Obstrução Urinária

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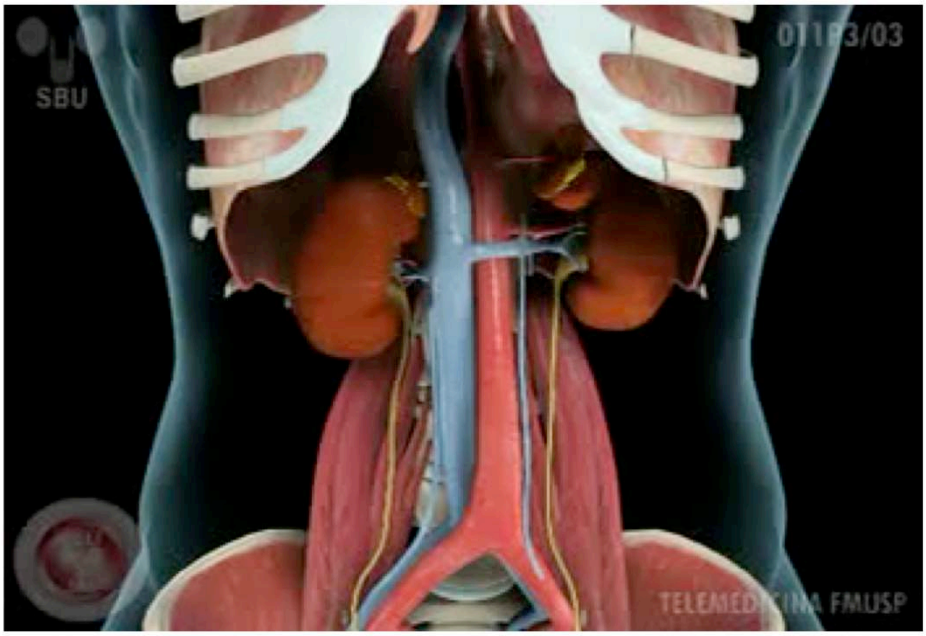
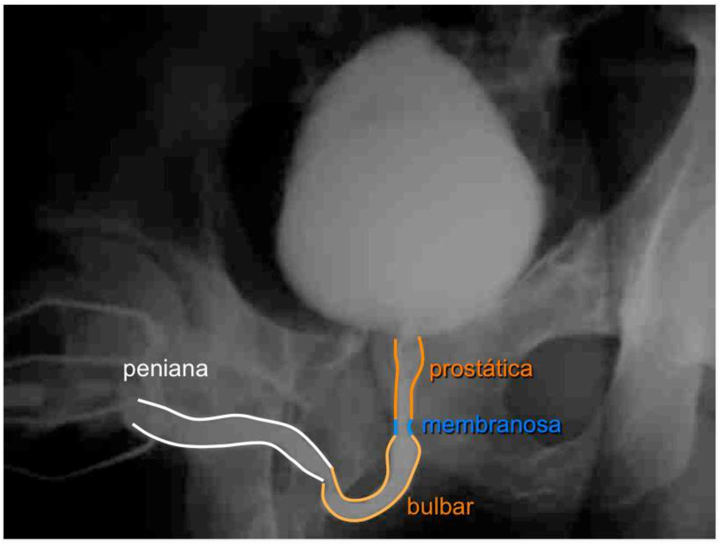
Considerações anatômicas



Visão geral do trato urinário e pontos de obstrução mais frequentes.

Considerações anatômicas



Uretra masculina e suas partes.

Conceitos

Taxa de filtração glomerular = Coeficiente de ultrafiltração (Pressão capilar glomeular − Pressão hidráulica tubular − Pressão oncótica)

área e permeabilidade da membrana

fluxo plasmático e resistência das arteríolas aferentes e eferentes

Fluxo plasmático renal = Pressão aórtica − (resistência vascular renal + pressão venosa renal)

- A constrição da arteríola aferente resulta em decréscimo da pressão capilar glomerular e da taxa de filtração glomerular
- A constrição das arteriolas aferentes ou eferentes ou ambas reduz o fluxo plasmático renal.
- O aumento da resistência da arteriola eferente aumenta a pressão capilar glomerular

A brief discussion of the determinants of glomerular filtration is in order to understand the interrelationships between changes in renal hemodynamics and alterations in the glomerular filtration rate (GFR) during and after obstruction. Factors influencing GFR are expressed in the following equation: GFR=Kf(PGC −PT −πGC) Kf is a glomerular ultrafiltration coefficient related to the surface area and permeability of the capillary membrane. PGC is the glomerular capillary pressure, which is influenced by renal plasma flow and the resistances of the afferent and efferent arterioles. The hydraulic pressure driving fluid into Bowman space is resisted by the hydraulic pressure of fluid in the tubule (PT), and also the increasing oncotic pressure (π) of the proteins remaining at higher concentrations in the late glomerular capillary and efferent arteriolar blood. Although filtered fluid is not completely free of small proteins, for practical purposes, its oncotic pressure is negligible. The net pressure determining glomerular filtration is referred to as the ultrafiltration pressure (PUF) and is derived from (PGC − PT − πGC). PGC is also influenced by renal plasma flow (RPF). RPF depends upon the renal perfusion pressure and intrarenal resistance to flow, the latter primarily mediated by the resistances in the afferent and efferent arterioles. The aforementioned relationships are depicted in the following equation: RPF = aortic pressure − renal venous pressure renal vascular resistance Thus constriction of the afferent or efferent arteriole or both would reduce RPF. Constriction of the afferent arteriole results in a decrease of PGC and GFR, whereas an increase in efferent arteriolar resistance increases PGC. Whole kidney GFR depends upon factors regulating perfusion of each glomerulus and also upon the percentage of glomeruli actually filtering. For each glomerulus, the single-nephron glomerular filtration rate (SNGFR) is determined by the previously mentioned GFR equation. Obstruction can transiently or permanently alter GFR and some or all of the determinants of GFR.

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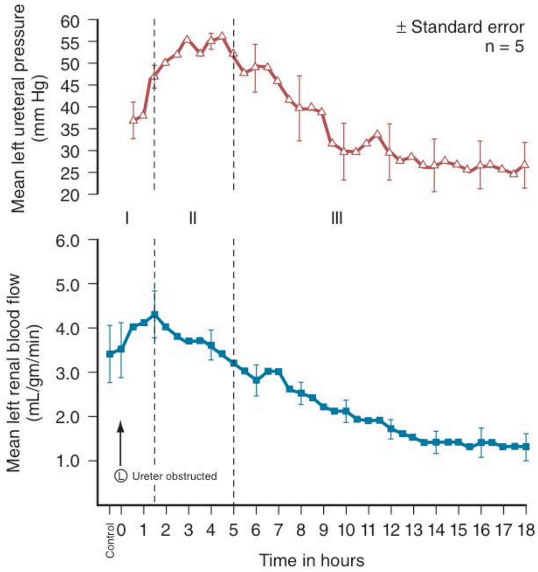
Alterações funcionais na obstrução unilateral

I: aumento da pressão e fluxo (PGE2 / NO)

II: diminuição do fluxo (angiotensina II). Pressão mantem-se elevada ou pode continuuar elevando-se.

III: Diminuição do fluxo (angiotensina II) e da pressão e consequente diminuição da ritmo de filtração glomerular.



Triphasic relationship between ipsilateral renal blood flow and left ureteral pressure during 18 hours of left-sided occlusion. The three phases are designated by roman numerals and separated by vertical dashed lines. In phase I, renal blood flow and ureteral pressure rise together. In phase II, the left renal blood flow begins to decline and ureteral pressure remains elevated and, in fact, continues to rise. In phase III, the left renal blood flow and ureteral pressure decline together. (From Moody TE, Vaughn ED Jr, Gillenwater JY. Relationship between renal blood flow and ureteral pressure during 18 hours of total unilateral ureteral occlusion. Implications for changing sites of increased renal resistance. Invest Urol 1975;13:246–51.) it is likely that both PGE2 and NO contribute to the net renal vasodilation that occurs early following unilateral ureteral obstruction. Reduced whole kidney GFR at this stage of obstruction is due not only to reduced perfusion of individual glomeruli, related to afferent vasoconstriction and reduced PGC, but also to global reduction in filtration related to no perfusion or underperfusion of many glomeruli. Angiotensin II is an important mediator of the preglomerular vasoconstriction occurring during the second and third phases of unilateral ureteral obstruction.

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Obstrução ureteral bilateral

Fase	Tempo	Unilateral		Bilateral ou rim solitário	
		Fluxo sanguíneo renal	Ritmo de flitração glomerular	Fluxo sanguíneo renal	Ritmo de flitração glomerular
Fase aguda	1 a 2 horas	↑	O	↑-↓	↓
Fase intermediária	2 a 5 horas	↓	↓	↓	↓
Fase tardia	24 horas	↓↓	↓↓	↓	↓↓
Pós obstrução	+ que 24 horas	↓	↓↓	↓	↓↓

The changes with BUO or obstruction of a solitary kidney are different. **In contrast to the early robust renal vasodilation with unilateral ureteral obstruction, there is a modest increase in renal blood flow with BUO that lasts approximately 90 minutes, followed by a pro- longed and profound decrease in renal blood flow that is greater than found with unilateral ureteral obstruction** (Gulmi et al, 1995). Ureteral pressure is higher with BUO than with UUO. Although in both cases ureteral and tubular pressures are increased for the first 4 to 5 hours, the ureteral pressure remains elevated for at least 24 hours with BUO, whereas it begins to decline and approaches preocclusion pressures by 24 hours with unilateral ureteral obstruction.

Ureteral pressure remains high because BUO passes through a phase of preglomerular vasodilation and then a prolonged postglomerular vasoconstriction. This explains the persistent elevation in ureteral pressure in spite of a decrease in RBF and increase in renal resistance. In contrast, in unilateral ureteral obstruction the initial preglomerular dilation and short-lived postglomerular vasoconstriction are followed by a more prolonged preglomerular vasoconstriction that tempers elevations in PGC and hence in PT. **This difference between the two pathophysiologic conditions has been hypothesized to be due to an accumulation of vasoactive substances in BUO that could contribute to preglomerular vasodilation and postglomerular vasoconstriction. Such substances would not accumulate in UUO because they would be excreted by the contralateral kidney. Atrial natriuretic peptide (ANP) appears to be one of these substances** (Purkerson et al, 1989)

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Alterações eletrolíticas e metabólicas

- Excreção aumentada de sódio: alteração da regulação pelo peptídeo natriurétrico atrial
- Excreção aumentada de magnésio
- Efeitos na excreção de peptídeos e proteinas:
  - Alguns peptídeos e pequenas proteinas são normalmente filtrados pelo glomérulo e reabsorvidos ao longo do nefron
  - Algumas enzimas e proteinas como as de Tamm-Horsfall e aquaporinas podem ser normalmente secretadas no fluido tubular
  - A obstrução pode exagerar ou romper a excreção dessas proteinas e peptídeos
- Alterações metabólicas : alteração do metabolismo oxidativo para anaeróbico, com redução do nível de ATP renal, do ADP e AMP, e aumento do lactato em relação ao piruvato[1]

Stecker et al, 1971; Middleton et al, 1977; Nito et al, 1978; Klahr et al, 1986

Sodium Transport: Although ANP appears to play a role in sodium diuresis after release of bilateral ureteral obstruction, it is unlikely to affect sodium transport defects associated with unilateral ureteral obstruction.
Hydrogen Ion Transport and Urinary Acidification Obstruction causes a deficit in urinary acidification that has been demonstrated in human subjects as well as animal models.
Magnesium excretion is markedly increased after the release of either UUO or BUO.
Effect of Obstruction on the Excretion of Peptides and Proteins: Some peptides and small proteins are normally filtered by the glomerulus and readily absorbed in the nephron. Some enzymes and proteins, such as Tamm-Horsfall protein and aquaporin may normally be secreted into the tubular fluid. Obstruction can exaggerate or disrupt the excretion of these proteins and peptides. Some changes simply represent alterations in transport, whereas others are due to tubular damage and remodeling.
Metabolic Determinants of Ion Transport Renal obstruction provokes a number of changes in the metabolic cascade. There is a shift from oxidative metabolism to anaerobic respiration. This shift results in a reduction of renal ATP levels, an increase in amounts of adenosine diphosphate (ADP) and adenosine mono- phosphate (AMP), and an increase in the renal lactate- to-pyruvate ratio (Stecker et al, 1971; Middleton et al, 1977; Nito et al, 1978; Klahr et al, 1986).

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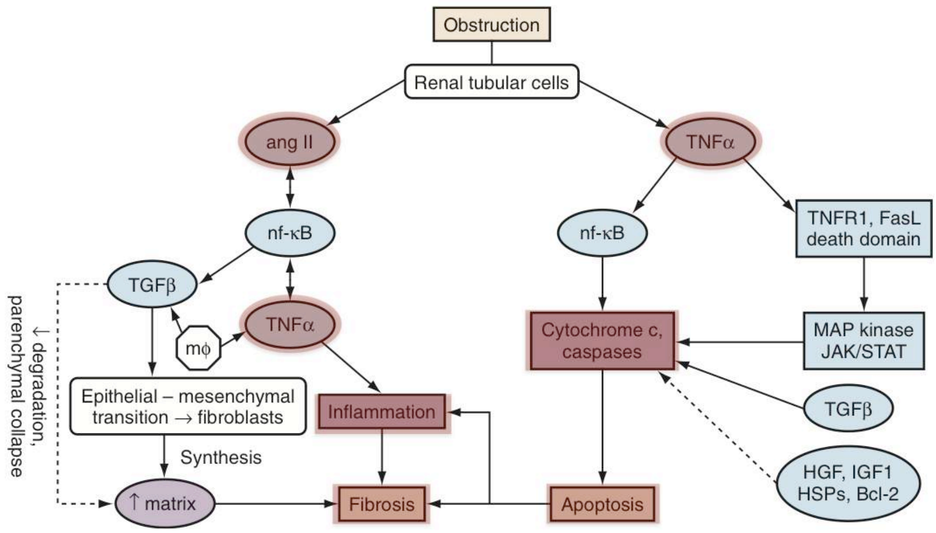
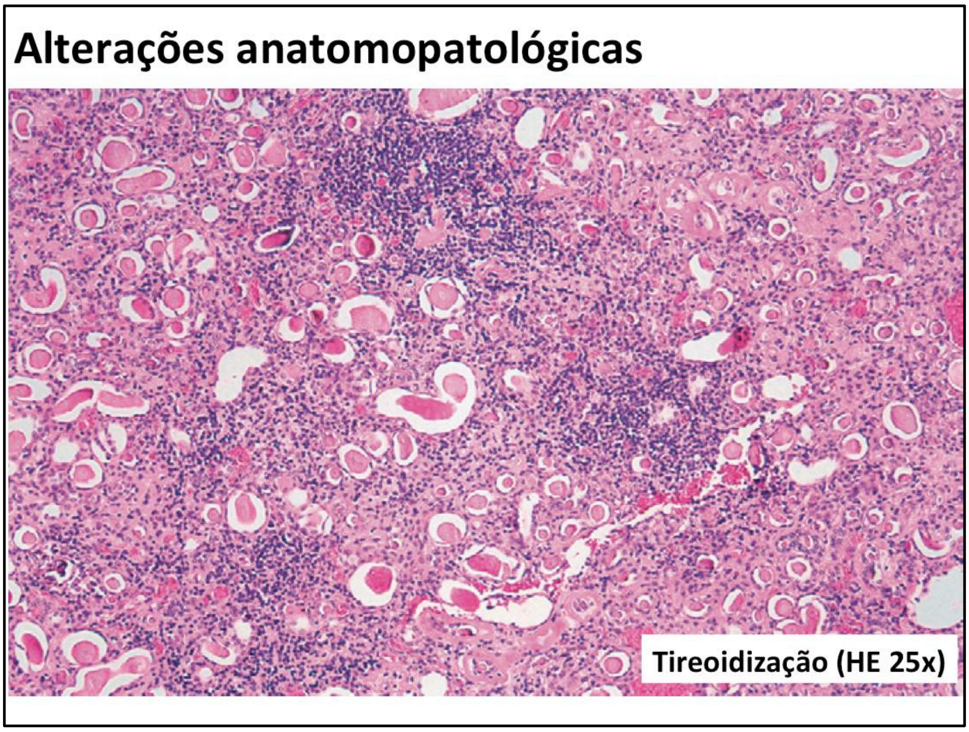
Alterações anatomopatológicas



Cellular and Molecular Mechanisms Leading to Tubular Cell Death through Apoptosis. Renal obstruction produces tubular atrophy and cell death. The major mechanism by which tubular cells die is apoptosis, a process that is normally involved in postnatal development and tissue renewal in adults. TNF-α can be a directly cytotoxic cytokine that can induce apoptosis in addition to its role in renal inflammation.

Cellular and Molecular Changes Leading to Fibrosis. Urinary tract obstruction leads to progressive and, eventually, permanent changes in the structure of the kidney, including the development of tubulointerstitial fibrosis, tubular atrophy and apoptosis, and interstitial inflammation. Obstruction increases the synthesis of tissue inhibitors of metalloproteinases (TIMPs) that reduce MMP activity, resulting in the accumulation of extracellular matrix. Although the events leading to fibrosis are thought to be initiated by increased angiotensin II, other profibrotic factors appear to play a significant role because inhibition of angiotensin synthesis by ACE inhibitors or antagonism of the AT1 receptors blunts but does not completely abolish the fibrotic process (Kaneto et al, 1993; Ishidoya et al, 1995; Pimental et al, 1995).

In summary, obstruction of normal urine outflow results in biochemical, immunologic, hemodynamic, and functional changes. It stimulates a cascade in which elevated levels of angiotensin II, cytokines, and growth factors lead to tubular cell apoptosis and cellular inflammation, increased net matrix formation, and tubulointerstitial fibrosis. Many of the mediators are intrinsic to the renal tubular cells, whereas others are contributed by fibroblasts and by migrating macrophages. Figure subtitle: Summary of major pathways leading to tubulointerstitial fibrosis and tubular apoptosis as a consequence of ureteral obstruction. Membrane proteins and regulators are discussed in the text. ang II, angiotensin II; HGF, human growth factor; HSPs, heat shock proteins; IGF, insulin-like growth factor; JAK/STAT, Janus kinase/signal transducers and activators of transcription; mϕ, macrophages; MAP, mitogen-activated protein; NF-κB, nuclear factor κB; TGF, transforming growth factor; TNF, tumor necrosis factor; TNFR1, tumor necrosis factor receptor 1.



While the kidney enlarges, an increase in the number of nephrons or glomeruli does not occur (Peters et al, 1993). However, an increase in the length of the proximal tubule has been described, which may be due to an increase in cell size (Moller, 1988). In addition, there is augmented extracellular matrix synthesis and growth of mesangial cells (Kasinath et al, 2006; Sinuani et al, 2006). Insulin-like growth factor I (IGF- I), a mitogenic and anabolic peptide, may play a role in compensatory renal growth after obstruction.
Other growth factors, cytokines, and enzymes may be involved in regulating CRG, including IGF binding protein–3 (IGFBP-3), vascular endothelial growth factor (VEGF), matrix metalloproteinase–9 (MMP-9), interleukin-10 (IL-10), and TGF-β (Yildiz et al, 2008).
Figure subtitle: Sections of deep cortex and outer medulla from a patient with chronic obstructive uropathy. Tubules demonstrate thyroidization-type atrophy interspersed with mononuclear inflammatory infiltrate. Hematoxylin and eosin stain; original magnification, ×25. (Courtesy of Dr. Sami Iskandar.)

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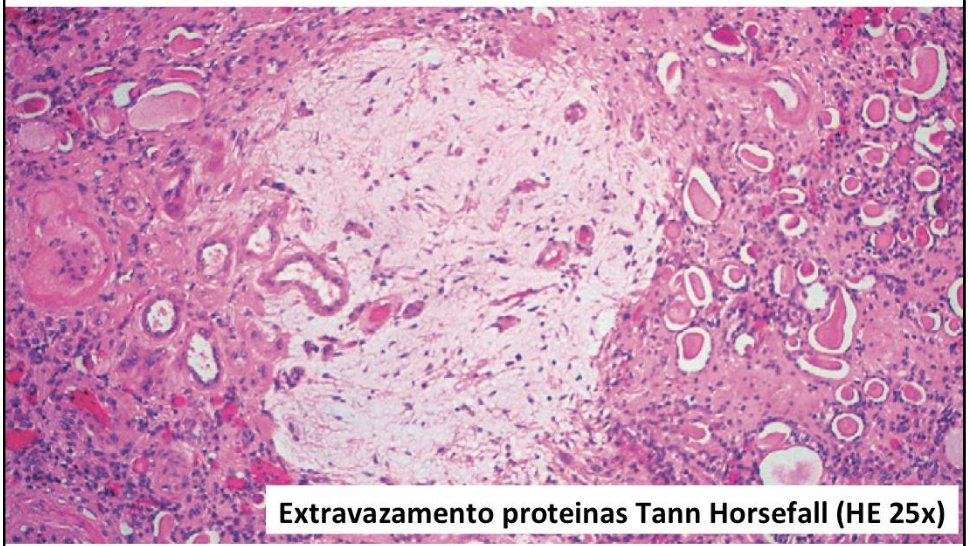
Alterações anatomopatológicas



Sections of deep cortex and outer medulla from a patient with chronic obstructive uropathy. Obsolescent glomerulus *(left edge)* and pool of extravasated Tamm-Horsfall protein *(center)* are seen. Hematoxylin and eosin stain; original magnification, ×25. (Courtesy of Dr. Sami Iskandar.)

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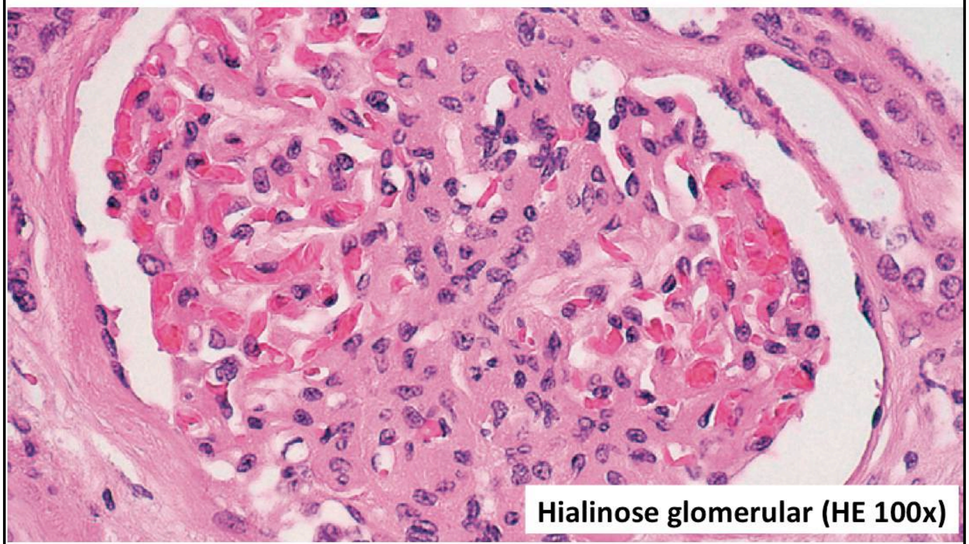
Alterações anatomopatológicas



Sections of deep cortex and outer medulla from a patient with chronic obstructive uropathy. Glomerulus with segmental tuft sclerosis *(center)* and hyalinosis is seen. Hematoxylin and eosin stain; original magnification, ×100. (Courtesy of Dr. Sami Iskandar.)

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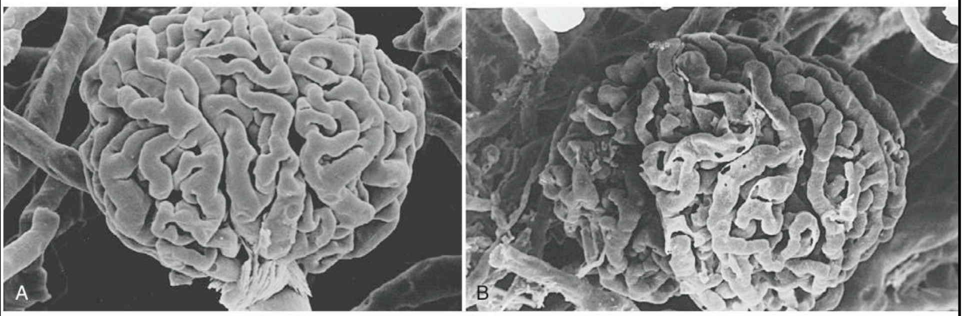
Alterações anatomopatológicas



Normal
MEV 390x

Obstrução:
Colapso e irregularidade
Capilar
MEV 390x

On the right Scanning electron microscopic appearance of a normal glomerular cast (×390).
On the left: Appearance of a glomerular microvascular cast after obstruction shows capillary collapse and irregularity (×390).

Leahy AL, Ryan PC, McEntee GM, et al. Renal injury and recovery in partial ureteric obstruction. J Urol 1989;142:199–203.

Recuperação renal pós obstrução

- Quanto tempo o rim resiste a obstrução?
- Cintilografia com ácido dimercaptosuccinico (DMSA)
- Duas fases:
  - Recuperação da função tubular (primeiras 2 sem)
  - Recuperação do ritmo filtração glomerular (10 sem)

When acute, complete ureteral obstruction is promptly relieved, full recovery of global GFR can occur. The fate of the human kidney after prolonged periods of obstruction is less well defined and may be unpredictable. Functional recovery has been reported after a 150-day period of UUO (Shapiro and Bennett, 1976). Permanent reductions in GFR may occur after UUO or BUO in humans. However, there may be a differential pattern of recovery after BUO in adults. Jones and associates (1988) described two phases of functional improvement: an initial phase during the first 2 weeks after relief of obstruction, when tubular function improved, and a later phase occurring over the next 10 weeks, when GFR gradually improved. Patients with BUO or obstruction of a solitary kidney may also be at risk for unique sequelae such as chronic urinary acidification and concentrating defects (Berlyne, 1961). Nuclear renography may help predict functional recovery. An assessment of functioning cortex with such imaging is the best predictor. For example, dimercaptosuccinic acid (DMSA), a cortical agent, has been shown to be superior to tubular selective agents, such as diethylenetriaminepentaacetic acid (DTPA) or mercaptoacetyltriglycine (MAG3), for the prediction of renal recovery. This was demonstrated in a prospective study in children undergoing pyeloplasty or ureteral reimplantation for ureterovesical junction obstruction (Thompson and Gough, 2001). It was postulated that DMSA better defines the functional cortex and is less affected by the dilated collecting system and dilution by undrained urine. Similarly, when the tubular agent *ortho*-iodohippurate was used, whole kidney activity was less predictive than the cortical phase (Kalika et al, 1981).

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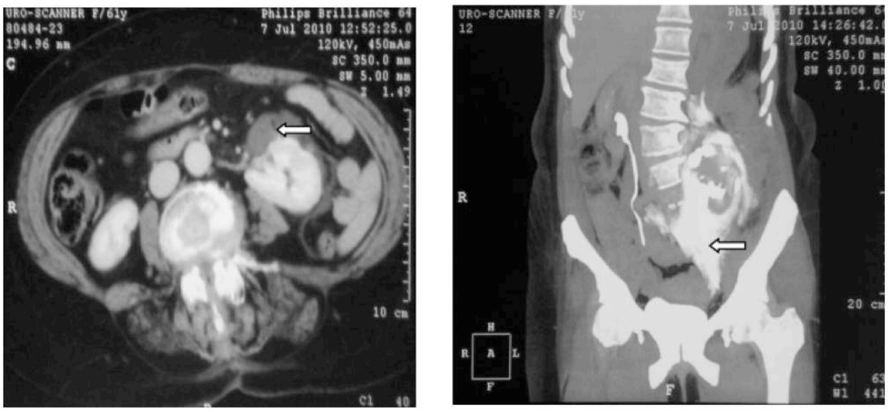
Mecanismos compensatórios

- extravasamento pericalicial
- Extravasamento para o sistema venoso
- Extravazamento linfático



Mecanismos naturais compensatórios visam diminuir a pressão no sistema pielocalicial e colaboram para preservação do parenquima renal.

Mecanismos compensatórios

- Poliúria pós obstrutiva (200 ml/h)
  - Pós obstrução bilateral: comum
  - Pós obstrução infravesical: comum
  - Pós obstrução unilateral: incomum
  - Pouca responsividade ao ADH
  - Alterações do peptídio natriurético atrial e *downregulation do* canais aquaporinos (alteração da concentração urinária)

Li et al, 2007

Following the relief of urinary tract obstruction, postobstructive diuresis—a period of significant polyuria— may ensue. Urine outputs of 200 mL/hr or greater may be encountered.

The increased endogenous production and altered regulation of ANP induce a saline diuresis (Kim et al, 2001b). In addition, other natriuretic peptides, such as the *Dendroaspis* natriuretic peptide, may play a role (Kim et al, 2002). Pathologic postobstructive diuresis is also marked by poor responsiveness of the collecting duct to antidiuretic hormone (ADH). This is thought to be due to a downregulation of aquaporin water channels in this segment of the nephron and perhaps in the proximal tubule, which has been demonstrated in experimental models of UUO and BUO (Nielsen and Agre, 1995; Frøkiaer et al, 1996; Kim et al, 2001a; Li et al, 2003).

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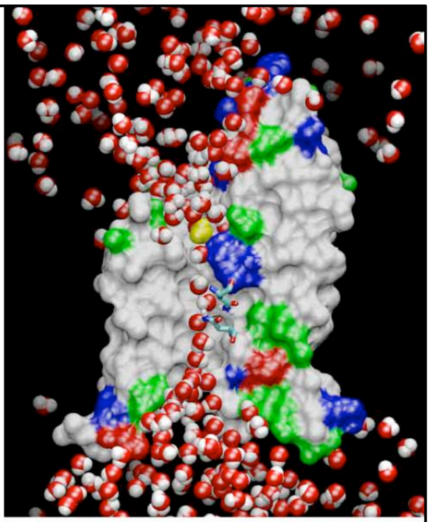
Concentração urinária

Fatores reguladores

- gradiente medular intersticial hipertônico (reabsorção ativa de sais no ramo ascendente da alça Henle)
- Refluxo da uréia do ducto coletor
- Permeabilidade à água do ducto coletor (mediada pela vasopressina e canais aquaporinos)



Normal urine concentrating ability requires a hypertonic medullary interstitial gradient because of active salt reabsorption from the thick ascending limb of Henle, urea back flux from the inner medullary collecting duct, and water permeability of the collecting duct mediated by vasopressin and aquaporin water channels.
Thus dysregulation of aquaporin water channels in the proximal tubule, thin descending loop, and collecting duct may contribute to the long-term polyuria and impaired concentrating capacity caused by obstructive nephropathy. Increases in angiotensin II may be a media- tor of these changes.

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Video: Proc Am Thorac Soc. 2006 Mar; 3(1): 5–13. doi: 10.1513/pats.200510-109JH PMCID: PMC2658677 The Aquaporin Water Channel.

Diagnóstico da obstrução - ultrassonografia

- Vantagens: sem contraste (IR), sem radiação (gestantes)
- Desvantagens: exame não é funcional

$$\text{Índice de resistividade} = \frac{\text{Velocidade de pico sitólica} - \text{Velocidade final diastólica}}{\text{Velocidade de pico sitólica}}$$

There is no associated ionizing radiation, and it is thus considered safe in pediatric and pregnant patients. Because there is no need for iodinated contrast material, ultrasonography can be used in those with azotemia or contrast allergy. Doppler ultrasonography allows measurement of the renal resistive index (RI), which has been used to assess for obstruction. The RI is defined as peak systolic velocity (PSV) minus the end-diastolic velocity (EDV) divided by the PSV. Several groups have assessed its ability to diagnose obstruction, but its utility has yet to be fully established.

Diagnóstico da obstrução - ureteropielografia

- Exame de exceção
- Impossibilidade de uso de contraste IV
- Má demonstração do ponto de obstrução por outros métodos de imagem
- Retrógrada (cateterismo ureteral) ou anterógrada (punção renal)

Retrograde pielogtaphy: It should be considered in those who have renal insufficiency or have other risks for receiving intravenous iodin- ated contrast material. It may also be used in cases in which anatomy is not sufficiently defined with other imaging studies.
Antegrade Pyelography: This technique may be helpful when other imaging studies do not adequately define collecting system or ureteral anatomy and when retrograde pyelography is not technically feasible.

Diagnóstico da obstrução - cintilografia

- Exame funcional: DTPA Tc99m ou MAG3 Tc99m
- Eliminação de 50% do marcador (meia vida)

  < 10 min: normal

  > 20 min: obstrução
- Associação com diuréticos para aumentar a sensibilidade

It provides a functional assessment without exposure to iodinated contrast material. Radiopharmaceuticals for renography are selected on the basis of the function to be studied. The glomerular agent technetium (Tc) 99m DTPA and the tubular agent 99mTc-MAG3 are most commonly used in the evaluation of obstruction. 99mTc-MAG3 has a high renal extraction rate, associated with rapid clearance, lower radiation dose, and tubular secretion. 99mTc-MAG3 has a 55% renal uptake in contrast with a 20% renal uptake associated with 99mTc- DTPA (Tremel et al, 1996). By convention, a half-time less than 10 minutes is considered normal, greater than 20 minutes is considered obstructed, and between 10 and 20 minutes is equivocal. The diuretic renogram is a modification designed to maximize flow and potentially distinguish truly obstructed collecting systems from those that are dilated but unobstructed. Because a given patient's response to the diuretic may be affected by the patient's baseline renal function, adjustments may need to be made on the basis of creatinine clearance (Upsdell et al, 1988). In diuretic renography, as classically described, the diuretic, typically furosemide (F), is administered 20 minutes after the tracer to induce a brisk diuresis (F+20 study).

Diagnóstico da obstrução - tomografia

- Exame funcional e anatômico
- Sem contraste: somente avaliação do volume renal (somente para urolitiase)
- Com contraste: alterações da perfusão, concentração, excreção e eliminação do contraste, edema e coleções perirenais

Noncontrast CT directly demonstrates calculi classically considered radiolucent when evaluated by plain radiography, including uric acid, xanthine, dihydroxyadenine, and many drug-induced stones. Exceptions, however, are calculi composed of protease inhibitors, which are not visualized by noncontrast CT (Gentle et al, 1997). Secondary CT signs of obstruction such as ureteral dilatation, nephromegaly, decreased parenchymal density of the involved kidney as compared with the contralateral renal unit, perinephric stranding, or fluid can facilitate the diagnosis of acute obstruction. These signs have been reported to have a positive predictive value of 99% and negative predictive value of 95% for identification of acute ureteral obstruction (Smith et al, 1996). This is also an excellent technique for evaluating patients suspected of having chronic obstruction who have renal insufficiency or other contrast risks.

Diagnóstico da obstrução – ressonância

- Exame funcional e anatômico
- Sem contraste: avaliação das imagens em T2
- Com contraste (gadolínio)
- Uso em pacientes alérgicos a contraste iodado ou uso de irradiação estiver contra-indicado

Magnetic resonance urography (MRU) can be executed essentially with two different imaging techniques: (1) static-fluid MRU that relies heavily on the T2-weighted turbo spin echo (TSE) sequences, and (2) gadolinium-enhanced excretory MRU, a dynamic sequence that relies on the administration of gadolinium with or without a diuretic. The risk of nephrogenic systemic fibrosis associated with the use of some gadolinium agents (especially gadodiamide) in patients with renal insufficiency (GFR < 30 mL/min), limits the utility of using contrast MRI in such patients (Broome et al, 2007; Frøkiaer and Zeidel, 2007; Sadowski et al, 2007; Abou El-Ghar et al, 2008b). The absence of ionizing radiation has allowed its use in the assessment of children, women of child-bearing age, and pregnant patients (Jorgensen et al, 2006) with flank pain and hydronephrosis. In children MRU has the potential to replace conventional uroradiological diagnostic tests in the near future. The present level of evidence does not suggest that MRI is clearly superior to combined ultrasonography (US) and CT imaging for the routine diagnosis and evaluation of obstructive uropathy.
On the whole, both MRU and CT have critical roles in evaluating select patients with urinary tract obstruction. Although these studies can estimate renal function (He and Fischman, 2008), nuclear renography remains the current gold standard.

Causas de obstrução

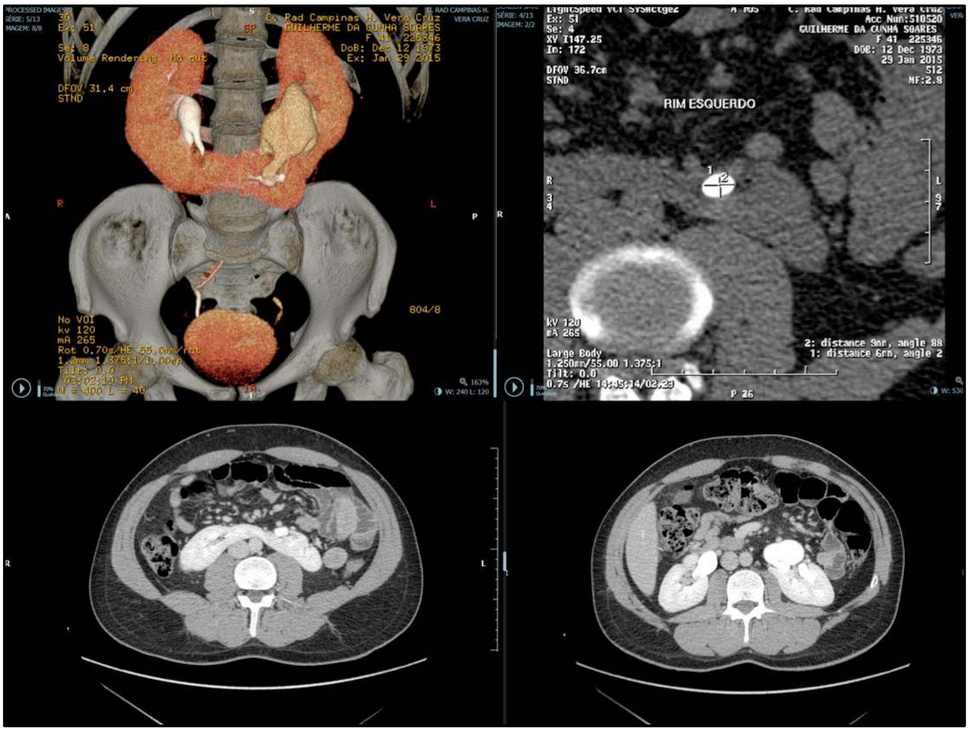
Ureteral

- Urolitiase
- Neoplasias
- Causas inflamatórias
- Malformações

Infravesical

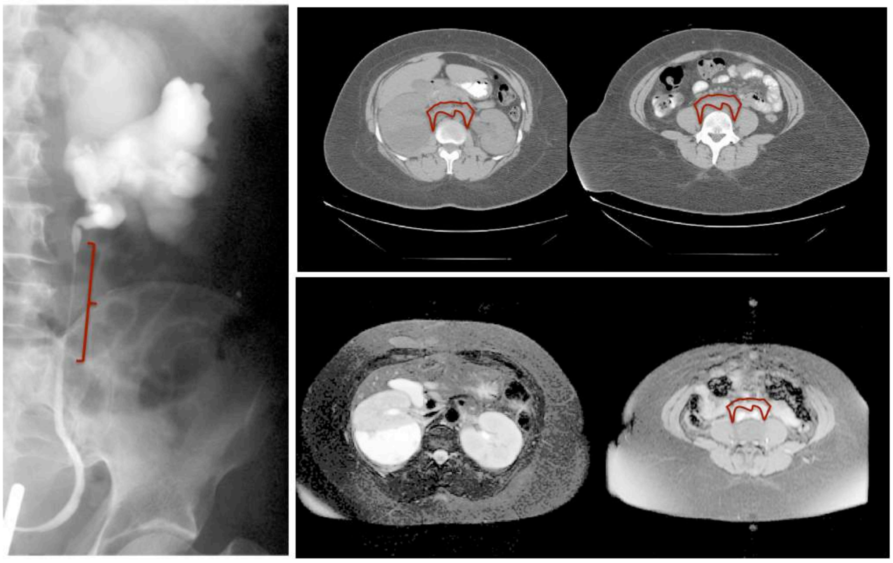
- Neoplasias
- Causas inflamatórias
- Aumento prostático benigno

Principais causas de obstrução ureteral (unilateral ou bilateral) e de obstrução infravesical. Na obstrução infravesical todo o sistema é comprometido.



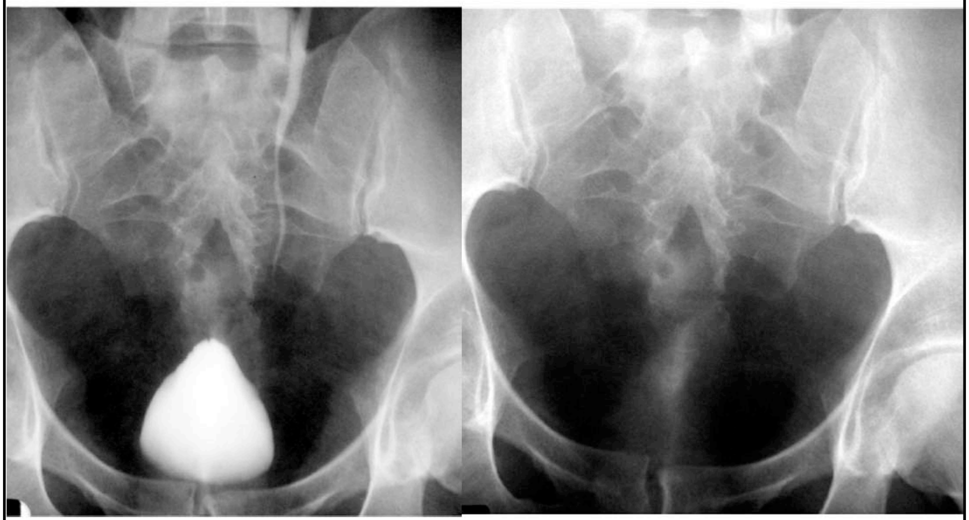
Rim em ferradura com calculo causando obstrução do ureter esquerdo. Os rins com malformação são mais propensoas a processos obstrutivos e, em gral, tem drenagem urinária mais lenta, que predispõe a cristalização de calculos.

Exemplos: fibrose retroperitoneal

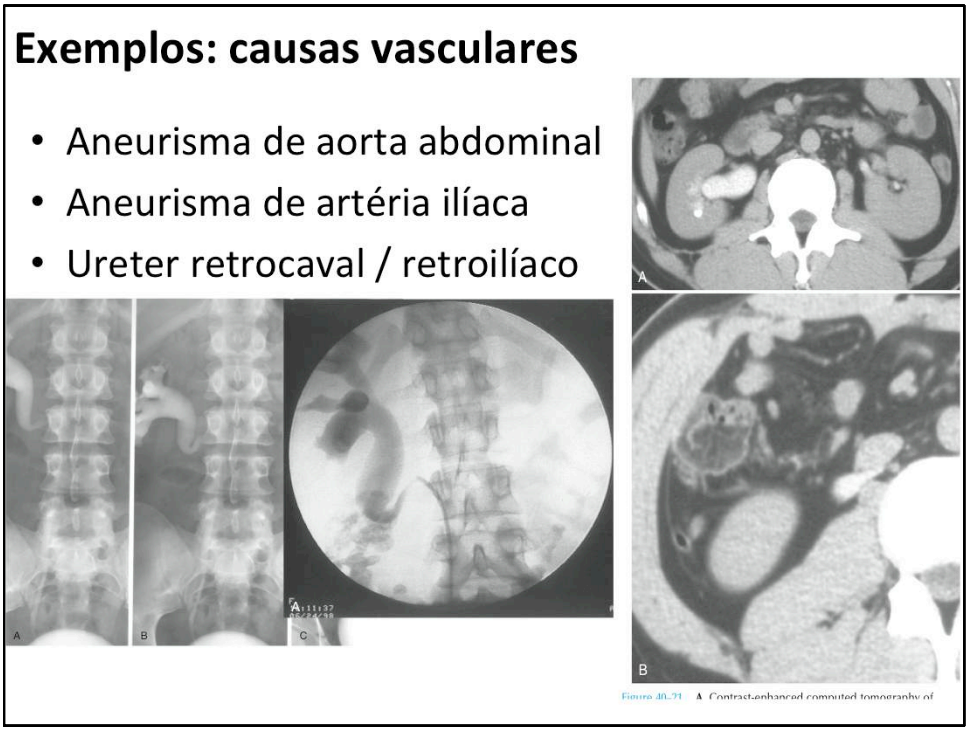


Exemplo de fibrose retroperitoneal. Nela, há proliferação de tecido conjuntivo denso retroperitoneal, que pode deslocar ou envolver os grandes vasos (aorta e veia cava inferior), determinando obstrução ureteral. No exemplo observa-se esse processo numa ureteropielografia retrógrada (a dir) e numa tomografia de abdome (a esquerda).

Exemplos: Lipomatose pélvica



Pelvic lipomatosis is a rare, benign condition marked by exuberant pelvic overgrowth of nonmalignant but infiltrative adipose tissue, usually in the abdominal and pelvic cavities. Physical findings may include a suprapubic mass, a high-riding prostate, and an indistinct pelvic mass.



Ectopias ureterais podem determinar obstrução de causa congenita.

Exemplos: malformações (anomalia de JUP)

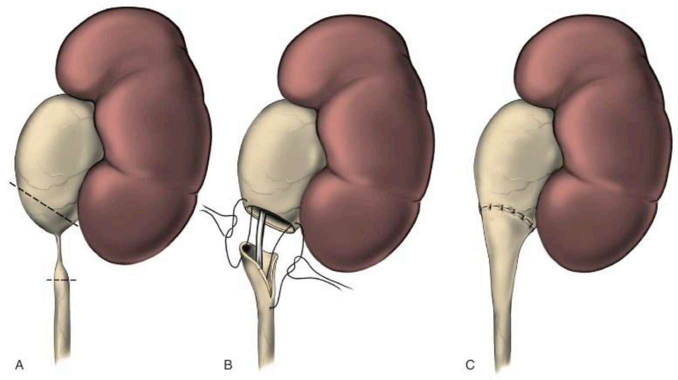
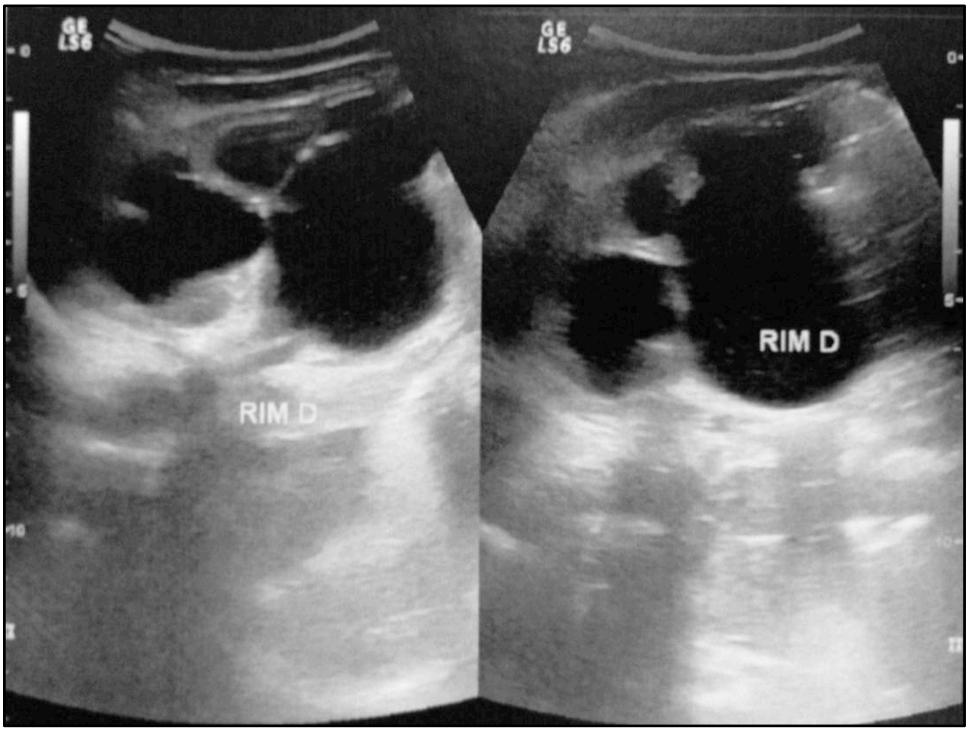


Diagrama exemplifica a pieloplastia desmembrada, proposta por Anderson-Hynes, para tratamento da estenose da junção pieloureteral, que é uma causa frequente de hidronefrose congenita.

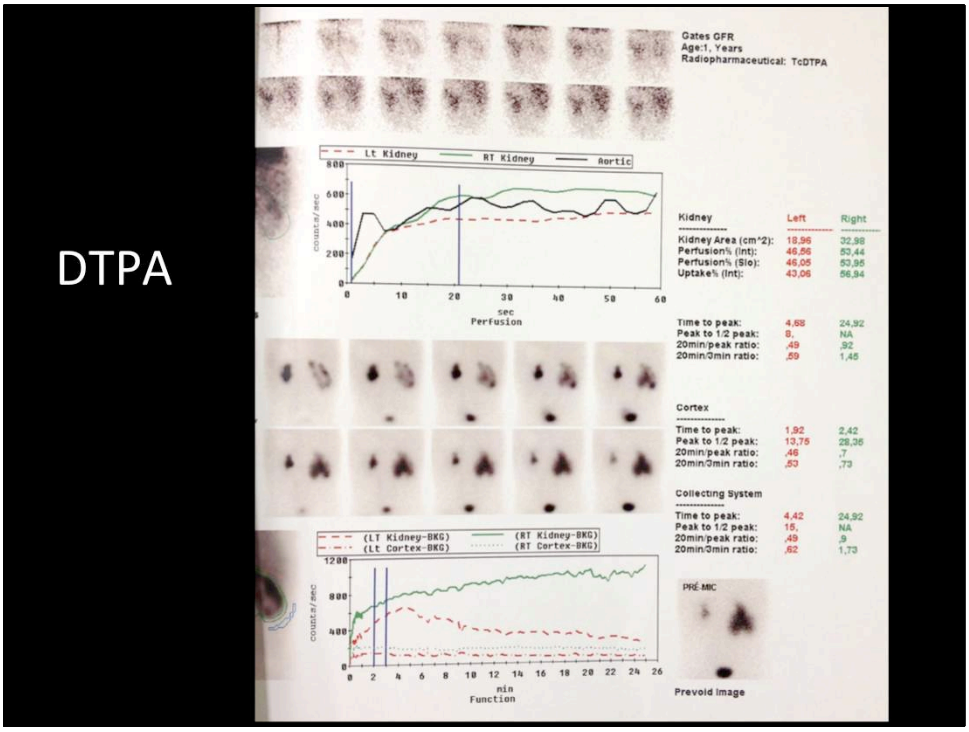
- Masc, 2 anos e 2 meses

- 2 episódios de ITU
- US Rim D dilatação moderada, ureter não vizualizado
- Uri/Uro negativos

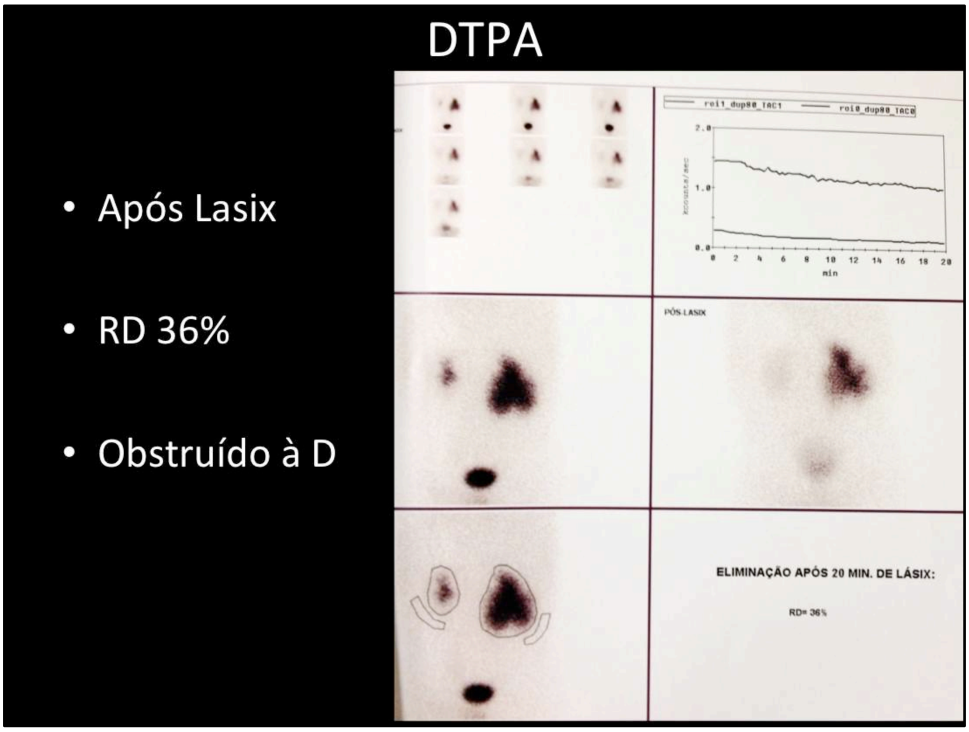
Caso clinico exemplificando estenose de junção pieloureteral (JUP)



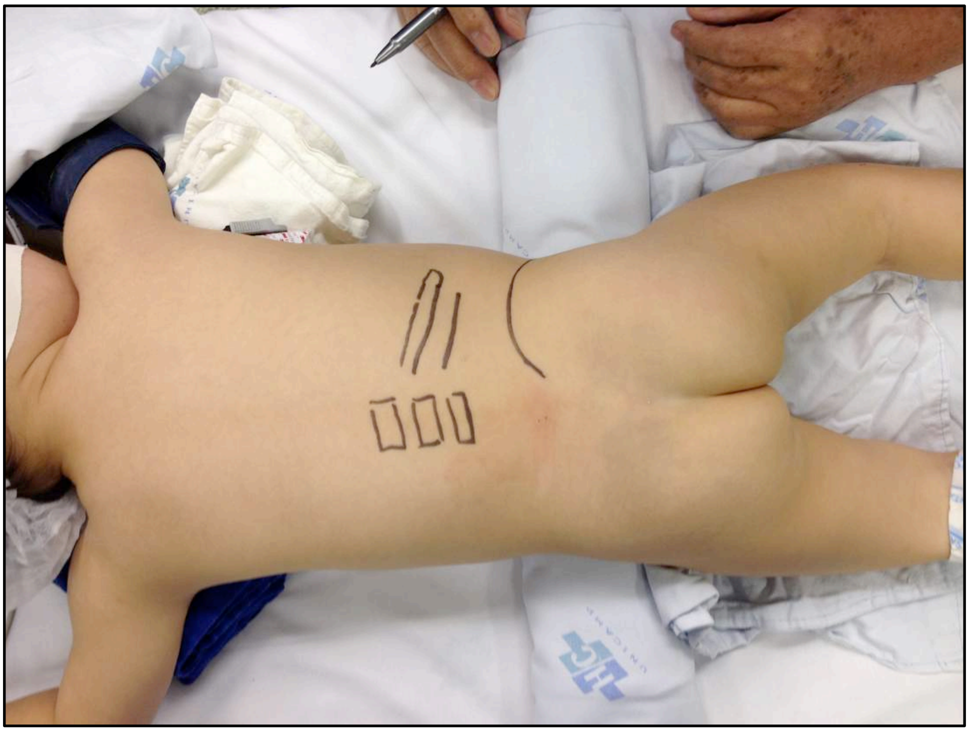
Caso clinico exemplificando estenose de junção pieloureteral (JUP): aspecto ultrassonográfico



Caso clinico exemplificando estenose de junção pieloureteral (JUP): cintilografia com DTPA

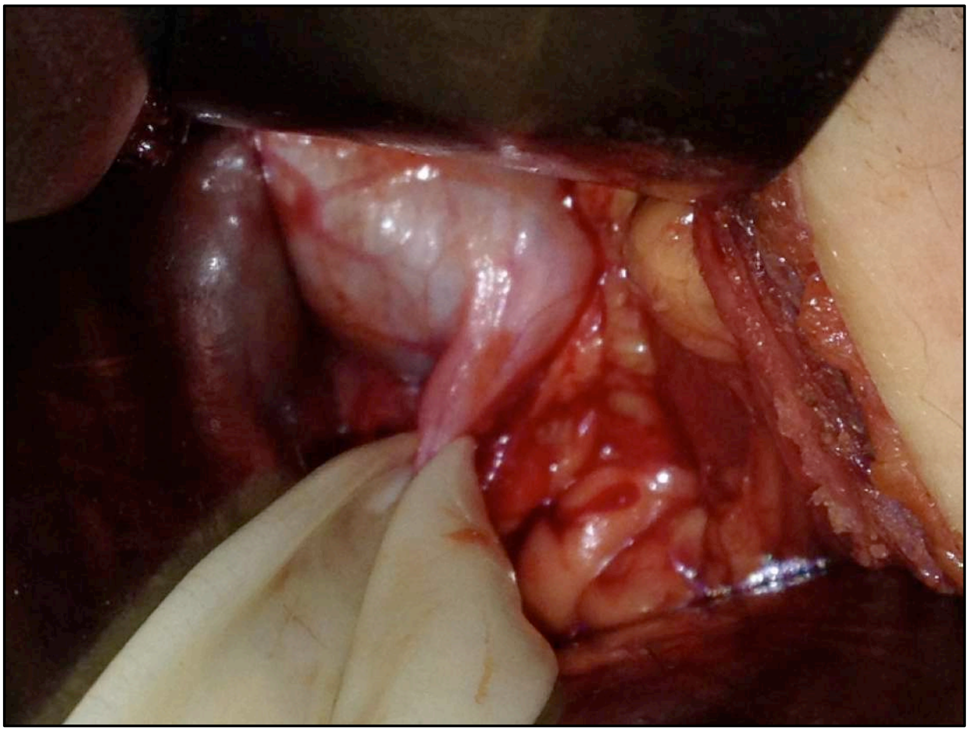


Caso clinico exemplificando estenose de junção pieloureteral (JUP): cintilografia com DTPA. O decaimento da taxa de radioatividade inferior a 50% após 20 min da injeção do contraste define a osbtrução.

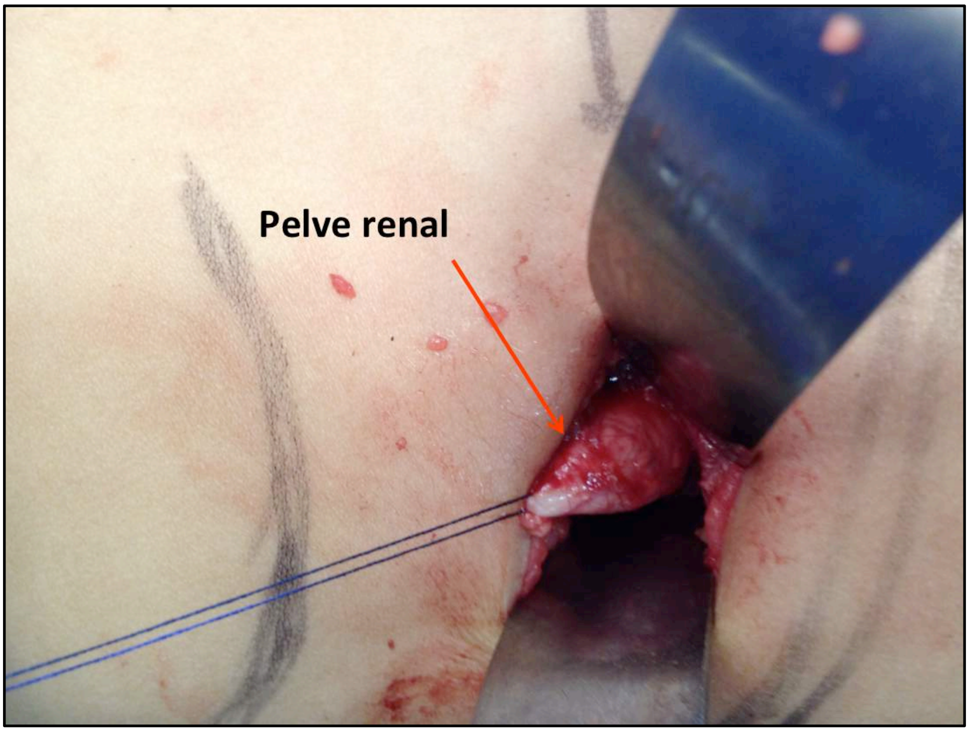


Pieloplastia desmembrada a Anderson-Hynes por incisão posterior. O procedimento pode ser realizado, também, por laparoscopia.

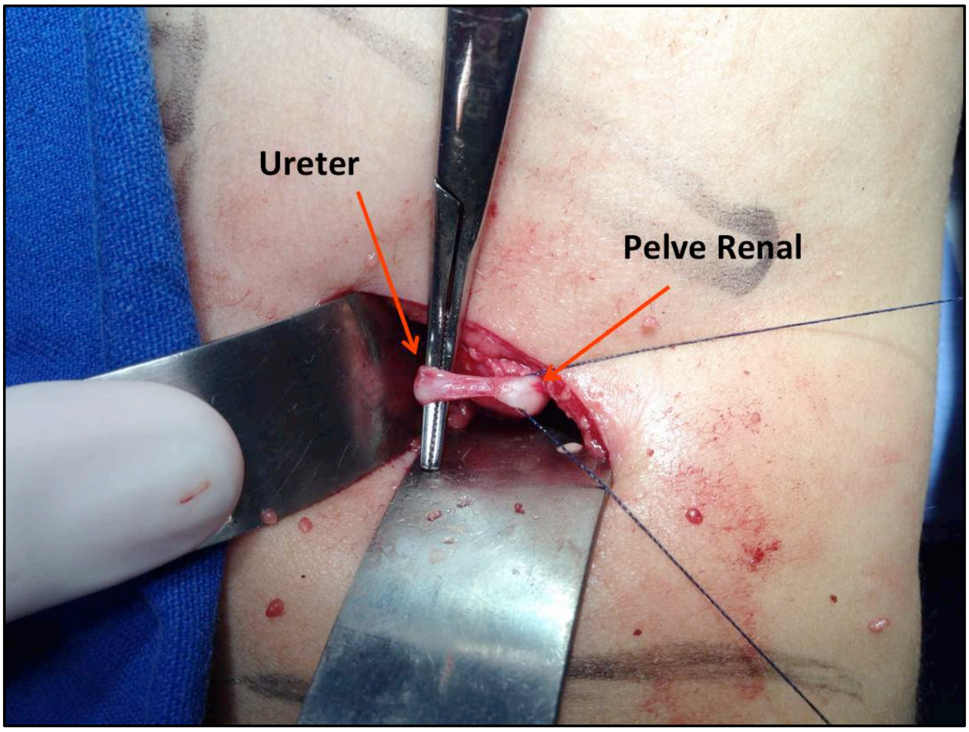
Pieloplastia desmembrada a Anderson-Hynes por incisão posterior. O procedimento pode ser realizado, também, por laparoscopia.



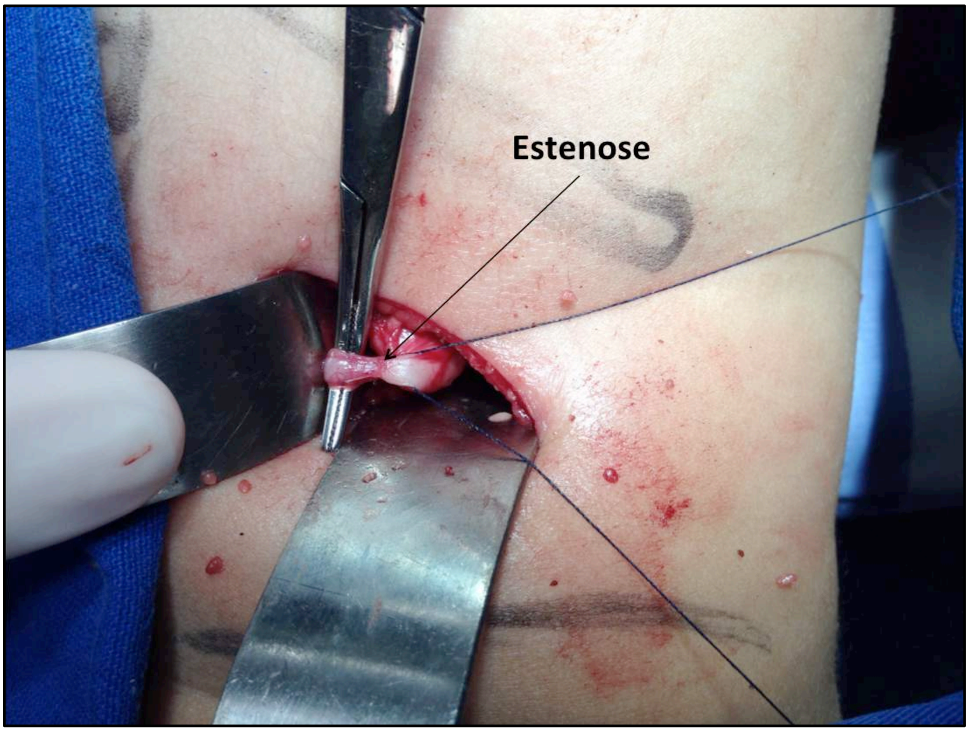
Pieloplastia desmembrada a Anderson-Hynes por incisão posterior. O procedimento pode ser realizado, também, por laparoscopia.



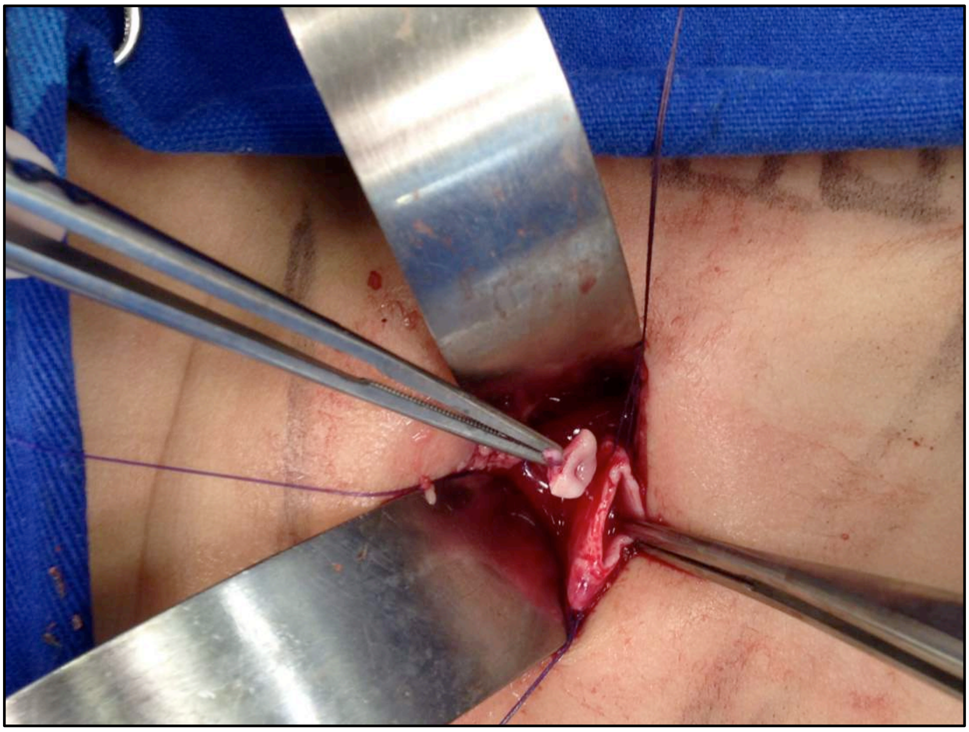
Pieloplastia desmembrada a Anderson-Hynes por incisão posterior. O procedimento pode ser realizado, também, por laparoscopia.



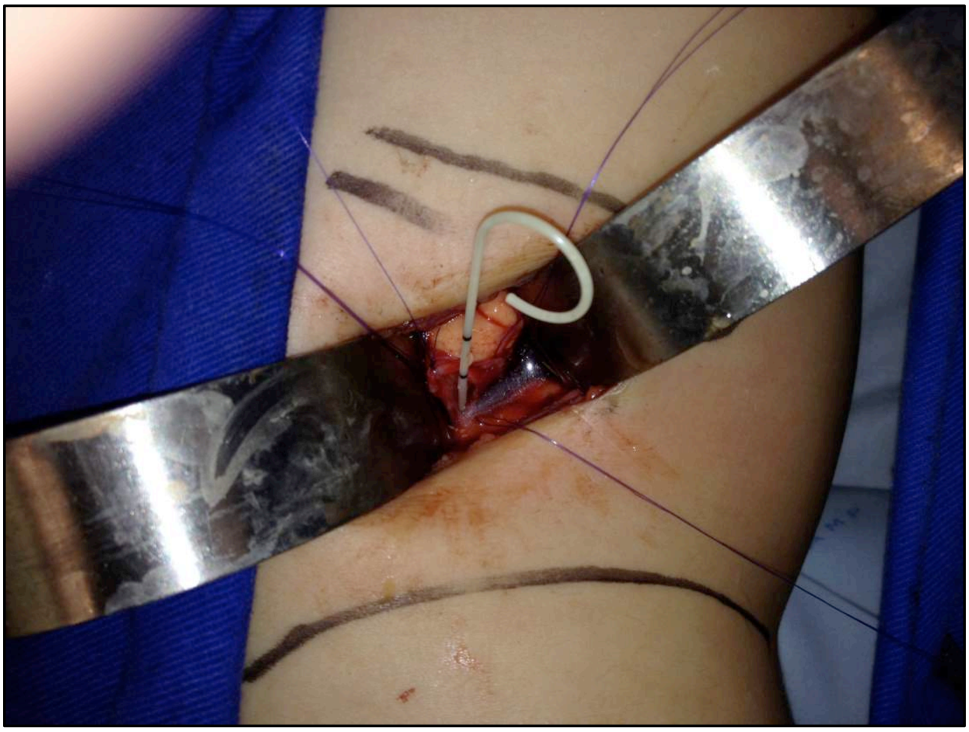
Pieloplastia desmembrada a Anderson-Hynes por incisão posterior. O procedimento pode ser realizado, também, por laparoscopia.



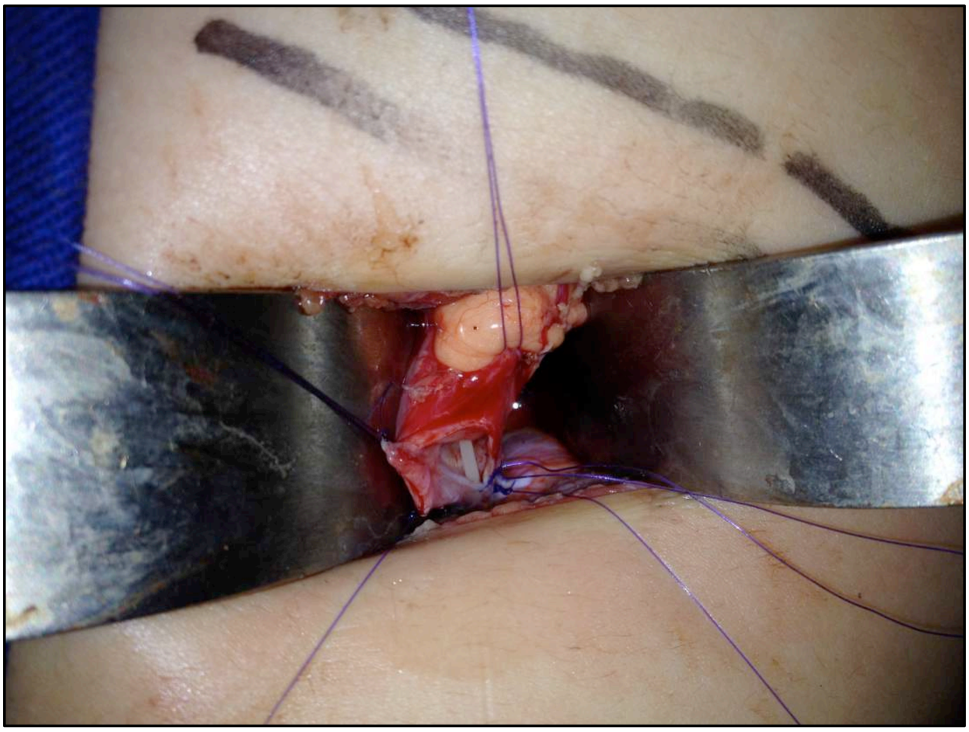
Pieloplastia desmembrada a Anderson-Hynes por incisão posterior. O procedimento pode ser realizado, também, por laparoscopia.



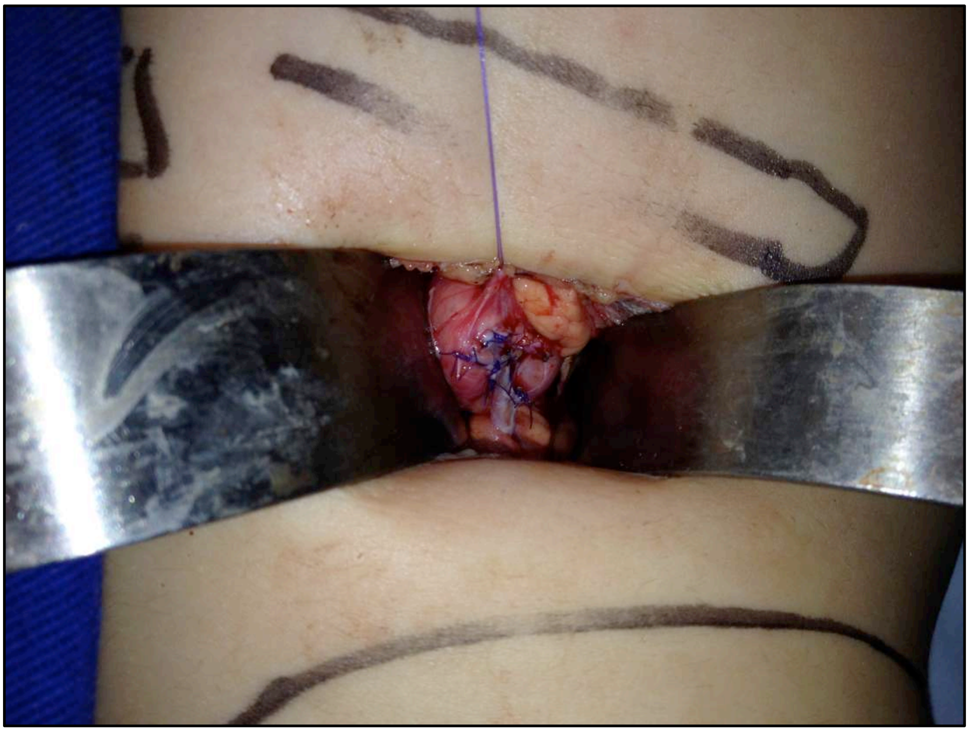
Pieloplastia desmembrada a Anderson-Hynes por incisão posterior. O procedimento pode ser realizado, também, por laparoscopia.



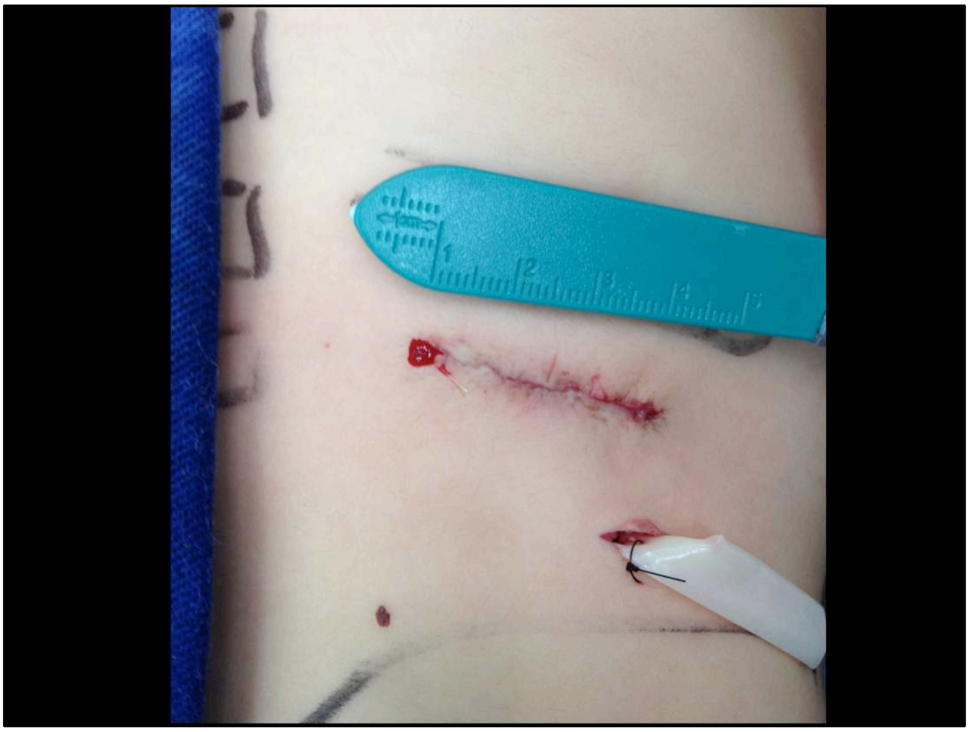
Pieloplastia desmembrada a Anderson-Hynes por incisão posterior. O procedimento pode ser realizado, também, por laparoscopia.



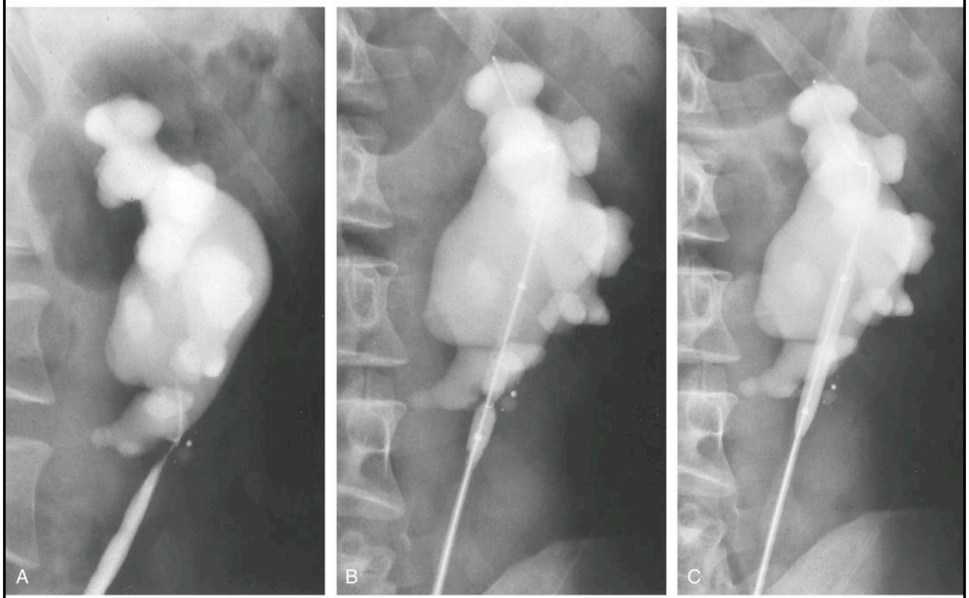
Pieloplastia desmembrada a Anderson-Hynes por incisão posterior. O procedimento pode ser realizado, também, por laparoscopia.



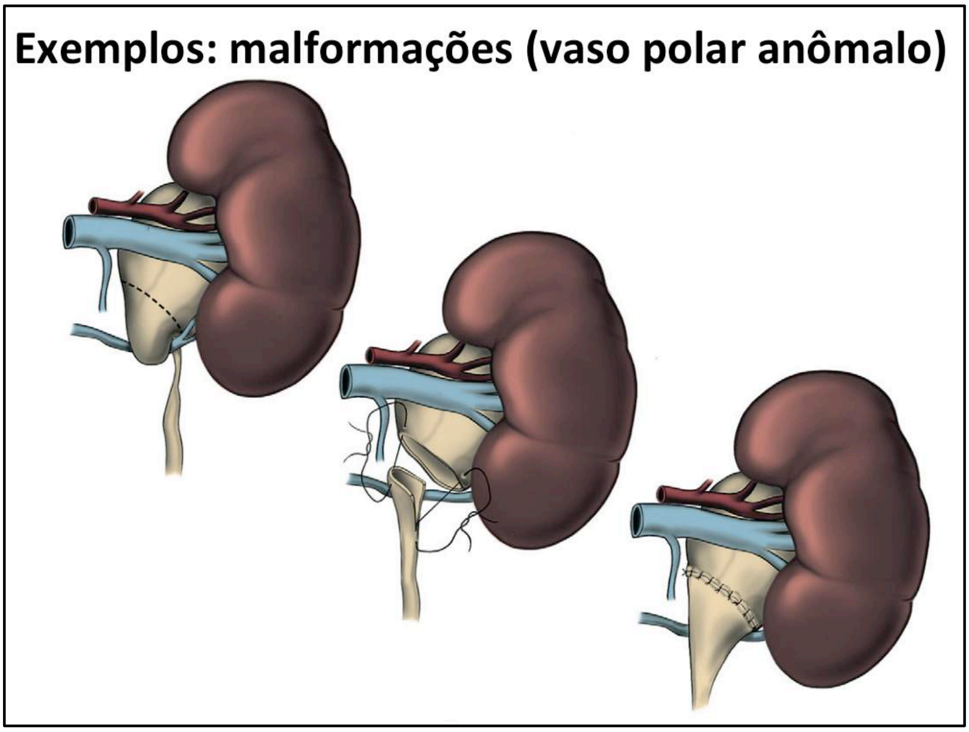
Pieloplastia desmembrada a Anderson-Hynes por incisão posterior. O procedimento pode ser realizado, também, por laparoscopia.



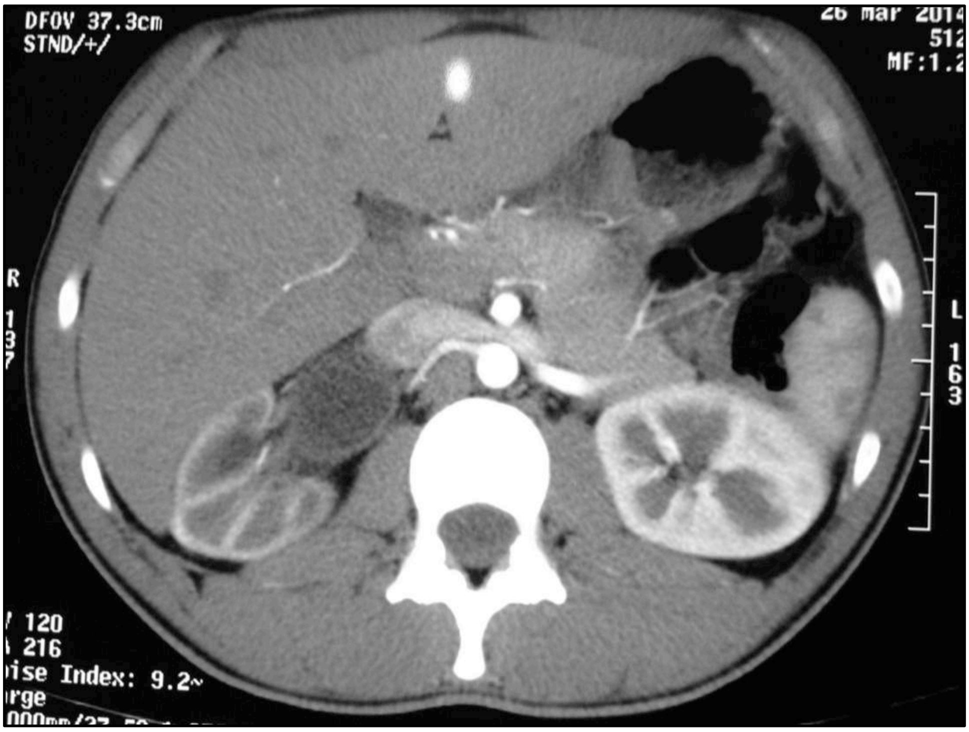
Pieloplastia desmembrada a Anderson-Hynes por incisão posterior. O procedimento pode ser realizado, também, por laparoscopia.



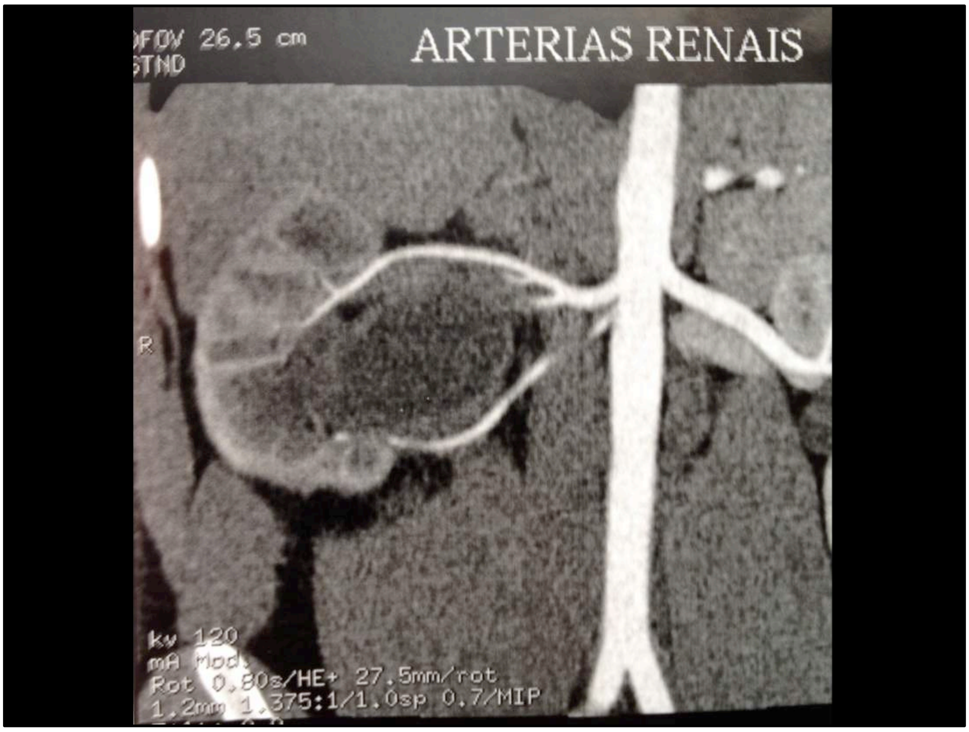
Rim com má-rotação (observe que nesse exame contrastado os calices não projetam-se em plano coronal). É demonstrado procedimento de dilatação da JUP com balão de dilatação uteteral.



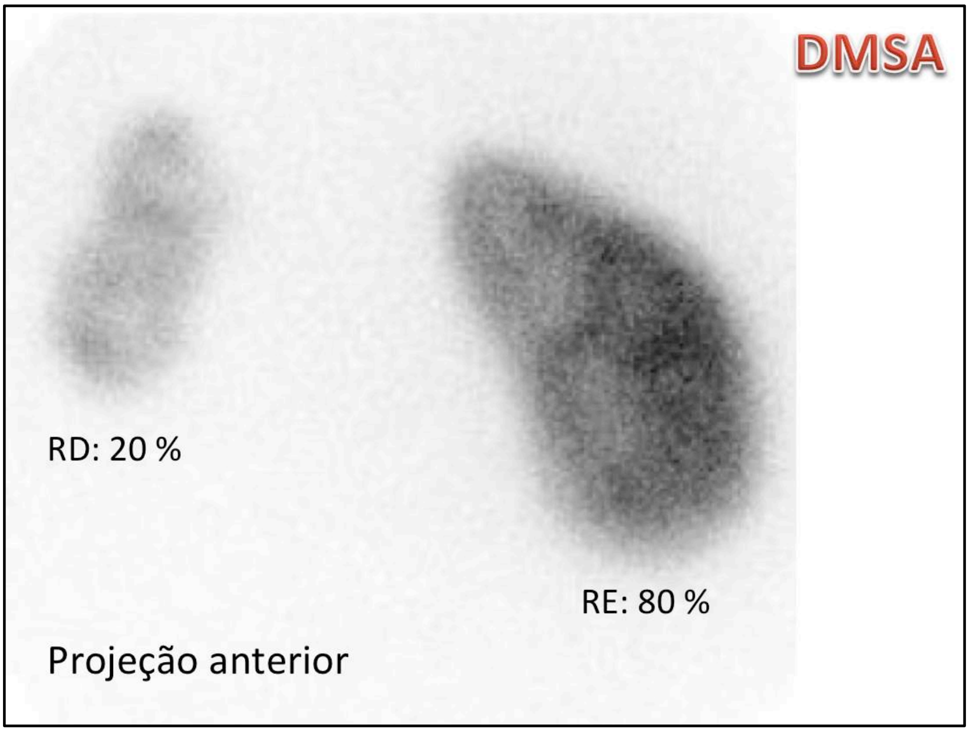
Esquema demonstrando a pieloplastia desmembrada a Anderson-Hynes em paciente com obstrução ureteral por vaso de localização anomala no polo inferior do rim esquerdo.



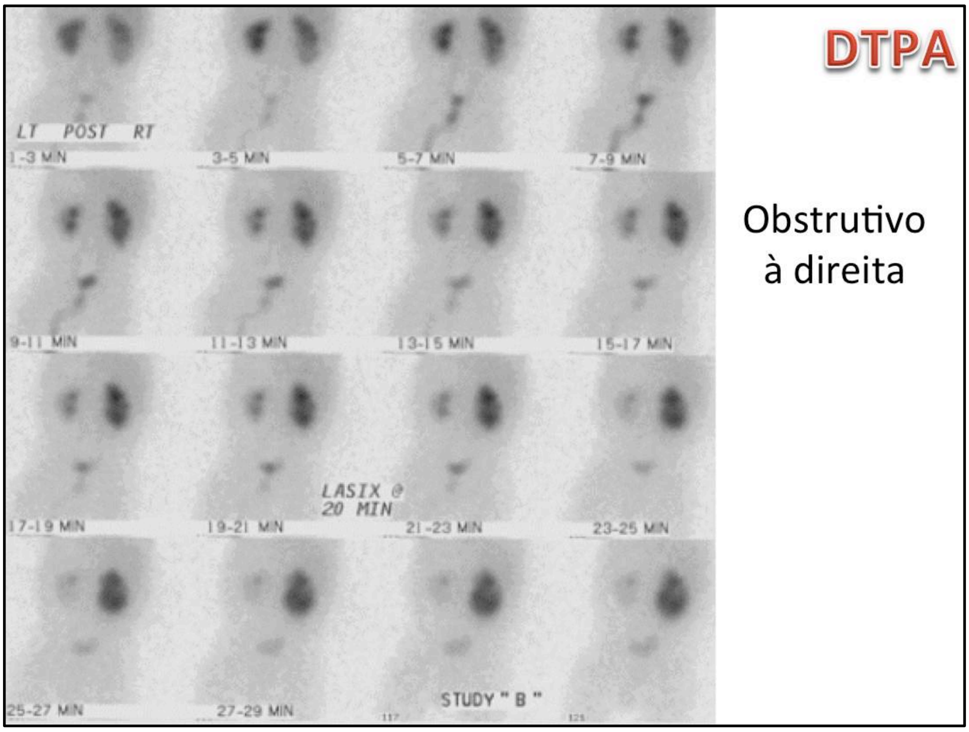
Tomografia evidenciando obstrução ureteral por vaso de localização anomala no polo inferior do rim direito



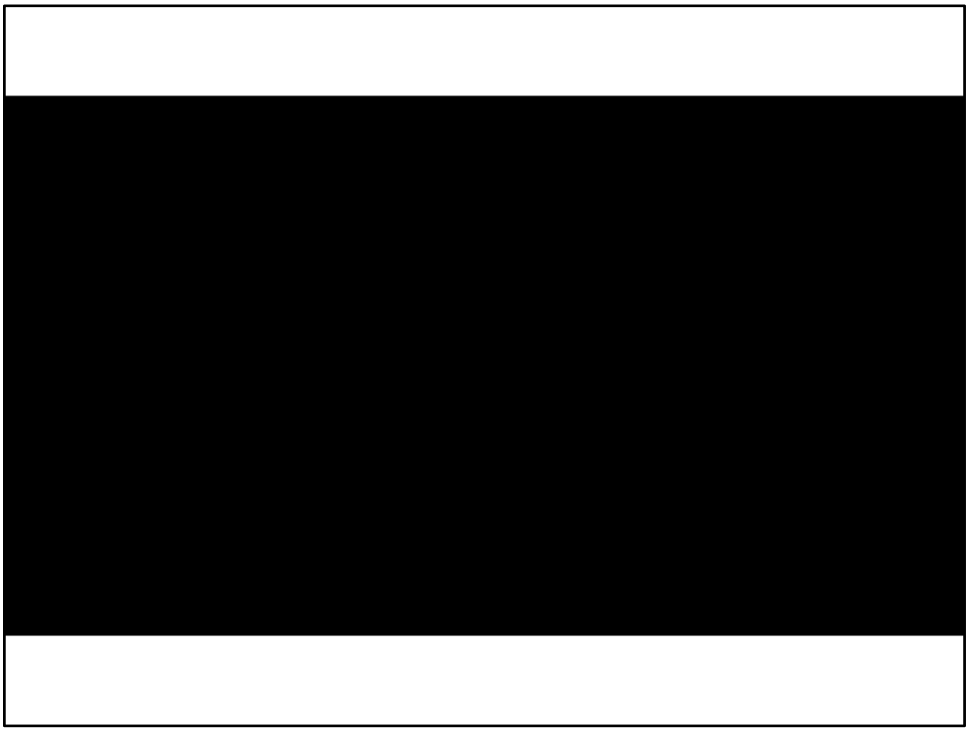
Tomografia evidenciando obstrução ureteral por vaso de localização anomala no polo inferior do rim direito



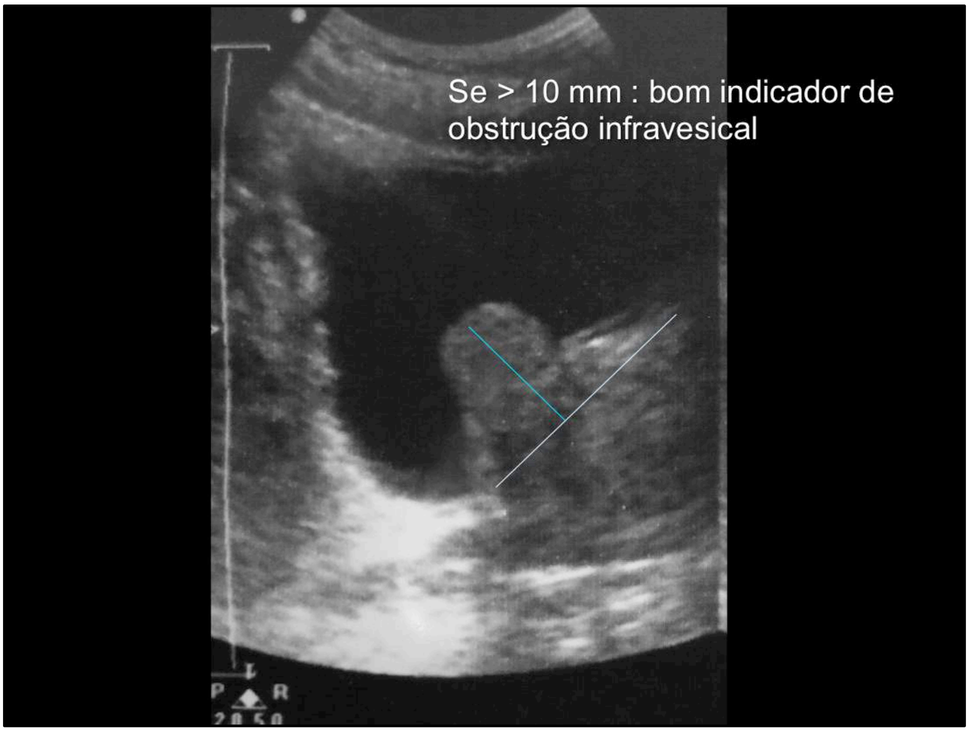
Cintilografia com DMSA evidenciando diminuição da função tubular no mesmo paciente, portador de obstrução ureteral por vaso de localização anomala no polo inferior do rim direito



Cintilografia com DTPA evidenciando diminuição da função tubular no mesmo paciente, portador de obstrução ureteral por vaso de localização anomala no polo inferior do rim direito



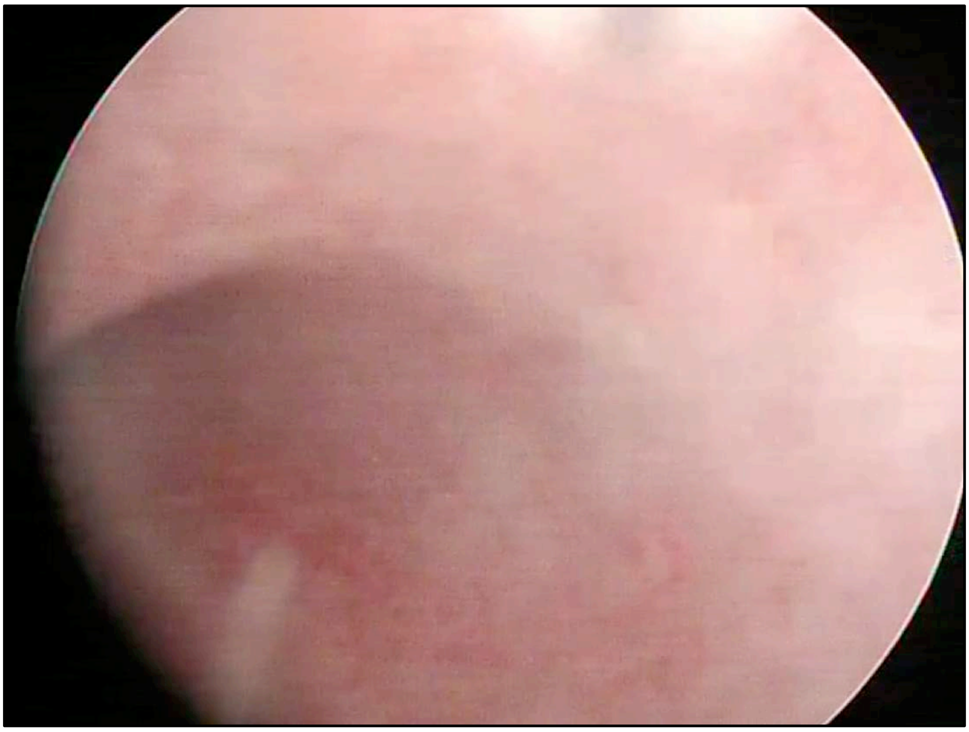
Video ilustrando pieloplastia laparoscopica em portador de obstrução ureteral por vaso de localização anomala no polo inferior do rim direito



US evidenciando protrusão prostática intravesical, que corresponde a um dos critérios imagenológicos para diagnóstico de obstrução infravesical secundária ao aumento prostático benigno.

Exemplos: aumento prostático

Este video ilustra uma das várias técnicas endoscopicas disponíveis para tratamento do aumento prostático benigno. Todas as tecnicas baseiam-se na remoção ou ablação do tecido hipertröfico. No exemplo apresentado o equipamento empregado é um bisturi de plasma.



Exemplo de uretrotomia endoscopica. Trata-se de uma técnica para tratamento de estenose da uretra peniana ou bulbar de curta extensão. Nela utiliza-se uma faca, chamada de faca de Sacshe, para realizar uma incisão na posição corresponde às 12 horas do relógio, a fim de abrir da uretra.

International Prostate Symptom Score (I-PSS)

Patient Name: ____________________ **Date of birth:** _______ **Date completed** _______

In the past month:	Not at All	Less than 1 in 5 Times	Less than Half the Time	About Half the Time	More than Half the Time	Almost Always	Your score
1. Incomplete Emptying How often have you had the sensation of not emptying your bladder?	0	1	2	3	4	5	
2. Frequency How often have you had to urinate less than every two hours?	0	1	2	3	4	5	
3. Intermittency How often have you found you stopped and started again several times when you urinated?	0	1	2	3	4	5	
4. Urgency How often have you found it difficult to postpone urination?	0	1	2	3	4	5	
5. Weak Stream How often have you had a weak urinary stream?	0	1	2	3	4	5	
6. Straining How often have you had to strain to start urination?	0	1	2	3	4	5	
	None	1 Time	2 Times	3 Times	4 Times	5 Times	
7. Nocturia How many times did you typically get up at night to urinate?	0	1	2	3	4	5	
Total I-PSS Score							

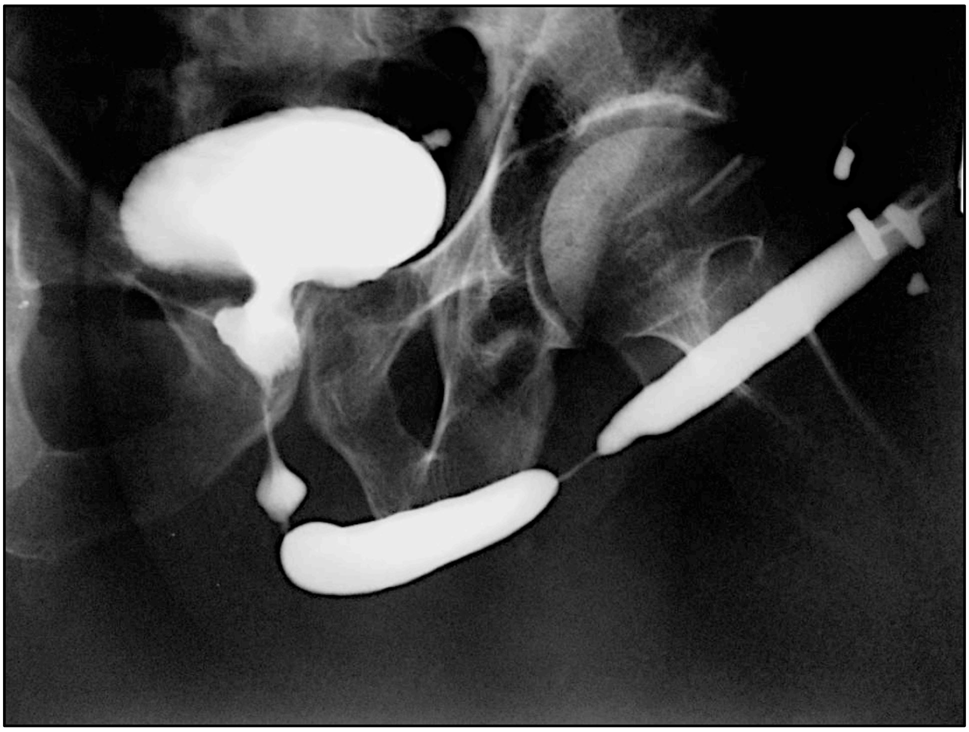
Score: 1-7: *Mild* 8-19: *Moderate* 20-35: *Severe*

Quality of Life Due to Urinary Symptoms	Delighted	Pleased	Mostly Satisfied	Mixed	Mostly Dissatisfied	Unhappy	Terrible
If you were to spend the rest of your life with your urinary condition just the way it is now, how would you feel about that?	**0**	**1**	**2**	**3**	**4**	**5**	**6**

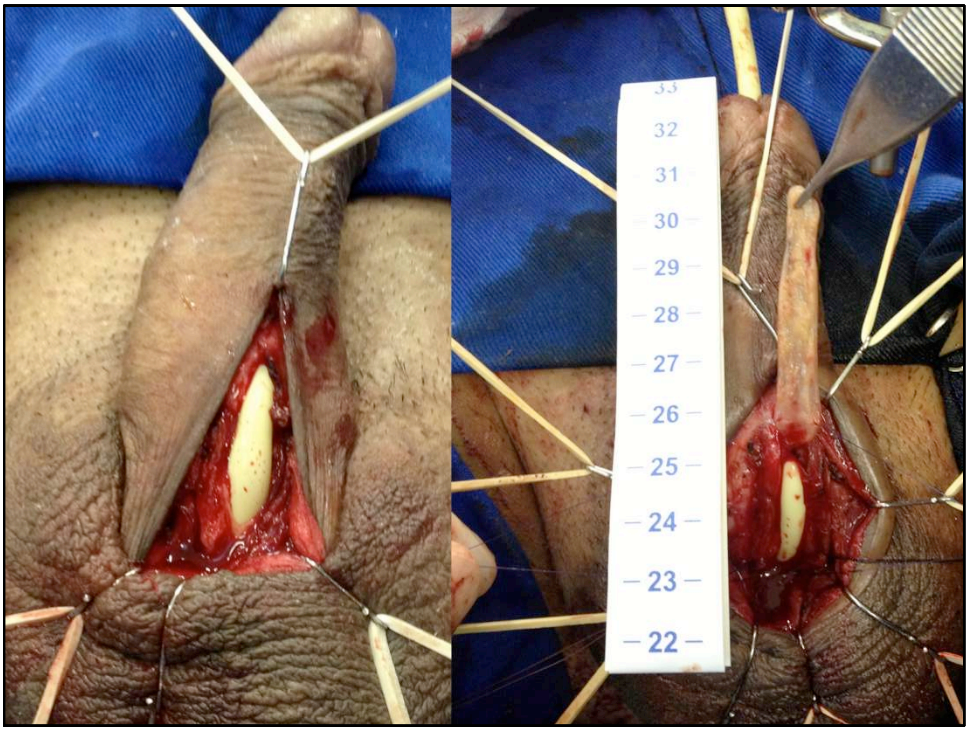
IPSS: 27

QV: 6

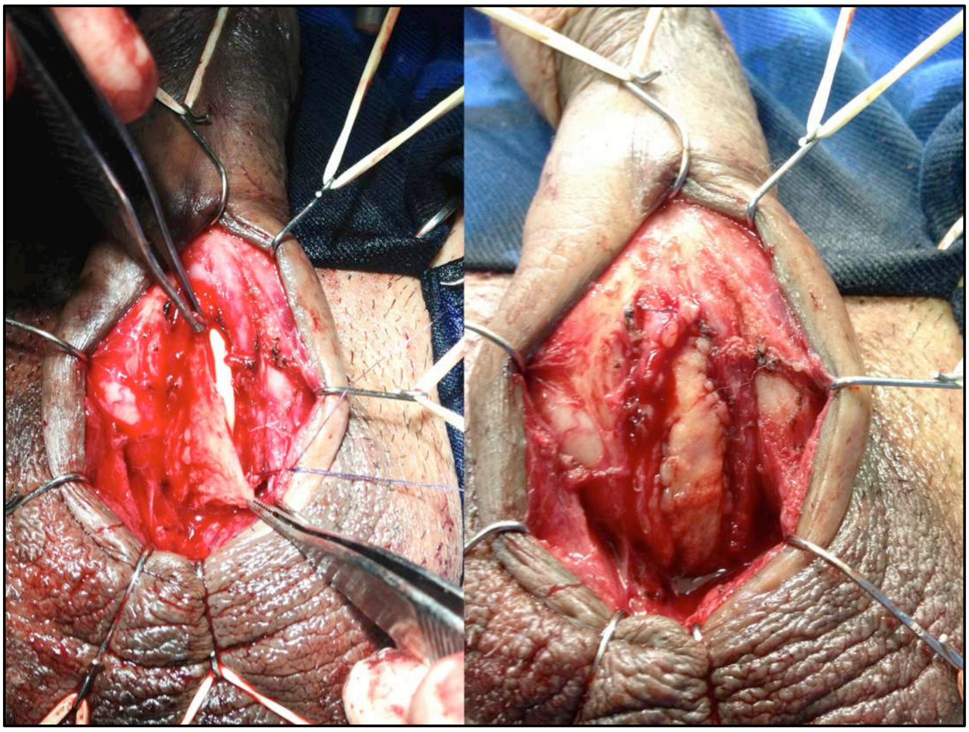
Escala internacional de sintomas prostáticos, que é empregada para a graduação de todos os pacientes com sintomas do trato urinário inferior do sexo masculino.



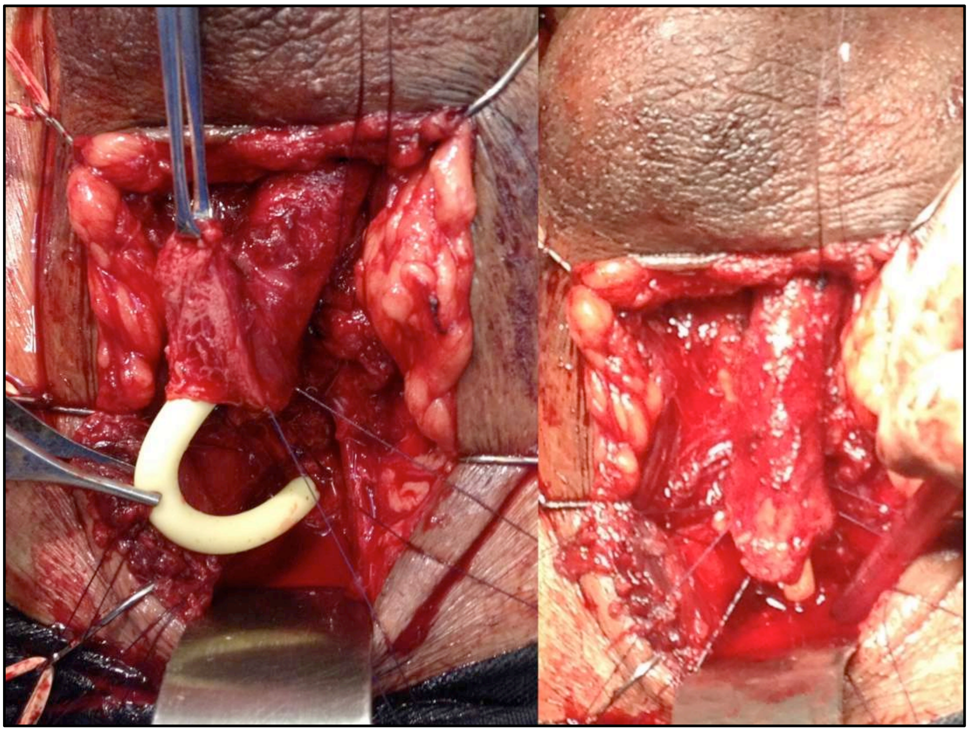
Uretrografia retrograda evidenciando uretra prostática aberta (com sinais de ressecção endocopica prévia, e duas estenoses, localizadas na uretra bulbar e peniana.



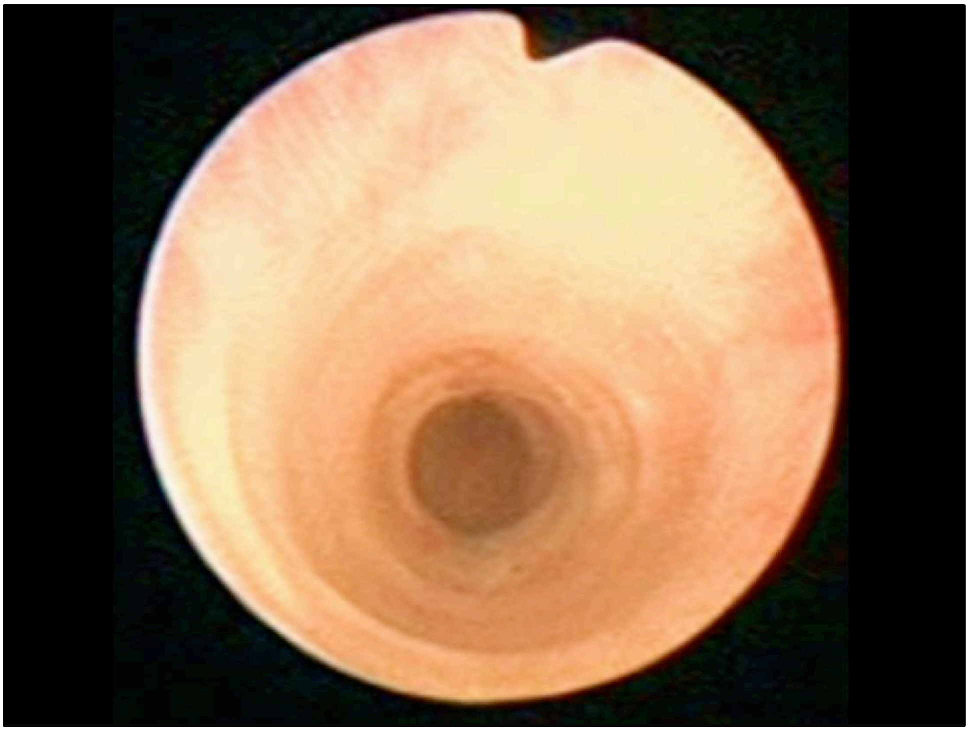
Uretroplastia com enxerto de mucosa oral do proprio paciente para tratamento de estenose de uretra bulbar.



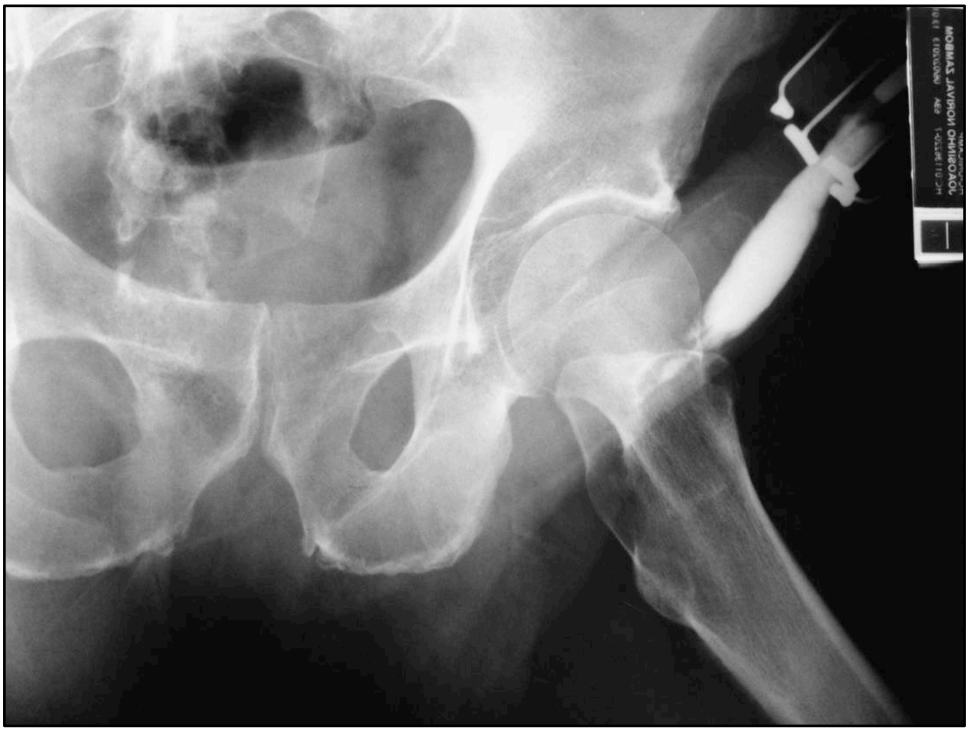
Uretroplastia com enxerto de mucosa oral do proprio paciente para tratamento de estenose de uretra bulbar.



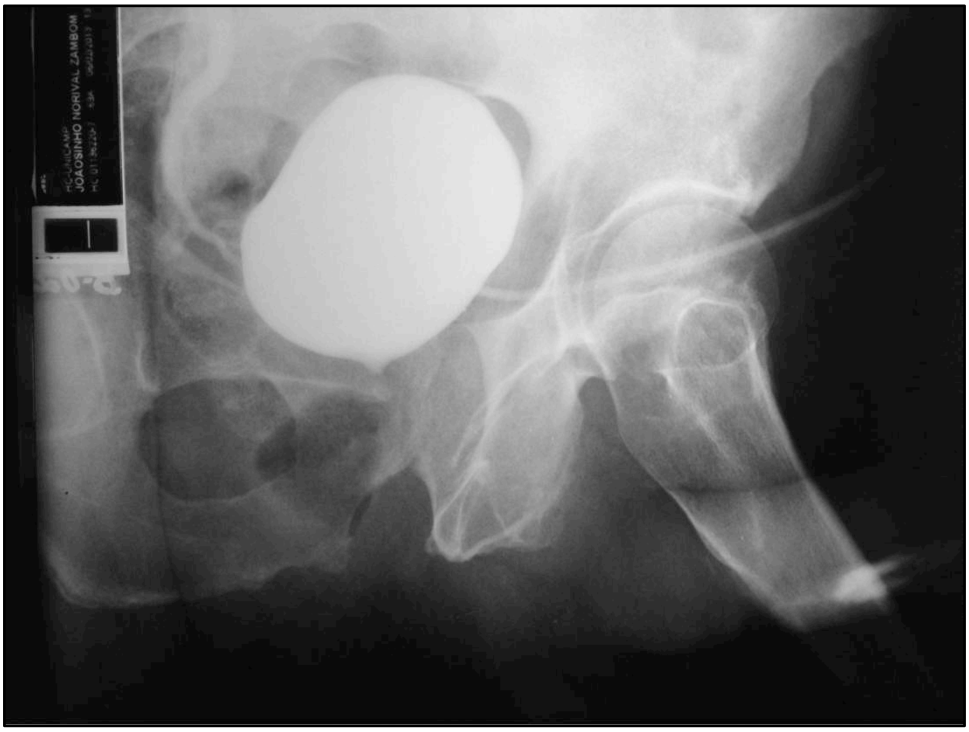
Uretroplastia com enxerto de mucosa oral do proprio paciente para tratamento de estenose de uretra bulbar.



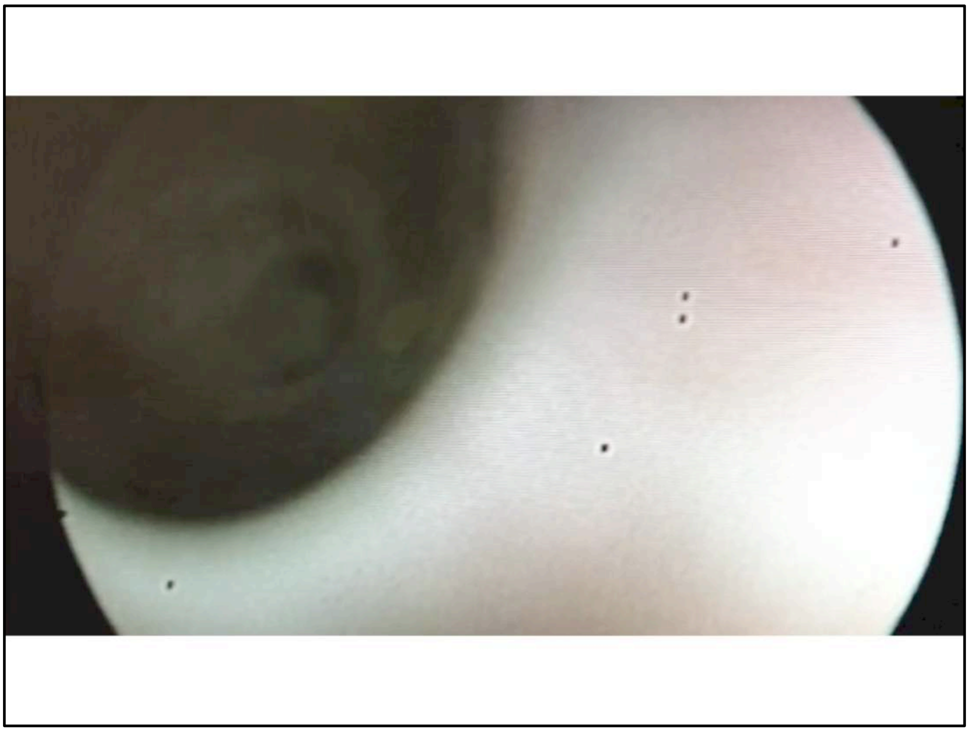
Uretroplastia com enxerto de mucosa oral do proprio paciente para tratamento de estenose de uretra bulbar. Aspecto edoscopico tardio.



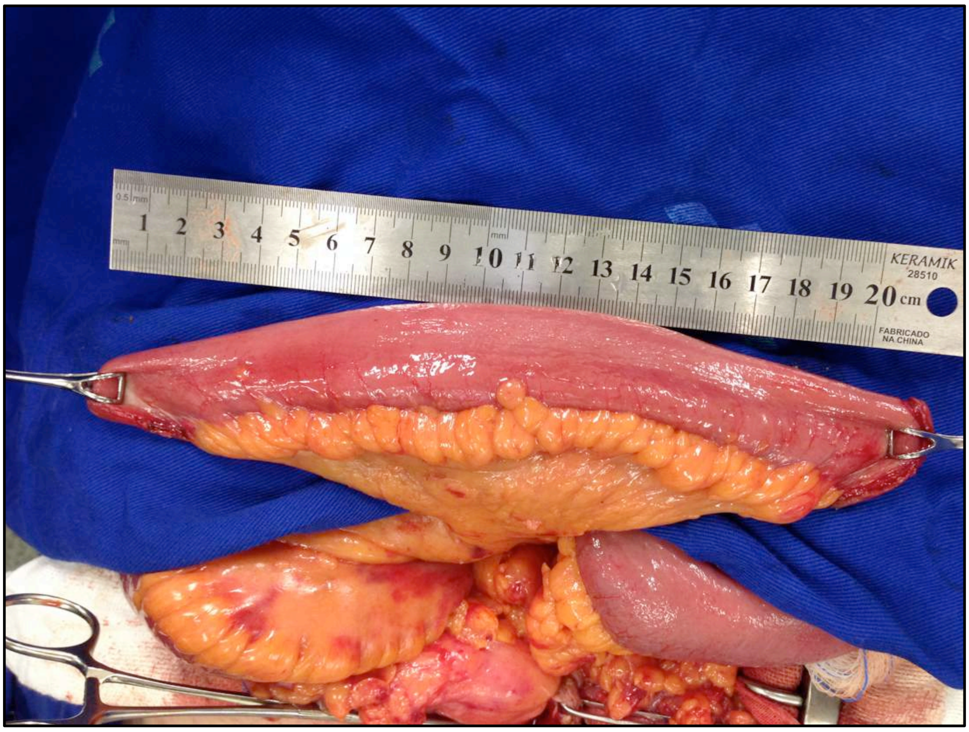
Uretrografia evidenciando estenose uretral extensa e completa.



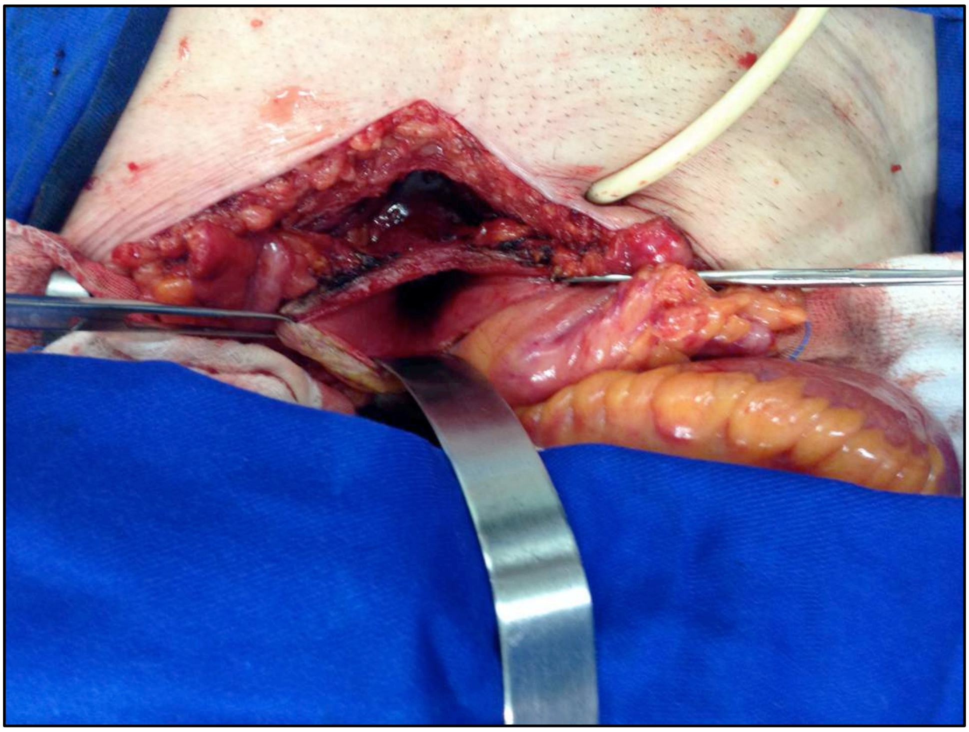
Mesmo paciente da imagem anterior, no qual foi realizada uma CISTOSTOMIA suprapúbica para derivação urinária, em virtude de estenose uretral completa.



Exemplo de estenose uretral grave. No presente caso o acesso à bexiga foi realizado por meio de uma cistoscomia suprapubica. Observe as alterações da parede verical decorrentes da obstrução/infecção.



Exemplo de derivação urinária definitiva em paciente com obstrução uretral completa. Trata-se de conduta de excessão, nos raros casos nos quais não há condições de reconstrução uretral. No exemplo a técnica denomina-se ileovesicostomia cutânea incontinente. Há inumeras técnicas de derivação urinária definitiva, e a seleção da técnica dependerá das caracterisitcas do(a) paciente.



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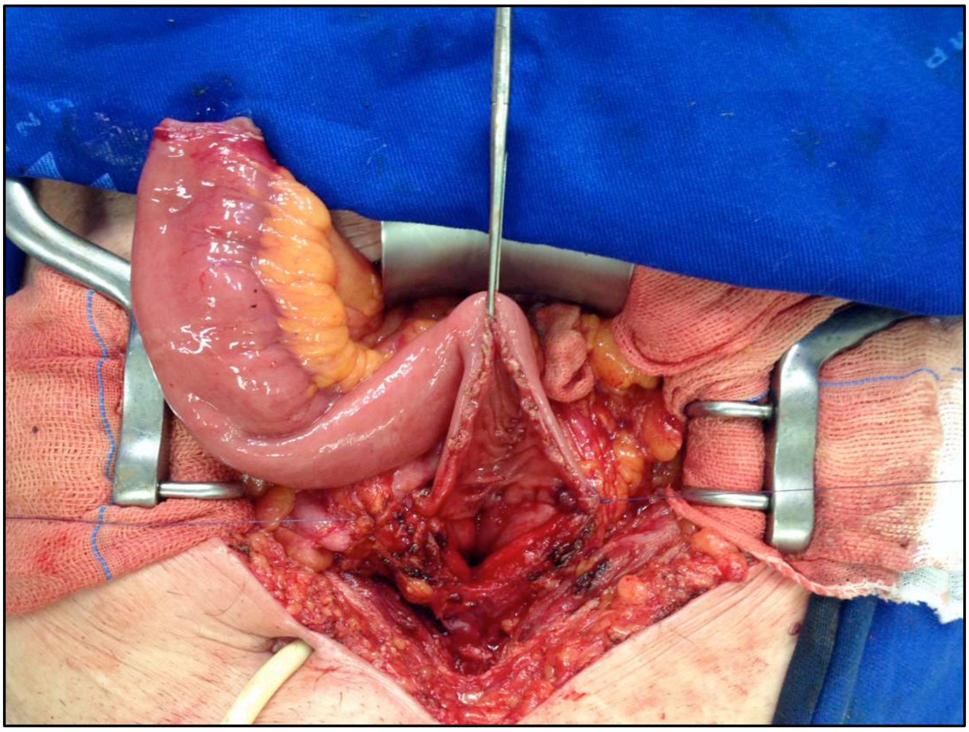

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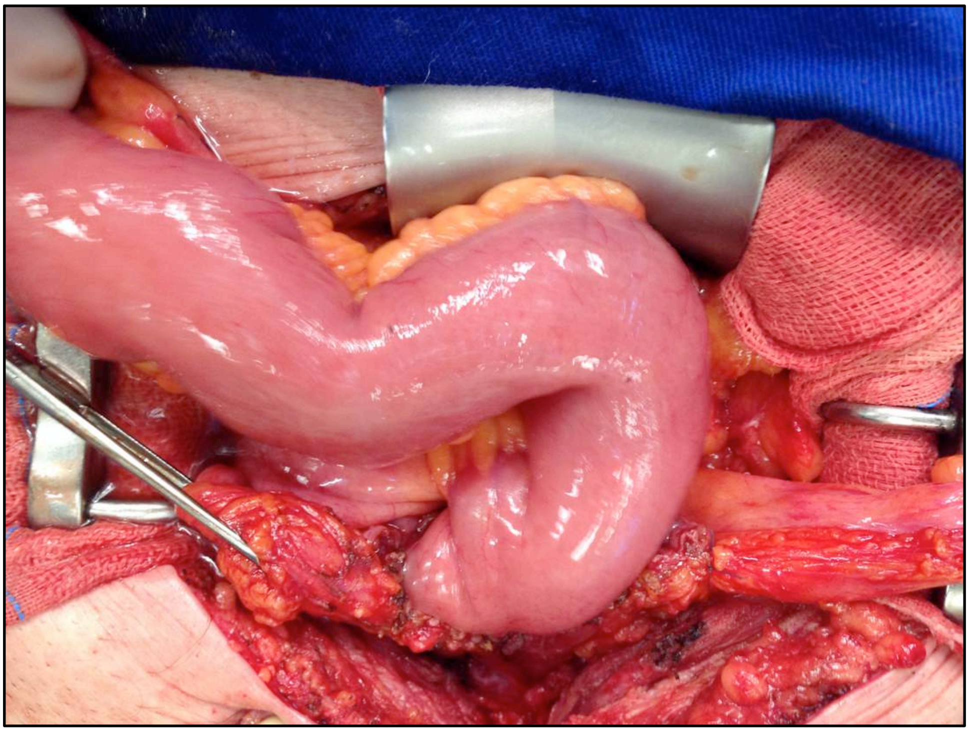

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Perguntas ?

Muito obrigado

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