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Diagnostic difficulties in cystic renal tumors

Trudności w diagnozowaniu guzów torbielowatych nerki

The kidneys are the organ in which different types of cystic changes develop most often (11). There is emphasized diversity of forms and divisions based on genetic, congenital or acquired etiology (7). Cystic tumors pose diagnostic difficulties in the assessment of the character of complex cystic mass (5).

The study aimed at determining the value of used imaging techniques in differentiating complex cystic masses making up the so-called undetermined renal masses.

MATERIAL AND METHODS

The material comprised 96 malignant tumors and 23 benign renal tumors, i.e. jointly 119 patients aged 13-86 years (73 men, 46 women). They were treated in the years 1992-1999 in the District Specialist Hospital, District Hospital No 2 in Rzeszów and in the Departments of Medical University within the Independent Public Teaching Hospital No 1 in Lublin. Examinations were performed in the X-ray Departments of these hospitals.

The patients were subjected to total nephrectomy with subsequent histopathological examinations. Thin-needle aspiration biopsy under ultra-sound control was also performed, especially in doubtful and unclear cases.

The group of malignant tumors consisted of bright cell cancers - 88 cases, malignant nephromas, neoplastic metastases to the kidney, undifferentiated cancers and from the cells of transitional epithelium - 2 cases each. The group of benign tumors comprised

complex cysts: hemorrhagic and inflammatory - 6 cases each, 4 calcified cysts, 2 parasitic, 5 cases of renal nephrocystosis.

The stage of tumor advancement was determined according to Robson's classification taking into account modem imaging techniques and specific in the assessment of renal tumors (11). In the assessment of the stage a significant role was also played by TNM classification in its IV version binding from January 1987. Robson's stage was correlated with operative findings. In all the patients urography, USG and CT examinations were done. In 18 cases angiography of renal arteries was performed and in 11 cases MRJ examination was carried out.

RESULTS

In 3 cases arc UKM modelling elongated separated calyces resembling spider's legs caused nephrocystosis (Fig. 1 a,b,c). Urosonography in 2 cases of nephrocystosis revealed spheric areas of decreased density. In 2 cases they got revealed in a late nephrographic stage. In 4 cases heterogenic cystic mass showed irregular calcifications of the wall with its doubtful enhancement. In 7 cases calcifications had peripheral character with circinate appearance (Fig. 2).

In 2 cases post-inflammatory cysts revealed in CT pictures thin calcifications of the partitions which did not get enhanced. In 3 cases of post hemorrhagic cysts the presence of thick irregular wall calcifications was shown.

Peripheral calcifications in the walls and partitions in 6 cysts were characterized by high echogenicity. In 3 cysts with the density about 15 H.u. linear calcifications were detected in the partitions and thin regular walls, which did not get enhanced.

Cystic cancers showed 16 times a slight solid component. Internal surfaces formed the structure resembling a honey plaster. USG examination showed the irregularities of the contour of thick walls and partitions. Distinct vascularisation and contrast enhancement of the walls was found in 8 cases of cystic cancers. A sixfold thickening of the wall had segmental character.

In 7 cases multicystic mass showed thick walls and partitions getting enhanced. 4 times a multi-chamber cyst revealed differentiated density with considerable enhancement of thick walls and partitions. In 8 cases the solid component of cystic tumors showed intensive contrast enhancement in CT examination. In 5 cases of polycavernous mass the presence of bright cell, multi-chamber cancer was confirmed (Fig. 3).

Spherical masses with increased density were found in 4 cases of infected cysts showing considerable enhancement of thickened walls. A ring of intensively enhanced parenchyma was shown in 2 cysts of parasitic origin (Fig. 4).

In 2 cases short term hemorrhagic cysts with high density (40-100 H.u.) corresponded with bleeding at the lack of contrast enhancement of the mass interior. In 3 cases of hemorrhagic cysts the central part showed small density. Complexity of the cyst structure

resulted from evolution of hemorrhagic changes. A similar structure was shown in 7 cases by cystic cancer. In diagnostically difficult cysts punctures under the control of CT and USG were performed. Masses with the density of 15 H.u. were assessed as undetermined, especially in combination with untypical CT symptoms. In 6 cases of complex cysts density coefficient after the administration of contrast increased by about 15 H.u. on 5 mm thick CT sections on which tumors of 0.5 diameter were distinctly revealed. In 7 cases of tumors with heterogenous structure a hypodensic area in the central part corresponded with necrotic cyst. Disintegration areas were found jointly in 14 malignant tumors.

In 16 cases undefined masses were constituted by intrarenal cysts with irregular contours and density of 25-30 H.u. In 3 cases contrast CT sections revealed well limited mass of 20 H.u. density with the presence of wall tumors not getting enhanced. A thick fringe hindered the exclusion of cystic tumor. Thickened walls and septa with irregular contours were found in 6 cases of complex cysts and in 4 inflammatory cases – once.

Complicated cysts usually exceeded 3 cm in diameter, showed dense wall septa of non-uniform thickness 2.5 mm and clear separation from surrounding parenchyma. There was lack of nodular component of the septa. In 3 cases of nephrocystosis intrarenal masses of sacculated cysts showed thick walls and septa with blood effusions in different evolution stages.

In 3 cases vascular nephrogram showed a significant network of fields without blood supply (Fig. 5). In 3 cases nodular masses (adenocarcinoma) was revealed in the kidney with cystoid degeneration (Fig. 6 b,c). Heterogenous contrast enhancement was especially characteristic of bigger tumors (Fig. 7).

In 2 abscesses wall thickening had irregular contours and necrotic center with small density. A zone of small parenchymal enhancement surrounding fresh abscesses corresponded with inflammatory congestion. Older abscesses had in 2 cases smooth contours and thick enhancing walls (Fig. 8). In 11 cases supplementary data were provided by MRI examination. It was performed in SE and FSE sequences in Ti and T2 dependent pictures in transverse planes before and after administration of 15 ml magnevist. In 8 patients MRI revealed the presence of liquid spaces within the tumor (Fig. 9).

DISCUSSION

A smooth wall, non-vascularised cyst can be a cystoid cancer (7). The detection of solid or nodular component in the cyst usually corresponds to cancer texture. Pathologic vascularisation only in half of cystoid cancers suggests malignancy (14). Diversified USG and CT picture forms, depending on the degree of necrosis, cystoid-solid mass.

Cystoid character not meeting CT criteria of a simple cyst was observed in 22% cancers (15). These were most often (39%) tumors of over 8 cm diameter (is). In other reports cystoid form was noted in 5-7% of renal cancers (10), 5-15% (14). The incidence of cancer in the cystic wall amounts to 18% (12). In complex cysts in 41% cases malig-

nant changes were found (2). Before USG-CT era the incidence of cysts co-existing with cancer amounted to 2.3-7% (3). The tumor can arise from cystic wall or co-exist with multicystoic degeneration. Undefined renal masses, however, have usually benign character (11). They are characterized by absorption co-efficient of over 20 H.u. and uncertain contrast enhancement (11). Both cysts and malignant tumors can have density co-efficient close to that of renal parenchyma which cannot be differentiated without the use of contrast.

Diagnostic difficulties are caused by complex cystoid masses with various wall pictures and content. This is connected with the presence of calcifications, hemorrhagic, suppurative content, septa and ward-like forms (5). Diagnostic problem is caused by high density cysts (hemorrhagic, infectious with calcium milk, with high protein content). Only 50% of hypodensic cysts meets the criterion of a simple cyst. After enhancement cysts can obtain the density of solid mass and show solid character in USG examination. Untypical or complicated cysts show dorsal enhancement at the lack or echoless inside (9).

Even slightly complex cystic mass can contain malignant cells. Numerous simple cysts present a possibility of cystoid cancer in one of them (7). Cystoid cancer can result from the necrosis of the cyst or from internal development when neoplastic cells of the endothelium line cyst walls or septa (cystoid-adenocarcinoma).

About 50% cancers, most often ward-like adenocarcinomas are not vascularised and angiographically are difficult to differentiate from cysts. Adenocarcinomas constituting about 90% tumors are characterized by mixed echo structures non-uniform enhancement and necrosis (11).

Bosniak classifies cysts into 4 categories (2, 12). I - classical simple cysts; II - slightly complex, homogenous, hyperdensic cysts among which he distinguished 3 subgroups: 1. cysts with delicate calcification of walls; 2. with small amount of internal septa; 3. hyperdensic with homogenous high density not getting enhanced. Category II is considered benign, however, requiring USG control after 3, 6 and 12 months. III - homogenously thickened nodular walls and septa getting enhanced, non-uniformly thickened thick wall irregular calcifications. 5% cysts from this category were malignant. Most cysts from category II-III with contrast enhancement below 10 H.u. giving picture of undefined mass were malignant. IV - thick, irregular walls of nodular character getting enhanced and solid elements defined malignant character.

There is emphasized the value of Bosniak's classifications in diagnosing cystoid masses of the kidney (4). In other reports the assessment of its usefulness is controversial (2).

Cystoid tumors produce intra-tumor necrosis, most often central, which forms the picture of untypical cysts in USG (9, 11, 15). It was found in 70% of cancers, especially light cell ones of the kidney (13, 15). The importance of internal echo structure and of acoustic enhancement has been emphasized (9).

In complicated cysts small cystic fields can result from necrosis of all fibroses, bleedings, calcifications or they constitute a cystoid component of the tumor. Nodules overlooked in USG can be visible in CT.

Central calcifications within the cystoid mass, usually in the septa produce a probability of malignant changes. Peripheral ones of arcuate character were found both in malignant tumors and hemorrhagic and echinonococcal cysts. In atypical cysts calcifications are tiny laminated in the walls and septa. Peripheral calcifications or those in the septa of cystoid mass which are thick and irregular occur in 20% cancers (1).In cysts they are four times more frequent than in cancers, diffuse ones suggest malignancy but similar can occur in hemorrhagic cysts (7). High density cysts are difficult to assess due to the enhancement caused by the effect of partial volume artificially increasing the coefficient of cyst weakening.

There are distinguished 4 types of histopathologic growth of cystoid cancer (8). 1 - mono-chamber, cystoid adenocarcinoma; 2 - multi-chamber (a) hypodensic, (b) hyperechogenic which in USG and CT can have solid appearance due to the interference (overlapping) of numerous surfaces and septa; 3 - cystoid necrosis (pseudocysts); 4 - formation of endothelium in previously existing simple cysts (12).

In thickened septa of multi-chamber renal cysts, in about 10-25% cases the texture of light cell cancer was revealed (13). In the cyst inter-chamber septa contain only fibrous tissue which is identified by Doppler USG determining blood flow. Irregular septa or ward-like formations penetrating into the cyst lumen account for malignancy. In necrotic, cystoid tumors and abscesses the thickness of the wall exceeds 2 mm and when it is over 3 mm cancer can be suspected (15). In the cysts of renal surface the wall thickness is below 2 mm with possible symptom of claw or beak (7). In cystoid cancers it can be as big as 10-40 mm (15). Besides septum thickness irregularity of its contours is also essential (2). Thickening of the wall, irregularity of contours, solid elements within, wall nodules, ward-like formations enhanced by contrast can suggest malignancy (2, *5,* 6, 12, 14).

Poorly vascularised ward-like cancer not showing enhancement produces falsely negative diagnosis. Cancer can be a flat tumor in the cyst wall or in multi-chamber cystoid mass (2, 6). Cancer growth in the wall of a simple cyst is considered a rarity (9). Cystoid multi-chamber nephroma seems the most accurate name for a multi-chamber intrarenal cyst forming a complex mass (11). Fibrous-vascular stroma enhances the septa which do not contain parenchyma. This, as a rule, characterizes cystoid tumors *(5).*

In the septa malignant cells were found forming Wilms' tumor (6). The effect of partial volume decreases the density of the septa, especially on 10 mm thick sections. This also hinders the assessment of atypical fluid in small cyst. Angiography shows a small solid component poorly vascularised with tiny pathologic vessels (8).

It is difficult to differentiate small, solid tumors and renal cysts (11). The assessment of tumors with the diameter below 3 cm, often turned as adenomas, when over 3 cm as cancer is often not univocal (11). A survey of cystoid neoplasms was presented by Hartman (8).

Hemorrhagic cysts form in USG and CT time-dependent pictures. Fresh cysts show a greater degree of echogenicity than mature echoless structures or the ones with tiny, diffuse internal echoless areas in consequence of clot formation and tissue residues. Heterogenous USG appearance of bleeding or infection, by increasing cyst density, produces difficulties in differentiating with a solid change (11).The walls of hemorrhagic cysts invisible in the initial stage give thick wall enhancement in aging cysts (5). Both hemorrhagic and infectious cysts can simulate a cystoid neoplasm and then aspiration of the doubtful mass is indicated (6).

Inflammatory cysts and abscesses form in USG hypoechogenic masses with tiny internal reflections. In time the contents of cysts gets more echogenic, density increases, walls get irregularly thickened, they get intensely contrast enhanced. A malignant change has, as a rule, greater density and clearer enhancement than a chronic abscess. On angiography abscesses and poorly vascularised cancers can resemble a cyst with displacement and modelling of arteries (4).

An untypical cyst which may be formed by a an abscess or tumor with central necrosis requires targeting thin-needle biopsy (7). In multi-chamber cysts multiple cystic spaces can decrease the value of puncture (2, 4).

In complex cystoic tumors MRI uses intensity differences of the signals of fluid and parenchyma. Determination of an intensity, however, has limitations since it can be identical in a fluid cyst and cystoid cancer. Despite the superiority of MRI of CT in the assessment of multi-chamber cystoid masses it does not exclude the presence of tumor in a complex cyst. Bleeding does not differentiate cystoid cancer and late bleeding in the cyst (10). Overlapping intensities of signals of a hemorrhagic cysts and the bleeding tumor blur the border of benign character of cystoid mass. In an unclear CT picture differentiating atypical, complex cysts from solid tumors is more certain due to a greater contrast enhancement of MRI.

CONCLUSIONS

1. Differentiation of cystoid atypical cancers from complex cysts is hindered by their common morphologic features.

2. Urography, USG and CT show correlations in the assessment of contours of walls, septa, structures, density and contrast enhancement enabling to assess their character in most cystoid masses.

3. Angiography of renal arteries and MRI supplement the diagnostics of doubtful masses revealing the presence of pathologic vessels and selective densities of fluid reservoirs.

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STRESZCZENIE

W materiale 96 złośliwych guzów torbielowatych i 23 torbieli złożonych, łagodnych nerek analizowano możliwości stosowanych technik obrazowania w określeniu charakteru guzów. Porównanie cech morfologicznych prawidłowych i patologicznych w obrazie różnych technik i w guzach różnej etiologii pozwoliło ustalić określone kryteria rozpoznawcze.

EXPLANATION TO FIGURES

Fig. 1. (a) – urography - deformation of UKM on the left, which is modelled on cystoid changes. Separated and displaced renal calyces. Pathologic nephrogram. (b) - USG in the medial part of the left kidney. Irregular hypoechogenic areas with thin wall septa. Pseudohydronephrosis. (c) - CT in the hilus of the left kidney hypodensic area. UKM gets modelled on the cystoid change.

Fig. 2. CT - hypodensic area of the right kidney pressing and displacing UKM and circinate calcification in the medial part. Ca - *clarocellulare tubulare partim cysticum, calcificans.*

Fig. 3. CT - multi cystoid tumor of left kidney projecting from its dorsal part.

Fig. 4. CT - a cyst of the right kidney of parasitic etiology.

Fig. 5. CT - cyst contours on a vascular nephrogram.

Fig. 6. CT - irregular tissue within cystoidally changed left kidney corresponds to the tumor. This area gets heterogeneously calcified showing calcifications. Numerous cysts of the right kidney.

Fig. 7. CT - giant heterogeneous tumor of the right kidney with areas of different density.

Fig. 8. CT - within a fatty cyst of the right kidney irregular, hypodensic area with intensely enhancing walls and septum (abscess). Displacement of the kidney to the anterior-medial direction with deformation of the dorsal contour. Within UKM hypodensic shadow corresponds to a concrement.

Fig. 9. MRI - in the lower pole of the left kidney from the rear oval tumor mass with heterogeneous signal in T1 and T2 dependent pictures with hyperintense areas in T2 dependent pictures corresponding to fluid spaces.

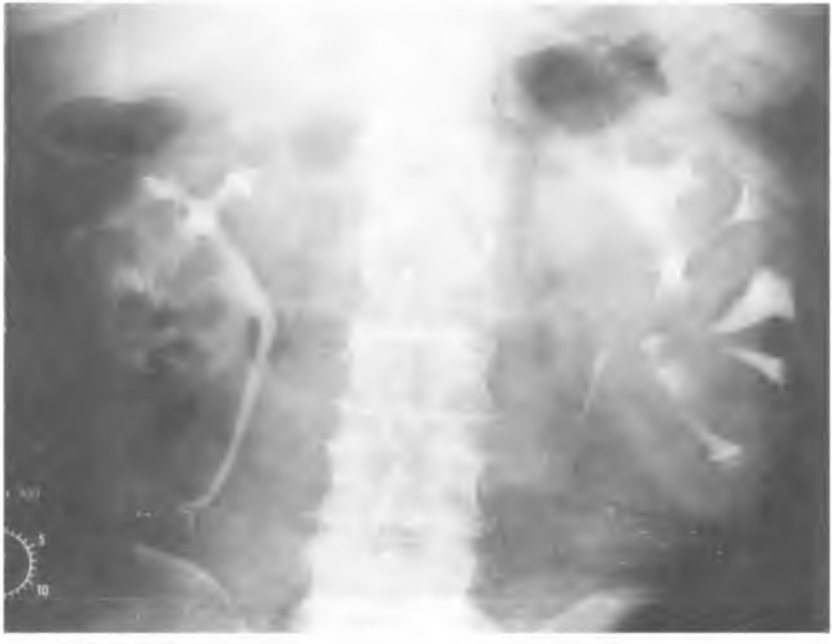


Fig. 1a

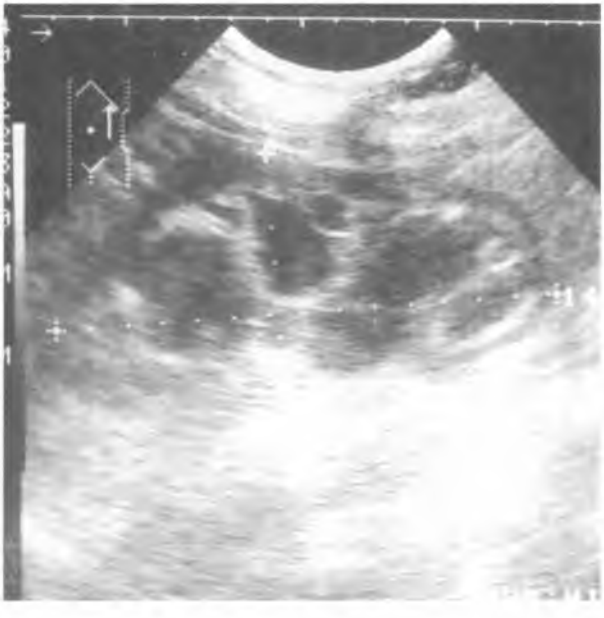


Fig. 1b

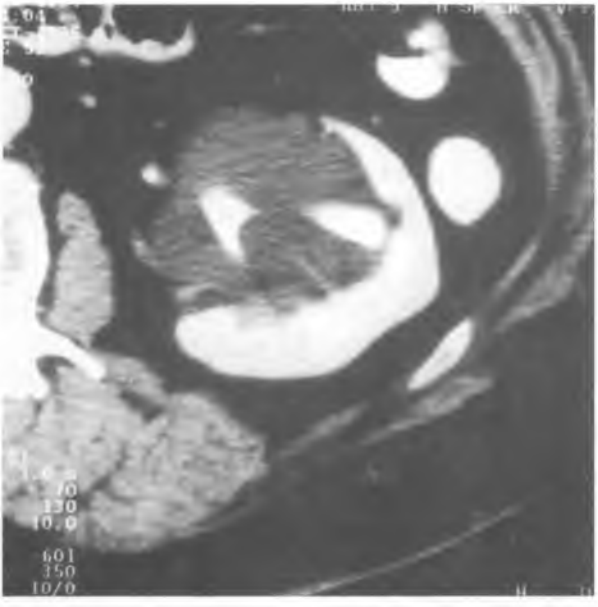


Fig. 1c

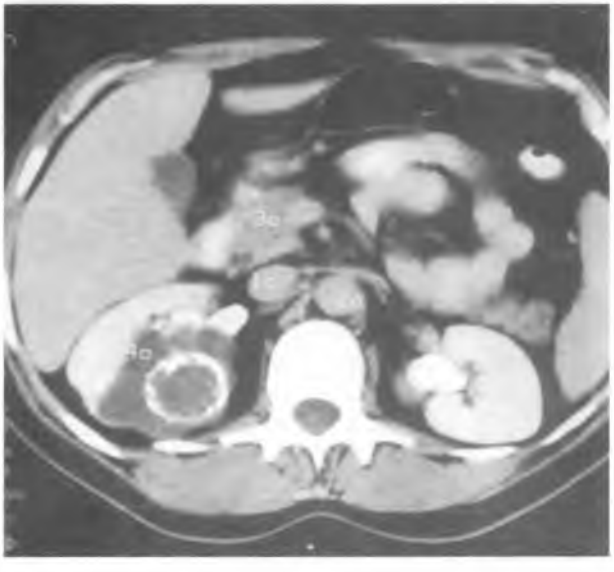
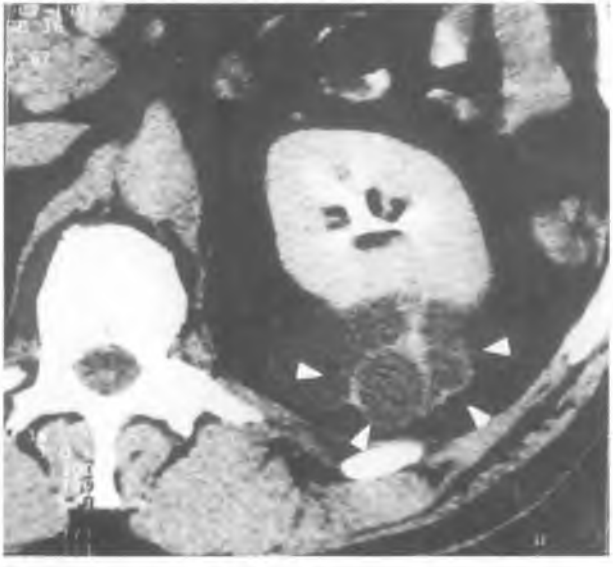


Fig. 2



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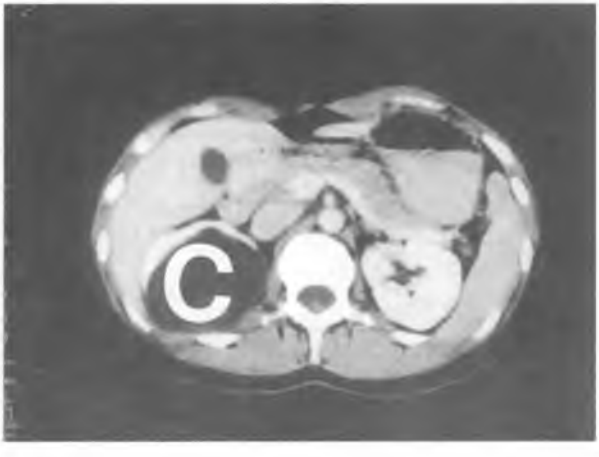

Fig. 4

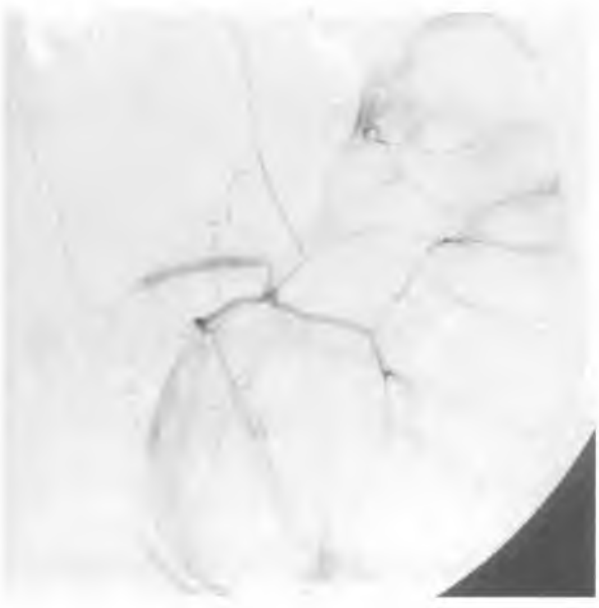

Fig. 5

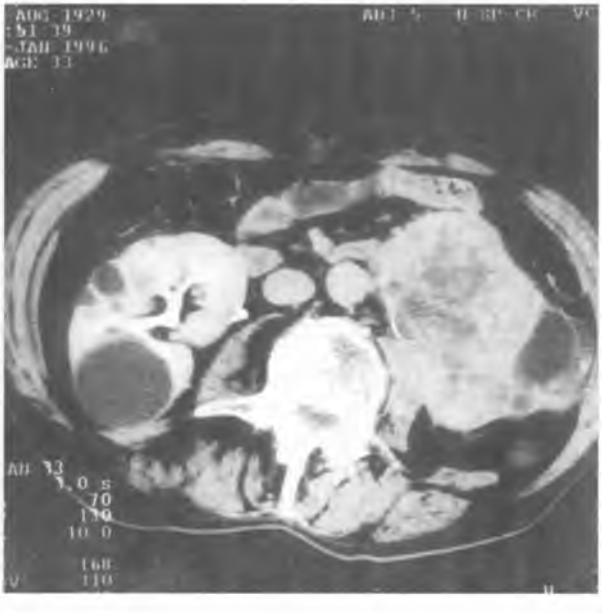

Fig. 6

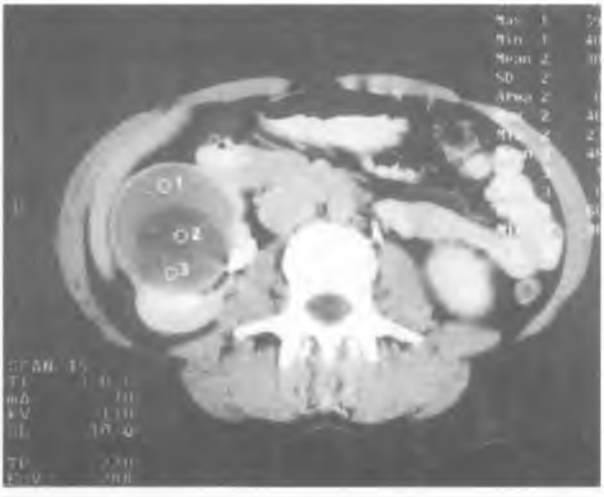

Fig. 7

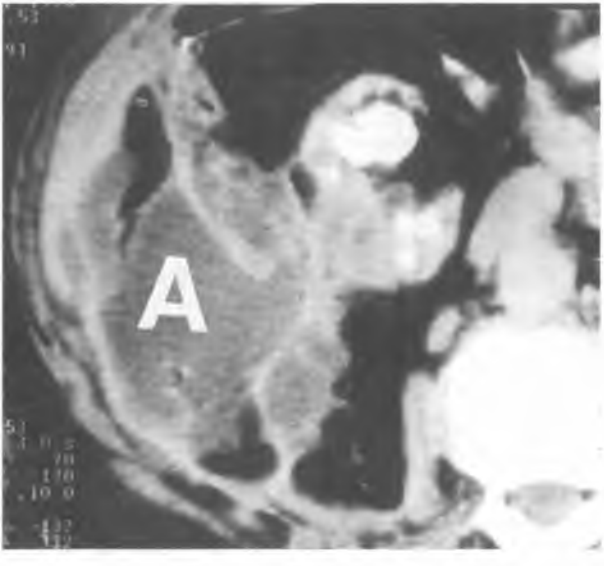

Fig. 8

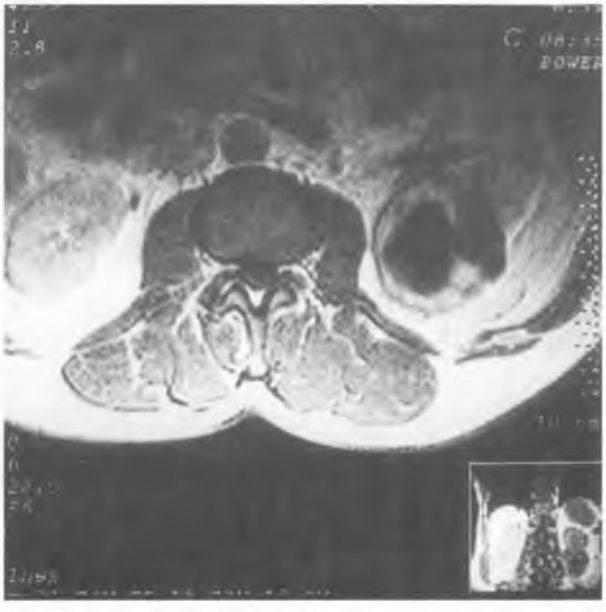

Fig. 9