment, it is estimated that mortality caused by the spontaneous rupture of the urinary bladder is about 47% [15].

Intra-peritoneal ruptures are divided in two categories, depending on their etiology: rupture of the bladder due to a pathological lesion and rupture by over-distension. The pathological conditions that can determine the rupture of the urinary bladder wall are: chronic infections [2], tuberculosis [9], urethral rupture of infected rupture, eosinophilic or necrotic cystitis [1, 2], inflammation [5, 12], pelvic irradiation for cervical cancer [17], urinary squamous cancer [4]. In children, spontaneous rupture may occur due to a congenital bladder diverticulum [7], and in newborn risk factors are represented by hypoxia and bladder diverticula [8]. The break of the urinary bladder wall by over-distension determined by the retention of urine determined by: tabes dorsalis, urethral obstruction by the gravid or fibroid uterus, puoerperal or postoperative retention [3, 6]. The spontaneous rupture of the bladder can be facilitated by excessive alcohol consumption [22].

Clinical aspects of the bladder rupture are varied. Before the rupture the victim suffers from commonly urinary tract symptoms, and after the rupture the signs and symptoms of peritonitis and urinary difficulties can be observed [3]. In many cases the clinical triad of pain, difficulty or impossibility of urinating and contracture of the abdominal wall can be identified [19]. In some instances, the rupture may remain painless for several days, but with progressive abdominal distension due to urinary ascites [15, 21]. After about 24 hours from the occurrence of the rupture, the phenomenon of self-peritoneal dialysis occurs resulting in the reabsorption of urea and creatinine, which determine metabolic disturbances with toxic blood concentrations of urea, creatinine, potassium (which can cause EKG changes), ammonia and hyponatremia, leading to metabolic acidosis and mimicking the biochemical acute renal failure. The peritoneal fluid, in turn, shows an increased concentration of creatinine [3, 13, 15, 17, 22].

Complications caused by the leakage of the urine into the peritoneal cavity and pelvis are: pelvic abscess, septic shock, abdominal or chest wall phlegmon [1].

Bladder rupture is difficult to diagnose due to nonspecific symptoms (abdominal aches and contracture of the abdominal wall), no history of trauma and low incidence. The diagnosis should be suspected when the following signs are identified: free peritoneal fluid, decrease demission of urine, hematuria, increased levels of urea and creatinine in serum and/or peritoneal fluid [21]. The investigation of choice for the detection of bladder rupture is cystography [3, 11, 20]. However, many times the spontaneous rupture of the bladder is not clinically suspected, being discovered by exploratory laparotomy or during autopsy [20].

This clinical entity is a surgical emergency and can be rapidly fatal if diagnosis and treatment are delayed, early and adequate treatment being essential [1]. The treatment consists of laparotomy and suture as continuous leakage of urine does not allow spontaneous closure of the breach [15].

Idiopathic spontaneous rupture of the urinary bladder wall appears in the absence of traumatic or pathological lesions being a very rare occurrence [10, 12, 14, 24], with an incidence of approximately 1% of all the ruptures [15]. The typical aspect is that of intraperitoneal rupture of the rear wall, most often affecting the bladder dome [17-19, 22].

The mechanism of the idiopathic spontaneous rupture of the urinary bladder is the excessive increase of the intra-vesical pressure [22], the most vulnerable part being the intra-peritoneal bladder [15].

The main cause of this clinical entity is the excessive consumption of alcoholic beverages [10, 15]. The acute alcohol intoxication increases the urine production due to the diuretic effects of alcohol and leads to over-distension in the context of the sensitivity disturbances due to the α - sympathomimetic effect and CNS depression produced by the alcohol. In men, congestion of the prostate and prostatic urethra aggravates the obstruction of the outlet [15, 22, 23]. Studies in animals showed that ethanol affects the contractility of the detrusor in vivo and in vitro and the chronic exposure to ethanol and acetaldehyde decreases the contractility of the urinary bladder. Therefore, chronic alcohol consumption is more prone to urinary retention than acute intoxication [23].

It is possible that the rupture to occur after minor trauma frequently forgotten by the patient due to the state of ethanol intoxication, resulting in diagnosis errors or delay. [11, 22]. Thus, in many cases initially classified as idiopathic were identified signs of minor trauma associated with the abuse of alcoholic beverages [15].

Urinary bladder rupture in the context of excessive alcohol consumption has a favorable prognosis if diagnosed and treated in due time [10]. Unfortunately, the morbidity and mortality of idiopathic rupture is increased as a result of late diagnosis and treatment. Bastable et al., in 1959 assessed a mortality rate of approximately 50% [22], while Festini et al., in 1991 reported a mortality rate of 12% [24].
Due to the difficulties of diagnosis and rapid evolution, idiopathic spontaneous bladder rupture may be primarily discovered during the autopsy. Such cases may be referred to the service of Forensic Medicine due to the suspect and sometimes sudden nature of the death. The post-mortem diagnosis of the idiopathic spontaneous rupture of the urinary bladder requires a thorough autopsy and laboratory examination. The correct diagnosis requires exclusion of any pathological or traumatic causes of breakage in addition to the discovery of the rupture itself accompanied by an important quantity of urine in the peritoneal cavity as an evidence of the bladder over-distension. As showed above, idiopathic spontaneous bladder rupture occurs mostly in the context of acute ethanol intoxication. As such a high blood alcohol concentration revealed by the toxicological examination is an important criteria for this diagnosis.

Although the idiopathic spontaneous rupture of the urinary bladder is a rarely encountered in the forensic practice, the professionals in this field have to be aware of this entity and collect all the evidences that support its post-mortem diagnosis.

References