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Decizie de indexare a faptei de plagiat la poziţia 00108 / 28.06.2014 şi pentru admitere la publicare în volum tipărit

care se bazează pe:

A. **Nota de constatare şi confirmare a indiciilor de plagiat** prin fişa suspiciunii inclusă în decizie.

Fişa suspiciunii de plagiat / Sheet of plagiarism's suspicion	
Opera suspicionată (OS)	**Opera autentică (OA)**
Suspicious work	**Authentic work**
OS	SINESCU, Ioanel. Litiaza aparatului urinal. In: SINESCU, I. ed. *Urologie clinică*. Bucureşti : Editura Medicală AMALTEA. 1998. p.141-161.
OA	SPIRNAK, J.P. and RESNICK, M.I. Urinary stones. In: TANAGHO, E.A. and McANINCH, J.W. eds. *Smith's General urology*. 13th edition, Norwalk, California: Appleton & Lange. 1992. p.271-298.
Incidenţa minimă a suspiciunii / Minimum incidence of suspicion	
p. 141: 34s - p.142:03s	p.271: 22s - p.271:29d
p.142: Tabel 11-2	p.275: Table 16-1
p.153: 26s – p.153:40s	p.290: 30d – p.290:45d
Fişa întocmită pentru includerea suspiciunii în Indexul Operelor Plagiate în România de la Sheet drawn up for including the suspicion in the Index of Plagiarized Works in Romania at www.plagiate.ro	

Notă: Prin „p.72:00" se înţelege paragraful care se termină la finele pag.72. Notaţia „p.00:00" semnifică până la ultima pagină a capitolului curent, în întregime de la punctul iniţial al preluării.

Note: By „p.72:00" one understands the text ending with the end of the page 72. By „p.00:00" one understands the taking over from the initial point till the last page of the current chapter, entirely.

B. **Fişa de argumentare a calificării** de plagiat alăturată, fişă care la rândul său este parte a deciziei.

Echipa Indexului Operelor Plagiate în România

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Fişa de argumentare a calificării

Nr. crt.	Descrierea situaţiei care este încadrată drept plagiat	Se confirmă
1.	Preluarea identică a unor pasaje (piese de creaţie de tip text) dintr-o operă autentică publicată, fără precizarea întinderii şi menţionarea provenienţei şi însuşirea acestora într-o lucrare ulterioară celei autentice.	✓
2.	Preluarea a unor pasaje (piese de creaţie de tip text) dintr-o operă autentică publicată, care sunt rezumate ale unor opere anterioare operei autentice, fără precizarea întinderii şi menţionarea provenienţei şi însuşirea acestora într-o lucrare ulterioară celei autentice.	
3.	Preluarea identică a unor figuri (piese de creaţie de tip grafic) dintr-o operă autentică publicată, fără menţionarea provenienţei şi însuşirea acestora într-o lucrare ulterioară celei autentice.	
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5.	Republicarea unei opere anterioare publicate, prin includerea unui nou autor sau de noi autori fără contribuţie explicită în lista de autori	
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7.	Preluarea identică de pasaje (piese de creaţie) dintr-o operă autentică publicată, fără precizarea întinderii şi menţionarea provenienţei, fără nici o intervenţie personală care să justifice exemplificarea sau critica prin aportul creator al autorului care preia şi însuşirea acestora într-o lucrare ulterioară celei autentice.	✓
8.	Preluarea identică de figuri sau reprezentări grafice (piese de creaţie de tip grafic) dintr-o operă autentică publicată, fără menţionarea provenienţei, fără nici o intervenţie care să justifice exemplificarea sau critica prin aportul creator al autorului care preia şi însuşirea acestora într-o lucrare ulterioară celei autentice.	
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10.	Preluarea identică a unor fragmente de demonstraţie sau de deducere a unor relaţii matematice care nu se justifică în regăsirea unei relaţii matematice finale necesare aplicării efective dintr-o operă autentică publicată, fără menţionarea provenienţei, fără nici o intervenţie care să justifice exemplificarea sau critica prin aportul creator al autorului care preia şi însuşirea acestora într-o lucrare ulterioară celei autentice.	
11.	Preluarea identică a textului (piese de creaţie de tip text) unei lucrări publicate anterior sau simultan, cu acelaşi titlu sau cu titlu similar, de un acelaşi autor / un acelaşi grup de autori în publicaţii sau edituri diferite.	
12.	Preluarea identică de pasaje (piese de creaţie de tip text) ale unui cuvânt înainte sau ale unei prefeţe care se referă la două opere, diferite, publicate în două momente diferite de timp.	

Notă:

a) Prin „provenienţă" se înţelege informaţia din care se pot identifica cel puţin numele autorului / autorilor, titlul operei, anul apariţiei.

b) Plagiatul este definit prin textul legii[1].

> *„ …plagiatul – expunerea într-o operă scrisă sau o comunicare orală, inclusiv în format electronic, a unor texte, idei, demonstraţii, date, ipoteze, teorii, rezultate ori metode ştiinţifice extrase din opere scrise, inclusiv în format electronic, ale altor autori, fără a menţiona acest lucru şi fără a face trimitere la operele originale…".*

Tehnic, plagiatul are la bază conceptul de **piesă de creaţie** care[2]:

> *„…este un element de comunicare prezentat în formă scrisă, ca text, imagine sau combinat, care posedă un subiect, o organizare sau o construcţie logică şi de argumentare care presupune nişte premise, un raţionament şi o concluzie. Piesa de creaţie presupune în mod necesar o formă de exprimare specifică unei persoane. Piesa de creaţie se poate asocia cu întreaga operă autentică sau cu o parte a acesteia…"*

cu care se poate face identificarea operei plagiate sau suspicionate de plagiat[3]:

> *„…O operă de creaţie se găseşte în poziţia de operă plagiată sau operă suspicionată de plagiat în raport cu o altă operă considerată autentică dacă:*
> *i) Cele două opere tratează acelaşi subiect sau subiecte înrudite.*
> *ii) Opera autentică a fost făcută publică anterior operei suspicionate.*
> *iii) Cele două opere conţin piese de creaţie identificabile comune care posedă, fiecare în parte, un subiect şi o formă de prezentare bine definită.*
> *iv) Pentru piesele de creaţie comune, adică prezente în opera autentică şi în opera suspicionată, nu există o menţionare explicită a provenienţei. Menţionarea provenienţei se face printr-o citare care permite identificarea piesei de creaţie preluate din opera autentică.*
> *v) Simpla menţionare a titlului unei opere autentice într-un capitol de bibliografie sau similar acestuia fără delimitarea întinderii preluării nu este de natură să evite punerea în discuţie a suspiciunii de plagiat.*
> *vi) Piesele de creaţie preluate din opera autentică se utilizează la construcţii realizate prin juxtapunere fără ca acestea să fie tratate de autorul operei suspicionate prin poziţia sa explicită.*
> *vii) In opera suspicionată se identifică un fir sau mai multe fire logice de argumentare şi tratare care leagă aceleaşi premise cu aceleaşi concluzii ca în opera autentică…"*

[1] Legea nr. 206/2004 privind buna conduită în cercetarea ştiinţifică, dezvoltarea tehnologică şi inovare, publicată în Monitorul Oficial al României, Partea I, nr. 505 din 4 iunie 2004
[2] ISOC, D. *Ghid de acţiune împotriva plagiatului: bună-conduită, prevenire, combatere*. Cluj-Napoca: Ecou Transilvan, 2012.
[3] ISOC, D. *Prevenitor de plagiat*. Cluj-Napoca: Ecou Transilvan, 2014.

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Smith's General Urology

thirteenth edition

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Urinary Stones

16

J. Patrick Spirnak, MD, & Martin I. Resnick, MD

Archeologic studies show that urinary tract stone disease was an affliction of humans earlier than 4800 BC (Shattock, 1905). Ancient Greek and Roman physicians recorded the symptoms and treatment of urologic stone disease, but little attention was directed to localization of the stone or to the cause of its formation. For a complete review of the historical aspects of urinary stone disease, see Resnick and Boyce (1979).

In the 20th century, advances in technology and microscopic techniques have led to a better understanding of the structural characteristics of calculi, their chemical composition, and the various components of urine. Many theories have been proposed to explain the cause and development of urologic calculi, but none have been able to answer fully the questions concerning stone formation. In all probability, stone disease will be found to result from the interaction of multiple factors, many of which are as yet unknown.

Theories of Stone Formation

A. Nucleation Theory: Stone formation is initiated by the presence of a crystal or foreign body in urine supersaturated with a crystallizing salt that favors growth of a crystal lattice.

B. Stone Matrix Theory: An organic matrix of serum and urinary proteins (albumin; α_1- and α_2-globulins and occasionally γ-globulins; mucoproteins; glycosaminoglycans and matrix substance A) provides a framework for deposition of crystals. These substances are present in urine and are incorporated into the matrix material.

C. Inhibitor of Crystallization Theory: Some urinary substances, eg, magnesium, pyrophosphate, citrate, phosphocitrate, mucoproteins, RNA, glycosaminoglycans, and various peptides, inhibit crystal formation. Absence or low concentration of inhibitors permits crystallization. Synthetic inhibitors (eg, diphosphates) have been developed but they have not been shown to influence stone formation.

Most investigators acknowledge that these 3 theories describe the 3 basic factors influencing urinary stone formation. It is likely that more than one factor operates in causing stone disease. A generalized model of stone formation combining these 3 basic theories has been proposed. After crystal nucleation, a period of abnormal crystalluria is required during which large crystals or aggregates of crystals are produced in the urine. In order for these crystals to continue to grow and propagate, a certain number of chemical factors must be present, ie, the urine must be supersaturated with the salt of the stone-forming crystal, certain inhibitors of crystallization must be reduced or absent from the urine, and a certain concentration of nucleating matrix material must be present.

Additional risk factors can influence the degree and severity of clinical stone disease. These include the metabolic state of the patient, which is influenced by genetic background as well as the presence of certain hormonal imbalances; environmental factors, which could lead to supersaturation of already saturated urine; dietary excesses; and anatomic abnormalities, which could lead to chronic infection or actually enhance the deposition of crystals in the upper urinary tract.

Anatomic Site of Stone Formation

There are several different theories as to where stone formation occurs in the kidney: (1) deposition of calcium on the basement membrane of collecting tubules and on the surface of papillae (*Randall's plaques*); (2) deposition of linear precipitates of calcium within the renal lymphatics to produce obstruction and breakdown of the membrane separating the lymphatics from the collecting tubules (*theory of Carr*); and (3) intratubular deposits of amorphous necrotic calcific cellular debris or organized microcalculi (or both) (*intranephronic calculosis*).

DIAGNOSTIC EVALUATION

Medical History

A personal as well as a family history should be obtained for all patients. A history of inflammatory bowel disease, recurrent urinary tract infection, prolonged periods of immobilization, gout, or familial occurrence of certain inherited renal diseases, eg, renal tubular acidosis or cystinuria, should be sought. Calcium oxalate stone disease is inherited in a multifactorial manner, and hypercalciuria has been shown to be inherited as an autosomal dominant trait. The presence of other endocrine or metabolic disorders should also be considered.

A complete list of all medications taken should be obtained. Acetazolamide, useful in the treatment

Table 16–1. Stone density as related to degree of radiopacity.

	Density	Degree of Radiopacity
Calcium phosphate	22.0	Very opaque
Calcium oxalate	10.8	Opaque
Magnesium ammonium phosphate	4.1	Moderately opaque
Cystine	3.7	Slightly opaque
Uric acid	1.4	Nonopaque
Xanthine	1.4	Nonopaque

acute ureteral colic, the plain film of the abdomen often shows a paralytic ileus that may obscure existing calculi. Plain-film tomograms may help to identify a stone otherwise obscured by overlying gas or feces.

It is not uncommon to see perirenal or periureteral extravasation of contrast medium in patients with obstructing ureteral calculi. The extravasation is believed to originate from a forniceal tear and is associated with the increased pressure caused by the obstructing stones. In the absence of infection, the condition is self-limiting and does not require further therapy. If infection is suspected, antibiotic therapy should be instituted.

3. Retrograde urography–Retrograde urograms are rarely needed to diagnose a stone; however, they are indicated when the diagnosis is suspect or the patient is allergic to contrast medium (Fig 16–4).

4. Ultrasonography–In patients in whom it is not possible to obtain an intravenous urogram, ultrasonic evaluation of the kidneys may aid in the diagnosis of renal stones. In pregnant women with flank pain in whom it is desirable to limit radiation exposure or in anuric patients or patients with chronic renal failure, the presence of hydronephrosis and acoustic shadowing may be diagnostic.

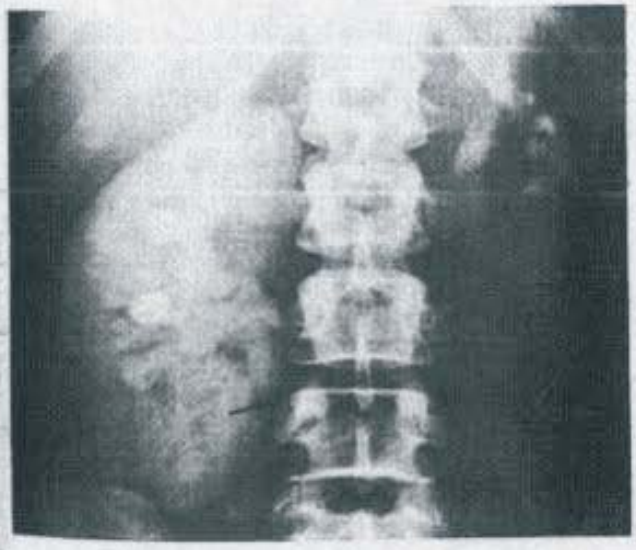

Figure 16–3. Ureteral stone. "Nephrogram" caused by acute ureteral obstruction. Marked density of renal parenchyma with moderate hydronephrosis. Arrow points to nonopaque (uric acid) stone.

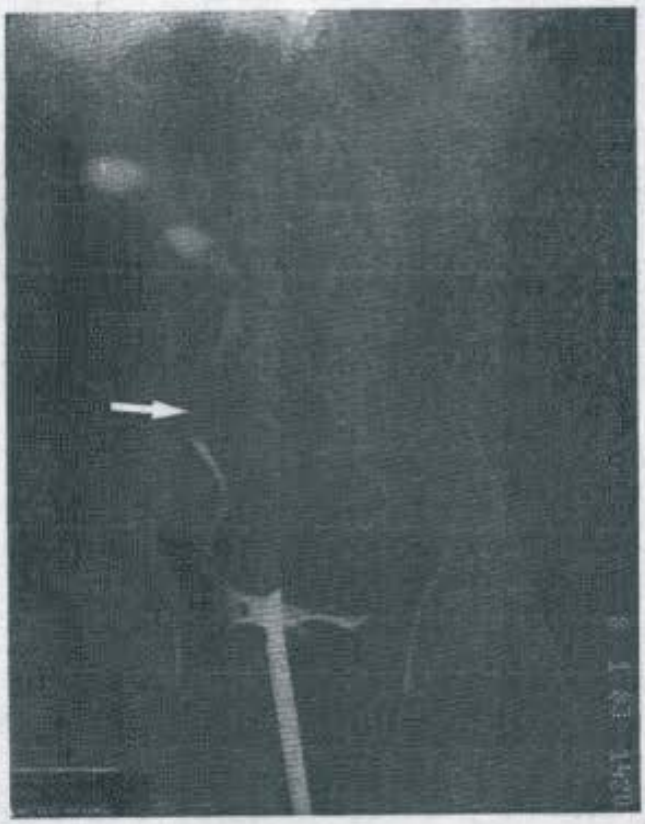

Figure 16–4. Retrograde urogram obtained in patient with kidney that did not visualize on intravenous urogram. Arrow points to a poorly calcified stone in the mid ureter.

5. CT scanning–CT scanning is seldom indicated as the first diagnostic study for the evaluation of a patient with a suspected urinary calculus. However, in cases where the presence of a nonopaque stone or a urinary tract tumor is being considered, CT scans have proved diagnostic.

Although a radiolucent stone cannot be detected on the plain film alone, the diagnosis should be suspected when hydronephrosis and a radiolucent filling defect are found on sonograms or urograms (Fig 16–5). A CT scan may help to differentiate a stone from a blood clot or tumor.

6. Magnetic resonance imaging (MRI)–MRI is an excellent technique for the visualization of the components of the urinary system, but unlike CT, it cannot be used to visualize stones. Its use in patients with urolithiasis is therefore limited.

CALCIUM STONES

Calcium-containing stones can occur as calcium phosphate or calcium oxalate or, more commonly,

dures can be performed under local anesthesia. Recovery time is shortened, and the patient can usually return to full activity in a short period of time.

Disadvantages include the occasional need for nephrostomy drainage for up to several weeks and the possibility of bleeding secondary to percutaneous stone manipulation. These are new techniques, and long-term effects are still uncertain, as is the success rate compared with that of more conventional surgical methods.

The criteria for percutaneous stone removal are identical to those for open procedures for stone removal. Patients should have complete laboratory studies, and all stones should be identified and located preoperatively. Antimicrobial drugs should be used to treat urinary tract infections before stone manipulation.

The cornerstone of percutaneous manipulative procedures is the accurate intrarenal placement of a percutaneous nephrostomy tube and the establishment of a nephrostomy tube tract of adequate caliber to accommodate the nephroscope. Intrarenal access may be achieved by either the antegrade or retrograde technique. Immediate dilation of the nephrostomy tract and delayed dilation of the tract over a 1- to 2-week period have both been successfully employed, but the former technique is used most often.

The methods of stone removal are varied, and the choice is based on the experience of the surgeon and the needs of the patient. Stones can be grasped or flushed out under fluoroscopic control or under direct vision using a nephroscope. Stone baskets or specially designed forceps may be employed. Large stones may be fragmented using either an ultrasonic, laser, or electrohydraulic lithotrite under direct vision.

Before ESWL was widely available, percutaneous stone removal was the treatment of choice for nearly all surgical stones. Complications and long-term morbidity have been minimal with this technique.

EXTRACORPOREAL SHOCK WAVE LITHOTRIPSY (ESWL) (See Chapter 17)

Extracorporeal shock-wave lithotripsy permits removal of renal stones without direct surgical intervention (Chaussy, 1981; Chaussy, Brendel, and Schmidt, 1980). The patient is given an epidural local or general anesthetic and lowered into a tank of distilled water at the bottom of which is placed the shock wave electrode used to produce the shock waves that fragment the renal stone. The shock waves produced by the electrode are focused and directed at the stone by a 2-dimensional radiographic scanning system and are keyed to follow the R wave of the patient's ECG. The average patient receives 1000–1500 shock wave pulses. After about 200 pulses, the stone begins to fragment. Small particles are passed in the urine over the next several days. Newer lithotriptors have eliminated the need for a water bath, can be used without anesthesia, utilize ultrasound to localize the stone, and generate shock waves by a variety of mechanisms.

In studies performed on dogs, the shock waves caused no tissue damage except to the lungs, but the dosage was 50 times greater than that used on humans. The shock waves did not damage bone tissue, because of the large protein matrix of bone.

This technique is being successfully used to treat nearly all renal calculi. Side effects are minimal. Early reports suggesting hypertension as a possible side effect have not been substantiated. Contraindications to the procedure include urinary tract obstruction and active urinary tract infection. Staghorn calculi may be managed using a combination of percutaneous and ESWL techniques. However, multiple procedures may be required. Patients with associated infundibular stenosis require surgical reconstruction in addition to stone removal and are not good candidates for ESWL therapy. It has also been recognized that calcium oxalate monohydrate and cystine stones do not fragment well with this technique.

TREATMENT OF URETERAL STONES

Ureteral stones originate in the renal collecting system and pass into the ureter, where they frequently become lodged and cause symptoms of ureteral colic (Fig 16–12). The right and left ureters are involved with equal frequency. Management depends on the size and location of the stone, age of the patient, presence or absence of urinary tract infection, anatomy of the urinary tract, and degree of symptoms. Treatment may be expectant, manipulative, or surgical.

Studies have shown that 31–93% of ureteral stones pass spontaneously. Size and location of the stone need to be considered when planning a course of therapy. Ninety percent of stones located in the distal ureter and measuring less than 4 mm in diameter were found to pass spontaneously, whereas only 50% of stones 4–5.9 mm in diameter passed spontaneously. Only 20% of stones greater than 6 mm in diameter passed without surgical intervention. Stones located in the proximal ureter are much less likely to pass spontaneously.

Expectant Therapy

Most ureteral stones are less than 5 mm in diameter and pass spontaneously. Expectant management consists of hydration and the liberal use of analgesics. Patients are instructed to strain all urine and to save the stone for analysis. Plain films of the abdomen and pelvis are obtained at 1- to 2-week intervals to monitor progress of the stone down the ureter. If the patient develops fever associated with a urinary