The place and the role of histological examination in diagnostic algorithm of urinary system tuberculosis

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Abstracts

The diagnosis of tuberculosis of renal and urinary tract is made by identifying Koch bacillus in special cultures and using histological examination of surgical removed pieces. Not in all cases the usual pathological techniques are very specific. Using special stain for acid-fast bacilli it can be certified the etiological diagnostic. Histological changes of renal parenchyma and/or upper urinary tract (renal pelvis and urether) structures in 57 patients clinical and paraclinical pyonephrosis diagnosed where studied. All the surgical removed pieces were studied using usual pathology methods. In order to find renal tuberculosis we performed on surgical pieces special staining (Ziehl-Nielsen), we noted the pathological finding in each case and we found 7 cases with certainly tuberculosis etiology.

Keywords: pathologic examination, tuberculosis, urinary tract.

☐ Introduction

Urogenital tuberculosis represents localization of Koch bacillus at urogenital system. Our study is done only to renal and urinary tract localization of tuberculosis. Urinary tract localization of tuberculosis is never primitive and always secondary to a digestive or pulmonary gate. From entrance gate, dissemination is done by lymphatic and/or hematogenic way, in two anatomic-clinical stages: parenchymal stage (closed period) and ulcer caseum stage (open stage). In first stage the patient can be without specific symptoms or only with sterile pyuria. In ulcer-caseum period the tuberculous lesions affects all the segments of urinary tract, initial bilateral involving both kidney, and after that with asymmetric evolution. The certitude of diagnostic is made only by histological examination of surgical pieces removed from the patients.

→ Material and methods

In order to conduct our study, the surgical pieces, removed from patients hospitalized in Urology Department Turnu Severin District Hospital, were processed by usual techniques of pathology. The biological materials were fixed in 10% neuter formaldehyde for 24 hours at the lab temperature. Then, the fixed fragments were processed by the classic histologic technique of wax embedding. We performed 3-5µm sections using wax microtome. The sections were colored with Haematoxylin-Eosine and Ziehl-Nielsen. Ziehl-Nielsen technique was used to identify acid fast bacillus stain (*Mycobacterium tuberculosis*).

☐ Results and discussions

We followed the change of renal parenchyma, renal pelvis and ureter in patients with pyonephrosis. We studied the surgical pieces of 57 patients with clinical

evidence of pyonephrosis, between 1996-2005. From all the cases, we found certainly tuberculosis etiology in 7 cases. The microscopic aspect of patients with tuberculosis infection is various from a case to another and also from various area of urinary tract. This is due to the different evolution stage of disease and because the patients have no the same immunological profile. Morphologic lesions have not the same intensity all over the urinary tract.

We found morphological changes firstly at cortical area of renal parenchyma. Latter appeared granuloma in medullar area. Initial, Koch bacillus is localized at glomerulus. A good influx of the blood in afferent arteriole and a high blood viscosity of efferent arteriole favor this first place of infection. Mesangium cells elaborate lymphokine and induce macrophage infiltration and formation of cortical granuloma. After this, infection is propagated at convolute proximal tube and Henle ansae and medullar granulomas are formed. Epiteloid cells from granuloma induce the fibroblastic proliferation and secretion of tumor necrosis factor. In this way appear tuberculoma and caseum necrosis that replace the collector duct and cavernas are formed (Figure 1). In this moment it can be identify Koch bacillus in urine.

A central zone of caseum necrosis, eosinophilic and no structured, with necrotic detritus, surround by a row of epitheloid macrophages and few giant cells, characterizes the caseating granuloma, in the classic situation (Figure 2).

Necrotic area is amorphous pink caseating material composed of the necrotic elements of the granuloma as well as the infectious organisms (Figure 3). Granuloma demonstrates that the epithelioid macrophages are elongated with long, pale nuclei and pink cytoplasm.

The macrophages organize into committees called giant cells (Figure 4). The typical giant cell for infectious granulomas is called a Langhans giant cell and has the nuclei lined up along one edge of the cell.

Necrotic area is ringed by the inflammatory component with epithelioid cells, lymphocytes, and fibroblasts. We found this typical aspect in 3 cases.

In 1 case we found few neutrophils inside necrosis area that has no specificity for tuberculosis. In 3 cases the necrosis were absent and only epitheloid, noncaseating, granulomas offered us the clue for etiology.

We performed in all cases Ziehl-Nielsen coloration for certitude of tuberculosis infection and we found the evidence of acid-fast bacilli stain in all cases (Figure 5).

For certify the etiological diagnostic of pyonephrosis is also very helpfully PCR but we cannot perform this investigation in our lab. Even in absence of PCR, Ziehl-Nielsen staining has a great diagnostic value.

Another type of renal lesions produced due to tuberculosis, but not by tuberculosis, is represented by amyloid deposition. Deposition of amyloid in renal tissue is due to the increase of inflammatory cell number and decrease of the defense capacity of the patient.

It is not specify for tuberculosis, renal amyloid deposits appear in any long standing chronic infection or in neoplasms (e.g. renal cell carcinoma). We found this aspect in only 1 case, where also granulomatous infection was present.

On H.E. staining we identify a widespread glomerular deposition of moderately eosinophilic homogeneous material and Red Congo stain shows positive results for amyloid deposits in glomeruli and vessels.

The clinical expression in this case was nephrotic syndrome and renal failure. Much type of glomerulophitis is identifying to be secondary renal tuberculosis but in our study we did not found anyone.

The same type of lesions where found in a more advanced stage to renal pelvis and ureter (Figure 6).

The intensification of fibroblast metabolism, increasing of some components of extra cell matrix (collagen and glicosaminoglicans) synthesis together with an associated good host response causes the appearance of a scar tissue.

This is a no specific pathological image due to proliferation of fibrocyte-fibroblast complex in order to repair lesions produced by pathogenic agents.

₽ Conclusions

- 1. Pyonephrosis represent a seriously complication of urinary tract infection with severe destruction of renal parenchyma.
- 2. The certitude of urinary tuberculosis diagnostic is made by histological examination of surgical removed pieces.
- 3. Tuberculosis of urinary tract can be treat by surgical and medical methods with morphological functional cure and social integration.

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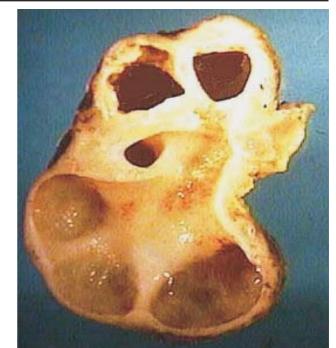


Figure 1 – Macroscopic image of renal parenchyma with cavernas

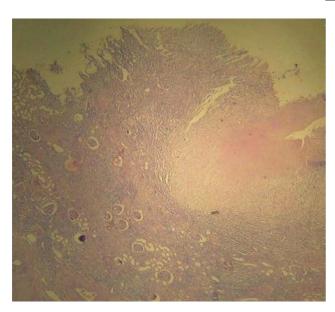
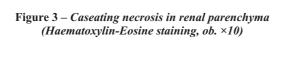


Figure 2 – Caseating granuloma in renal parenchyma (Haematoxylin-Eosine staining, ob. ×10)





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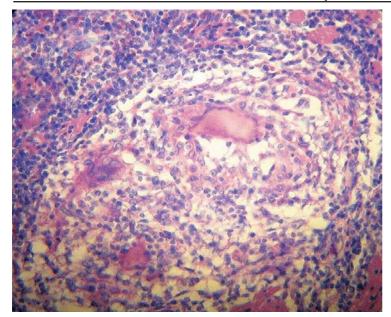


Figure 4 – Giant cells in granuloma in renal parenchyma, without necrosis (Haematoxylin-Eosine staining, ob. ×40)

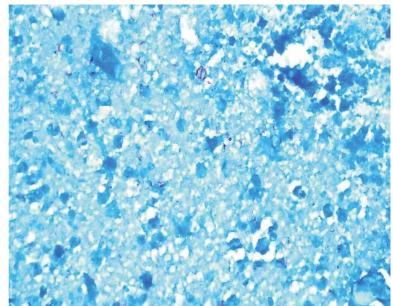


Figure 5 – Ziehl-Nielsen staining that shows acid fast bacilli (ob. ×40)

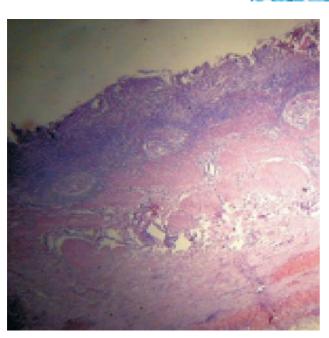


Figure 6 – Tuberculous granuloma in the wall of ureter (Haematoxylin-Eosine staining, ob. ×10)