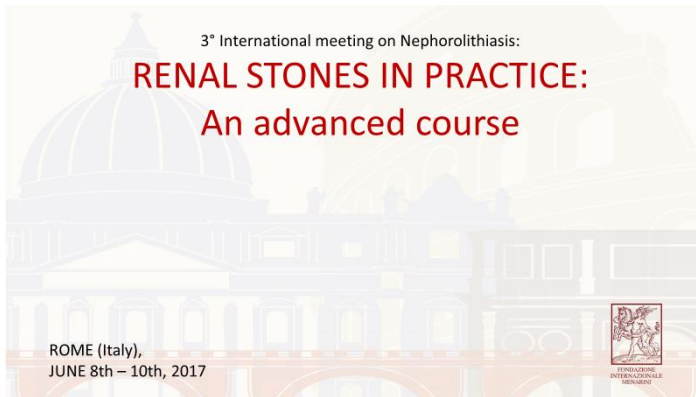


**3rd International Meeting on Nephrolithiasis:
RENAL STONES IN PRACTICE:
an advanced course
Rome (Italy), June 08-10, 2017
Highlights**

Introduction



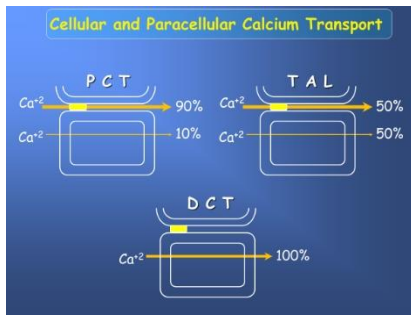
Prof. Gambaro and Prof. Croppi, chairmen of the symposium, opened the congress, by highlighting the high scientific level of this meeting, dedicated to Nephrolithiasis and renal stones in practice, from the nephrologist and the urologist point of view. Prof. Gambaro briefly explained the structure of this symposium finalized to a wide discussion and consensus between urologists and nephrologists on the main

topics of this disease. The congress has been attended by many of the top researchers of this field coming from Italy, other European and extra-European countries, together with many young physicians attending the University of Rome.

To follow the presentations of this congress, click on the link below:

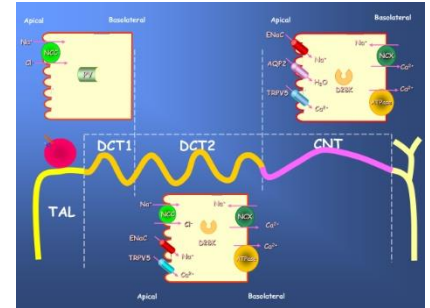
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Renal physiology and tubular disorders significant in nephrolithiasis

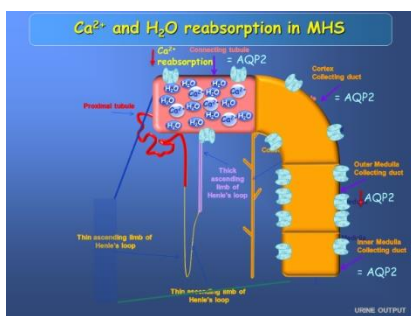


Renal physiology and tubular disorders significant in nephrolithiasis, was the topic discussed by Prof. Capasso in his lecture. The speaker, coming from Naples (IT), went deeper in his talk and presented very interesting data on the transport of calcium along the nephron with particular attention to its cellular and paracellular transport. In the main part of his lecture Prof. Capasso talked about the relationship between hypertension, hypercalciuria and

the onset of the stone disease and presented very interesting and unpublished data based on an animal model, the so-called Milano Hypertensive Rats in order to explain the role played by duodenum and vit. D in the calcium homeostasis. In the second part of his presentation, Prof. Capasso spoke about the phosphate renal excretion and



presented very interesting data on the role played by NaPi IIa in this pathway. More in particular the speaker talked about the role played by the extracellular calcium sensing receptors in the MHS aquaporin 2 down regulation leading to the higher urine excretion. In conclusion, Prof. Capasso pointed out that the MHS animals do not form renal stones, despite urinary calcium wasting and hyperphosphaturia, perhaps as a result of their significant polyuria and of the renal CaSR role in both processes.

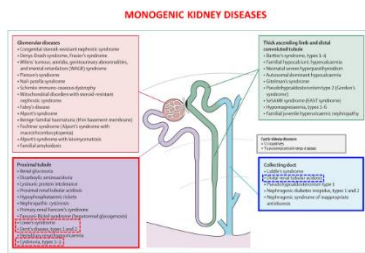


- What is the relationship between sodium and calcium in the calcium nephron pathway?
- Why hypertension is associated with hypercalciuria?
- Why the hypertensive subjects have a higher rate of stone disease?
- What's about the role of Calbindin based on the data presented by the speaker?
- What is the aquaporin distribution along the nephron, based on the data presented by the speaker?
- What's about the role played by NaPi IIa in the phosphate excretion, based on the data presented by the speaker?
- Why aquaporin 2 are downregulated in MHS, based on the data presented by the speaker?

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Functional studies of the tubule and diagnosis of tubular disorders in nephrolithiasis



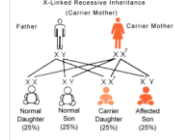
Over 200 genes are known to be associated with monogenic renal disorders

Prof. Unwin from London (UK), spoke about Functional studies of the tubule and diagnosis of tubular disorders in nephrolithiasis. The speaker talked about the variety of exchangers and coupled transporters and channels and on the monogenic kidney diseases. Going deeper in his lecture, Prof. Unwin, presented very interesting data on the more common stone types and talked about Cystinuria, its

historical landmarks, pathogenesis, diagnosis, complications and management. The speaker talked also about a very interesting clinical case of a 36 yo female affected by non-genotyped Cystinuria. In the main part of his lecture, Prof. Unwin presented very interesting data on the DENT 1 and 2

DENT 1 and 2 DISEASE & LOWE SYNDROME

- Variants of inherited renal Fanconi syndrome:
- ✓ Hypophosphataemia
 - ✓ Aminoaciduria
 - ✓ +/- glycosuria
 - ✓ Hypouricaemia
 - ✓ Bicarbonate wasting
 - ✓ Low molecular weight proteinuria
 - ✓ Hypercalciuria
 - ✓ Rickets
 - ✓ Growth retardation
 - ✓ (Renal failure)



- Dent & Friedman 1964
- Wrong 1994 coined 'Dent's disease' (Scheinman XRN and XLRH = Dent's) – CLCN5
- Oculocerebrorenal syndrome of Lowe 1952
- OCRL (NPP5F, inositol polyphosphate – regulates PIP2) – Attree 1992; Nussbaum 2002

disease and on the Lowe syndrome as variants of the inherited renal Fanconi syndrome. More in particular, the speaker described the main transport alterations at the proximal tubule level,

based on genetic analyses. Finally, Prof. Unwin spoke about the renal tubular acidosis (RTA) and presented very interesting data on the simplified models responsible for the tubular acidosis and on the main underlying gene mutations. The speaker talked also about the differential diagnosis between the complete and the incomplete RTA and about complications and treatment, with a particular attention to bone loss and osteoporosis. In conclusion, Prof. Unwin pointed out that behind every thin bone might be a distal

BONE TURNOVER AND LOSS IN PATIENTS WITH dRTA AND STONES

	Stone formers with dRTA	Stone formers without dRTA
(serum)	OSTEOCALCIN	osteocalcin
(urinary)	HYDROXYPROLINE	hydroxyproline

Increased bone turnover in dRTA stone formers

(Daher et al., Urol Res, 1993)

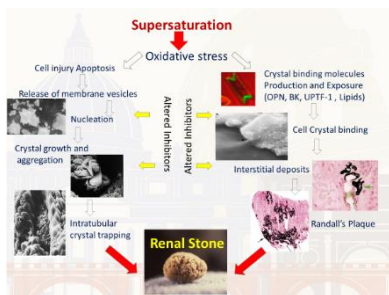
RTA.

- What are the more common stone types presented by the speaker?
- What are the methods for measurements of cystine, presented by the speaker?
- What are the main genetic mutations responsible for the Dent 1 and 2 diseases, based on the data presented by the speaker?
- What are the main mutations responsible for the onset of the renal tubular acidosis, presented by the speaker?
- What's about the RTA classification presented by the speaker?
- What are the main tests for the RTA diagnosis, presented by the speaker?
- What's about the alkali therapy from the speaker point of view?

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Urinary physico-chemistry in nephrolithiasis and its investigation



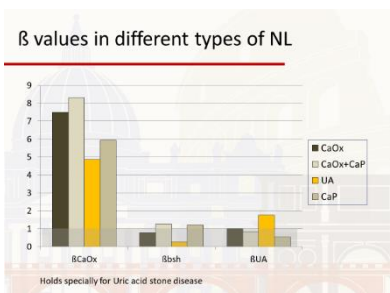
The speaker presented also very interesting data on the main pathways leading to the renal stone formation starting from the supersaturation and the related oxidative stress. In the main part of his lecture, the speaker talked about saturation, its definition, calculation and its principles. Prof. Marangella,

Urinary physico-chemistry in nephrolithiasis and its investigation, was the topic Prof. Marangella spoke about in his lecture. The speaker coming from Turin (IT), started his talk, by presenting very interesting data on supersaturation and stone disease. Going deeper in his lecture, Prof. Marangella, talked about the correlation between saturation, uric acid and cystine. The

presented very interesting data on Lytho risk and its new version Lytho risk 2, that is a software developed for the calculation and the display of the nephrolithiasis risk profiles. In the second part of his presentation, the speaker talked about the state of saturation and its effects and presented very interesting data on the promoters and inhibitors of crystallization. Finally, Prof. Marangella talked about the procedures to be applied in patients and more in particular on the computer calculation.

The variables that concur to the state of saturation can be divided into:

- > **Strong**
 - Calcium, Magnesium, Oxalate, Citrate, Sodium, Phosphate, pH
- > **Weak**
 - Potassium, Chloride, Ammonium, inorganic Sulphate

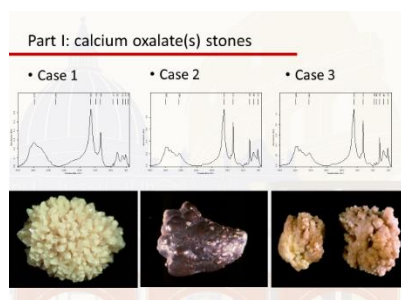


- Is supersaturation the main cause of kidney stone, based on the data presented by the speaker?
- What is the role played by calcium phosphate in the development of the renal stones, based on the data presented by the speaker?
- What are the main processes involved in Crystallization, based on the data presented by the speaker?
- What are the strong and the weak variables concurring to the state of saturation, based on the data presented by the speaker?
- What's about the state of saturation and its effects, based on the data presented by the speaker?
- What are the promoters and the inhibitors of the Crystallization process, based on the data presented by the speaker?

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Stone analysis and clinical correlations



Stone analysis and clinical correlations, was the topic discussed by Prof. Letavernier and Daudon. The speakers, coming from Paris (Fr), spoke about the stone analysis. In the first part in this lecture, Prof. Letavernier presented very interesting data on the calcium oxalates stones and Prof. Daudon on their possible different compositions. More in particular the speakers talked about three clinical cases

characterized by the presence of the same stones but with a different chemical composition despite a quite similar gas chromatographic profile. In the second part of this talk, Prof. Letavernier presented very interesting data given by two



other clinical cases of patients affected by type II diabetes the first and by Sjögren syndrome the second one, both of them with nephrolithiasis characterized by calcium phosphate stones. More in particular the speaker talked about aetiology and morphology, by highlighting the tight relationship between them. In conclusion, Prof. Letavernier pointed out that the morphological stone examination provides critical information regarding the stone origin and the underlying diseases.

calcium phosphate stone: aetiologies according to morphology

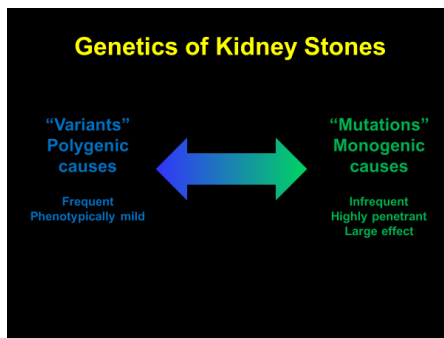
Etiology of stones (mainly composed of calcium phosphate)	n (%)	Type I/II	Type I/II
Idiopathic distal renal tubular acidosis	77 (100)	3 (3.9)	74 (96.1)
Digoxin syndrome	30 (100)	7 (23.3)	23 (76.7)
Carbonic anhydrase inhibitors (acetazolamide or topiramate)	52 (100)	50 (96.2)	2 (3.8)
Medullary sponge kidney	211 (100)	152 (72.0)	59 (28.0)
Primary hyperparathyroidism	93 (100)	93 (100.0)	0
Urinary tract infection	375 (100)	377 (99.5)	2 (0.5)
Idiopathic hypercalcaemia	298 (100)	298 (100.0)	0
Transparietal wall	23 (100)	23 (100.0)	0
Total	1091 (100)	951 (87.2)	140 (12.8)

- What's about the chemical composition of the calcium oxalates stones, based on the data presented by the speaker?
- What are the correlations between morphology and biology based on the data presented by the speaker?
- What are the main factors influencing the stone morphology, based on the data presented by the speaker?
- What's about the correlation between calcium phosphate stone formation and the metabolic syndromes from the speaker point of view?

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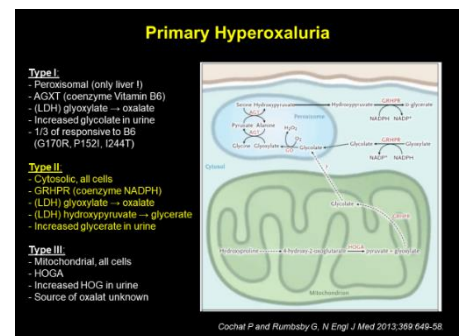
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Inherited disorders in nephrolithiasis and their diagnosis



The Inherited disorders in nephrolithiasis and their diagnosis, was the topic discussed by Prof. Fuster. The speaker, coming from Bern (CH), talked about heritability of nephrolithiasis and the genetics of the kidney stones. Going deeper in his lecture, Prof. Fuster presented very interesting data on the main genes responsible for the major "mutations" monogenic

causes of Nephrolithiasis. In the main part of his presentation, the speaker talked about a clinical case of a 55 yo patient affected by recurrent CaOx stones and presented very interesting data on the type II Primary Hyperoxaluria, its clinical presentation, diagnosis and therapy. More in particular Prof. Fuster highlighted the importance of a correct diagnosis and the related treatment, for the risk of the onset of the systemic oxaluria in these patients. In the second part of his lecture, the speaker talked about a case of familial hyperparathyroidism and presented very interesting data starting from genetics and highlighted that the interaction with geneticists was critical for the correct diagnosis formulation. Finally, Prof. Fuster presented very interesting data on a third clinical case, characterized by a 67 yo female affected by nephrocalcinosis and nephrolithiasis secondary to a parathyroid adenoma. Also in this case the diagnosis was possible thanks to the performance of the genetic analyses, the speaker pointed out.



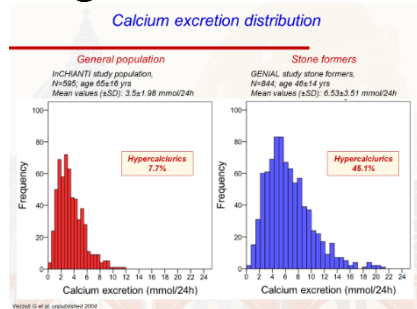
causes of Nephrolithiasis. In the main part of his presentation, the speaker talked about a clinical case of a 55 yo patient affected by recurrent CaOx stones and presented very interesting data on the type II Primary Hyperoxaluria, its clinical presentation, diagnosis and therapy. More in particular Prof. Fuster highlighted the importance of a correct diagnosis and the related treatment, for the risk of the onset of the systemic oxaluria in these patients. In the second part of his lecture, the speaker talked about a case of familial hyperparathyroidism and presented very interesting data starting from genetics and highlighted that the interaction with geneticists was critical for the correct diagnosis formulation. Finally, Prof. Fuster presented very interesting data on a third clinical case, characterized by a 67 yo female affected by nephrocalcinosis and nephrolithiasis secondary to a parathyroid adenoma. Also in this case the diagnosis was possible thanks to the performance of the genetic analyses, the speaker pointed out.

- What are the main monogenic causes of nephrolithiasis presented by the speaker?
- What's about the diagnosis of the 55 yo male with recurrent CaOx stones presented by the speaker?
- What's about the renal survival in PH type 1,2 and 3 based on the data presented by the speaker?
- What is the treatment of the PH type 2, based on the data presented by the speaker?
- What are the red flags for genetic diseases presented by the speaker?
- What's about the correct aetiology of the Fanconi Syndrome, considered as an idiopathic syndrome till now, based on the data presented by the speaker?
- What is the main difficulty in the recognition of the genetic causes of kidney stones, based on the data presented by the speaker?

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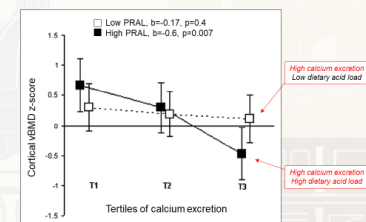
Mineral metabolism disorders in nephrolithiasis and their diagnosis



The Mineral metabolism disorders in nephrolithiasis and their diagnosis, was the topic of Prof. Vezzoli presentation. The speaker, coming from Milan (IT), talked about hypercalciuria starting from a clinical case running in his center. Going deeper in his speech, Prof. Vezzoli presented very interesting data on the correlation between BMD and the calcium excretion, by highlighting that higher is the calcium excretion, lower is the BMD more in particular at the spine and

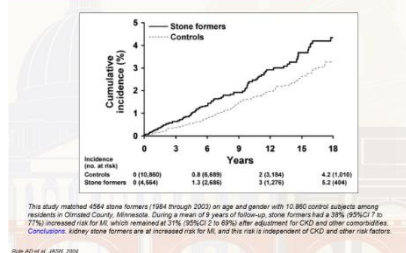
at the femoral level. In the main part of his lecture, the speaker talked about bone histology, sodium excretion, dietary acid and protein load intake and calcium absorption in hypercalciuric stone formers. Prof. Vezzoli presented also very interesting data on the genes associated with calcium stones and highlighted that hypercalciuria may be caused by a defect of the calcium transport expressed in bony and intestinal cells, leading to hyperabsorption of calcium and bone

Association of hypercalciuria with dietary acid load may lead to a decrease in bone mass



The study included 154 healthy children and adolescents. Potential renal acid load (PRAL) was determined according to urine nitrogen by subtracting measured quantitatively important mineral cations from nonabsorbable anions. Urinary calcium excretion was significantly associated with volumetric vBMD (P=0.04).

Kidney stones are associated with the risk of myocardial infarction



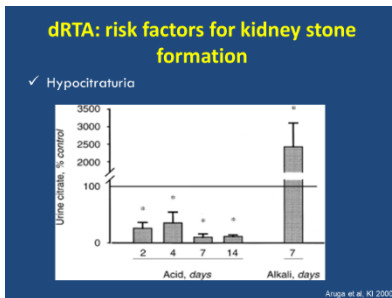
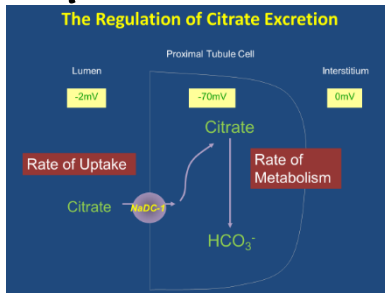
resorption. In the second part of his presentation, Prof. Vezzoli talked about the relationship between hypertension, CVDs and stone formers and presented very interesting data given by many clinical studies, all of them demonstrating the tight correlation between the calcium metabolism disorders and the cardiovascular diseases. In conclusion, Prof. Vezzoli pointed out that stone former patients may be predisposed to soft tissue calcification and this factor could explain the vascular calcification severity in these patients.

- What's about the correlation between the lumbar spine fracture incidence and the stone former condition, based on the data presented by the speaker?
- What are the main bone damages in stone patients presented by the speaker?
- What's about sodium excretion in hypercalciuric patients based on the data presented by the speaker?
- What are the main genes associated with calcium stones presented by the speaker?
- What are the main indications explaining the stone formation from genetic findings?
- What are the main factors predisposing to Randall's plaque formation, based on the data presented by the speaker?
- May tubular Ca-PO₄ crystals stimulate local cells to produce Randall's plaque?

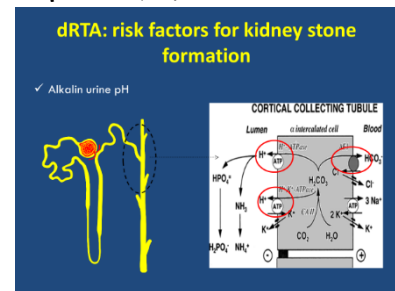
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Renal physiology and tubular disorders significant in Nephrolithiasis



Renal physiology and tubular disorders significant in Nephrolithiasis, was the topic of Dr. Capolongo presentation. The speaker, coming from Naples (IT), talked about Hypocalciuria and presented very interesting data on one of the main clinical conditions associated with hypocitraturia: the intracellular acidosis, given by a clinical case running in her center. Going deeper in her lecture Dr. Capolongo presented a lot of data on the dRTA diagnostic criteria and on its risk factors for the kidney stone formation. Finally, the speaker talked about the therapy applied to this patient, by highlighting that therapy is able to prevent the renal damage and the other systemic complications due to the metabolic acidosis.



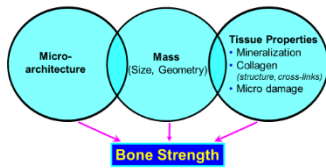
- What are the main clinical conditions associated with hypocitraturia, based on the data presented by the speaker?
- What are the main risks factors for kidney stone formations in patients affected by RTA, based on the data presented by the speaker?
- What's about the key points of the therapy from the speaker point of view?
- What are the dRTA main diagnostic criteria presented by the speaker?

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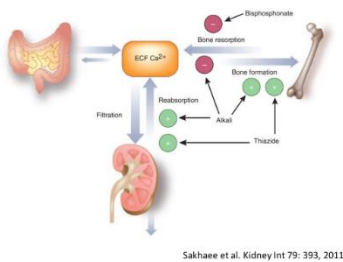
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Bone disease, nephrolithiasis and diagnosis

Determinants of Bone Strength

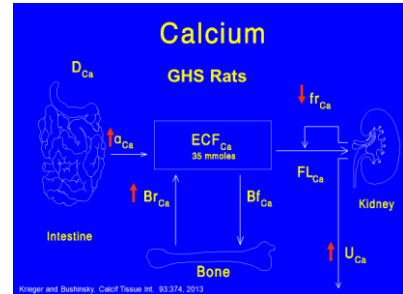


part of his lecture the speaker presented very interesting data on the correlation between hypercalciuria and bone, thanks to an animal model of hypercalciuric stone-forming (GSH) rats, feeding and living in controlled state for all their life. More in particular Prof. Bushinsky was able to demonstrate that in GSH



Sakhaee et al. Kidney Int 79: 393, 2011

Prof. Bushinsky coming from New York (USA) spoke about bone disease, nephrolithiasis and diagnosis and presented very interesting data on a clinical case of a male physician affected by bone disease and osteoporosis at only 62 years of age. Going deeper in his lecture Prof. Bushinsky talked about the determinants of bone strength related to the incidence of vertebral fracture in symptomatic urolithiasis patients. In the main



Krieger and Bushinsky. Calcif Tissue Int. 93:374, 2013

rats the osteoclast number and activity is increased compared to osteoblasts and that the BMD is decreased in this animal model compared to SD rats. In the second part of his presentation, the speaker talked about the effect of alkali and thiazides in patients affected by nephrolithiasis and presented very interesting data given by many animal and clinical studies demonstrating that all these drugs improve BMD in osteoporotics affecting by hypercalciuria.

- What's about the relative risk incidence of wrist fracture according to the kidney stone history in postmenopausal women, presented by the speaker?
- What's about the relationship between hypercalciuria and osteoporosis from the speaker point of view?
- What's about the main data produced by the use of primary cell cultures to determine the osteoblast and the osteoclast activity in GSH vs. SD rats?
- What's about the chlorthalidone effects on bone based on the data presented by the speaker?

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Nephrolithiasis of intestinal origin

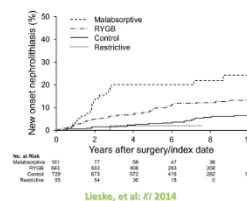
What are kidney stones made of?

	Number	Percent
Calcium oxalate	29,318	67.3%
Ca Phos (Apatite)	6996	16.1%
Uric Acid	3613	8.3%
Struvite	1316	3.0%
Artifact	1399	3.2%
Ca Phos (Brushite)	374	0.9%
Other	170	0.4%
Cystine	151	0.35%
Ammonium urate	105	0.2%
Drug	60	0.1%
Rare	1	

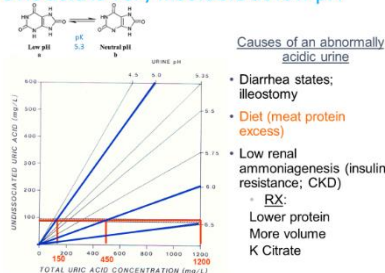
The Nephrolithiasis of intestinal origin, was the topic at the core of Prof. Lieske presentation. The speaker, coming from Rochester (USA), presented very interesting data starting from a clinical case of a 45 years old airline pilot affected by nephrolithiasis characterized by calcium oxalate monohydrate stones. Going deeper in his lecture, Prof. Lieke talked about the calcium oxalate urolithiasis risk factors and highlighted that the so called “idiopathic” hypercalciuria it is likely to be a “genetic”

one. In the main part of his presentation, the speaker talked about another clinical case characterized by a 58 years old mail patient affected by obesity treated with gastric bypass and by nephrolithiasis due to calcium oxalate stones with a very high oxalate concentration. Prof. Lieke presented very interesting data on the oxalate metabolism and highlighted the tight correlation between hyperoxaluria and fat malabsorption. More in particular the speaker talked about the oxalate dietary balance, its intestinal absorption and its control at the glomerular level. Prof. Lieke presented also very interesting data on the effects of the gastric bypass on the oxalate malabsorption, by highlighting that the bariatric surgery is a common cause of enteric hyperoxaluria and renal stone production. In the second part of his

Bariatric surgery a common cause of Enteric hyperoxaluria and stones



Uric acid is very insoluble at low pH



presentation, the speaker talked about the treatment for enteric hyperoxaluria and about the ideal diet. Prof. Lieke presented also very interesting data on the correlation between the oxalate metabolism, the oxalobacter formigenes and the stone risk level. Finally, the speaker starting from a third clinical case of a 64 years old male with a colostomy and affected by nephrolithiasis due to uric acid stones, presented very interesting data on the main causes of an abnormally acidic urine concentration.

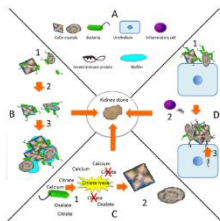
- What are the main calcium oxalate urolithiasis risk factors, based on the data presented by the speaker?
- What is the conservative dietary treatment for genetic CaOx stones?
- What is the relevant oxalate concentration in hyperoxaluric patients, based on the data presented by the speaker?
- What is the correlation between bariatric surgery and enteric hyperoxaluria, based on the data presented by the speaker?
- What’s about the comparison between obese control and post bariatric patients for the risk of new CKD, based on the data presented by the speaker?
- What is the treatment for hyperoxaluria presented by the speaker?
- What are the main oxalate-degrading bacteria, based on the data presented by the speaker?

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Nephrolithiasis and urinary tract infections

Speculative mechanisms for bacterial contribution to CaOx stones



Barr Beare PLoS ONE 2015

Prof. Tasca from Vicenza (IT), spoke about Nephrolithiasis and urinary tract infections and presented very interesting data starting from a 47 years old male with a radical cystectomy and camey 2 procedure, affected by a recto-urinary fistula causing lithogenesis. In the main part of his lecture, Prof. Tasca talked about the struitive stones composed by magnesium ammonium phosphate and carbonate

apatite, the main steps of stone formation, the urea splitting organisms, the patients clinical profile, its epidemiology and the interactions between bacteria and CaOx kidney stones. Prof. Tasca, presented also another clinical case of a 25 years old male patient treated with a bodycast for spinal fractures, ventricular-peritoneal bypass and hypercalciuria and spoke about the evaluations to be performed. More in particular the speaker presented very interesting data on three possible treatment options, like ESWL, PCNL or open surgery. The third clinical case presented by Prof. Tasca, was about a 76 years old male with a previous right nephrectomy for renal stones, this patient was also affected by type 2 diabetes, urinary tract

MANAGEMENT OF STAGHORN CALCULI

- Evidence suggests that pts. with staghorn calculi are best managed with PCNL based therapy either as a single technique or in combination with ESWL
- If combination therapy is undertaken (with ESWL) PCN nephroscopy should be the last procedure
- Low SFR have been reported for comb. approaches where ESWL was the last combination procedure

AUA guidelines, J Urol 2005

★ LONG-TERM RENAL FATE AND PROGNOSIS AFTER STAGHORN CALCULUS MANAGEMENT

Renal deterioration

Kidney status:			
Solitary	13	10 (77)	<<0.001
Nonsolitary	99	21 (21)	
Stone burden:			
Partial staghorn	32	4 (13)	0.02
Complete staghorn	80	27 (34)	
Hydronephrosis:			
Present	10	3 (30)	0.86
Absent	102	28 (28)	
Neurogenic bladder:			
Yes	30	14 (47)	0.006
Normal	82	17 (21)	
Diversion status:			
Diverted	26	15 (58)	<<0.001
Not diverted	86	16 (19)	

Long-term renal preservation in the staghorn calculus patient may depend on kidney status, staghorn size, absence of diversion or voiding dysfunction and complete stone eradication

Teichman J Urol 1995

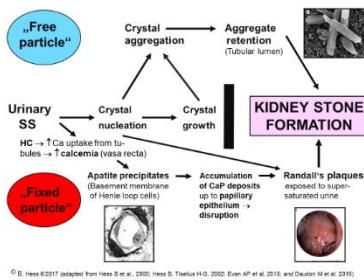
infection and presented a staghorn stone in the single left kidney with pyelectasis. The speaker talked about the possible treatment, by highlighting the rate of positive outcome compared to that one of no treatment. In conclusion, Prof. Tasca, pointed out that the best management of patients with staghorn stones is the percutaneous nephrolithotomy.

- What's about the mechanisms for the bacterial contribution to CaOx stones, based on the data presented by the speaker?
- What's about the urine microbiology in pre PCNL patients, based on the data presented by the speaker?
- Is it necessary the metabolic evaluation in patients with infected nephrolithiasis, based on the data presented by the speaker?
- What's about sepsis after PCNL based on the data presented by the speaker?
- What are the best predictors of urosepsis after PNCL, based on the data presented by the speaker?
- What's about the renal fate and prognosis after staghorn stone management, from the speaker point of view?

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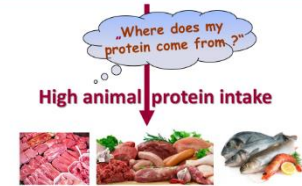
Idiopathic calcium nephrolithiasis



Idiopathic calcium nephrolithiasis, was the topic discussed by Prof. Hess from Zurich (CH), more in particular the speaker presented very interesting data on the basic steps of the kidney stone formation. Going deeper in his lecture, Prof. Hess talked about the main mechanisms leading to stone formation in the kidney, the diagnostic work-up and which analyses are mandatory, like the stone analysis, the blood and the urine analyses and the Uro-CT. In the main part of his lecture, the speaker presented very interesting data on the idiopathic

hypercalciuria, its pathogenesis, the main complications also linked with the need for a low calcium dietary intake, their consequences due to the high animal protein intake and the related metabolic complications. Prof. Hess talked also about mild hyperoxaluria, its relationship with hypercalciuria and about the oxalate metabolism. More in particular the speaker presented a clinical case of a 48 years old man affected by recurrent kidney stones despite the normal levels of oxaluria due to the increase of the calcium intake. The speaker pointed out that a high consumption of chocolate-wheat energy bars during exercise, that is rich in oxalate, was responsible for 3 more stone

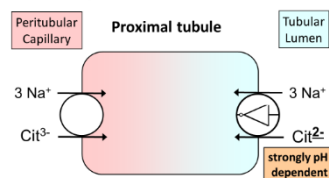
Low calcium diet - consequences



© B. Hess 2017 (various sources)

episodes. In the last part of his presentation, Prof. Hess talked about low volume, hypocitraturia and their correlation with kidney stone formation. Finally, the speaker presented very interesting data on prevention through the DASH diet application and on medications like thiazides, alkali-citrate and their side effects. In conclusion, the speaker pointed out that, together with diet also the changes in life style finalized to the reduction of the psychosocial stress is very effective in the

Urinary Citrate & Acid-Base



Example: tubular fluid pH drops from 7.4 to 6.9
 \Rightarrow 3-fold increase in $[\text{Cit}^{2-}]$ (i.e. \uparrow dietary acid)

© B. Hess, 2017 (from Hess B, in Hess M, Pfeiffer G, Karger M, eds. [eds] Urinary Tract Stone Disease, Chap. 14, Springer, London, 2011)

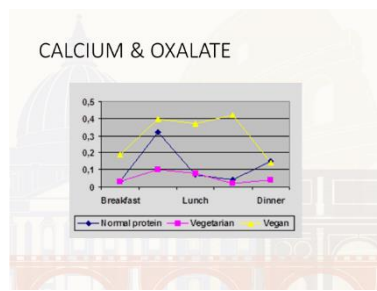
management of the idiopathic calcium nephrolithiasis.

- What is mandatory in the idiopathic calcium nephrolithiasis work-up, based on the data presented by the speaker?
- What are the main features found in recurrent idiopathic nephrolithiasis, based on the data presented by the speaker?
- What's about the idiopathic hypercalciuria pathogenesis based on the data presented by the speaker?
- What's about the calcium intake and the BMD level in idiopathic calcium nephrolithiasis patients?
- What's about the bone fracture risk in nephrolithiasis from the speaker point of view?
- Where does urinary Ox come from?
- What is the prevalence of hypocitraturia in kidney stone formers?

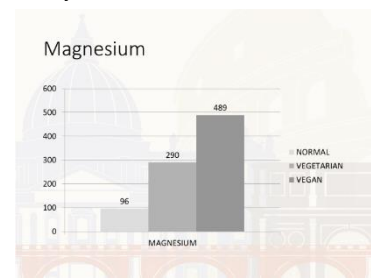
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Nutrition and nephrolithiasis and the nutritional investigation

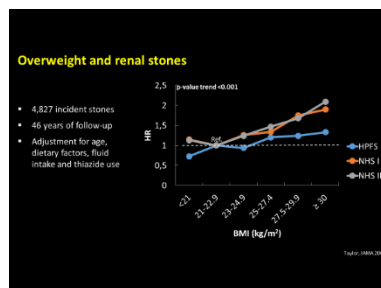


Prof. Ferraro from Rome (IT) and Prof. Trinchieri from Lecco (IT), spoke about Nutrition and nephrolithiasis and the nutritional investigation. More in particular, the speakers talked about the dietary assessment. Going deeper in this lecture, Prof. Trinchieri presented very interesting data on the methods used for the calculation of food and nutrient intake. More in particular

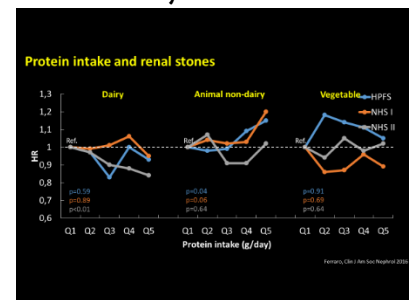


the speaker talked about the dietary recall, food frequency questionnaires, food screeners and their scores, urinary markers and about fluids. In the main part of his lecture, Prof. Trinchieri presented very interesting data given by clinical cases of three dietary regimens: the standard diet, the vegetarian and the vegan ones. The speaker discussed

the data related to the intake of protein, carbohydrates, fat and electrolytes like calcium, oxalate, magnesium, pral and vitamins. Prof. Ferraro spoke about the correlation between dietary factors and the renal stones. More in particular the speaker presented very interesting data on the dietary studies and the available cohorts like the channing cohorts, the women's Health Initiative and the cohort



of the Swedish Men. Going deeper in his lecture, Prof. Ferraro, spoke about the nutritional risk factors and presented very interesting data on the correlation between overweight, calcium, oxalate, sodium, animal proteins, fruit & vegetables, fructose, beverages, vitamins and renal stones.

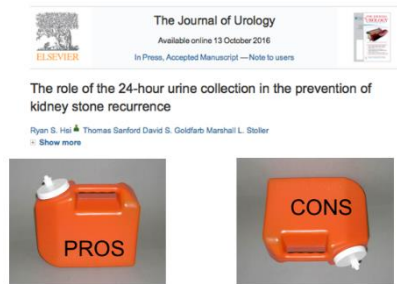


- What are the main characteristics of the dietary recall presented by the speaker?
- What's about the food frequency questionnaires, based on the data presented by the speaker?
- What are the main urinary nutritional markers presented by the speaker?
- Why is important to measure the alkali intake, based on the data presented by the speaker?
- What is the lowest protein diet among the three dietary regimens presented by the speaker?
- What are the key points of the correlation between overweight, calcium and uric acid stones?
- What's about the correlation between calcium and oxalate in the urinary oxalate excretion, based on the data presented by the speaker?
- What proteins are significantly correlated with the renal stone risk?

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Secondary prevention of nephrolithiasis



The Secondary prevention of nephrolithiasis, was the topic discussed by Prof. Goldfarb from New York (USA), more in particular the speaker talked about stones and their prevention. In the main part of his lecture, Prof. Goldfarb presented a very interesting clinical case of a 28 years old man affected by nephrolithiasis. More in particular the speaker talked about the assessment of risk, by highlighting the importance to collect the 24-hour urine in these patients. Prof. Goldfarb presented also very interesting data on the

AUA guidelines of the medical management of the kidney stones and on the role of the 24-hour urine collection in the prevention of the kidney stone recurrence. In the second part of his lecture the speaker talked about the treatment for patients with low urine volume and for patients with “occupational” kidney stones, by highlighting the importance to drinks beverages enriched with potassium citrate and to eat full calcium aliments. Prof. Goldfarb presented also very interesting data on treatment and more in particular spoke about thiazides and potassium citrate and presented very interesting data on the effect of allopurinol on stone recurrence and more in particular on febuxostat compared to allopurinol in the hyperuricosuria reduction. In the last part of his presentation, Prof. Goldfarb spoke about

What is the treatment for low urine volume?
What is the treatment for **occupational kidney stones**?



Potassium citrate:

- Surgeon: before going to the OR
- Baseball player: before going on the field
- Teacher: before class
- Cab driver: before getting into the taxi
- Steel worker: before entering work place
- Everyone: at bedtime

Summary: Calcium phosphate stones

- Fluids
- Na restriction
- More fruits and vegetables
- Citrate/thiazide

Calcium-Phosphate stones and presented very interesting data on prevention of recurrent Ca stone formation with potassium citrate therapy in dRTA. Finally, the speaker talked about acid uric stones, their etiology and treatment and presented very interesting data on alkaline therapy with citrate and diet. In conclusion, the speaker pointed out that the best therapy for the CaP stones is composed by fluids, NA restriction, more fruits and vegetable and the administration of citrate and/or thiazide.

- What is the cascade of care for stones based on the data presented by the speaker?
- What are the pro and cons of the 24-hour urine collection based on the data presented by the speaker?
- What is the treatment for low volume urine from the speaker point of view?
- What is the treatment for occupational kidney stones?
- What's about the empiric treatment for calcium stones presented by the speaker?
- There is any correlation between the citrate use and the shift from CaOx to CaP stone conversion, based on the data presented by the speaker?

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Urological treatment of renal stones

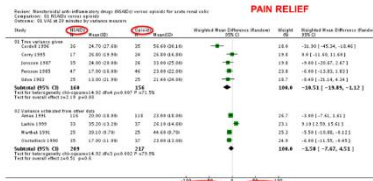
PAIN MANAGEMENT

Nonsteroidal anti-inflammatory drugs (NSAIDs) versus opioids for acute renal colic

Holdgate A, Pollock T, 2005.

THE COCHRANE LIBRARY

Independent, high quality evidence for health care decision making



Prof. Sarica from Istanbul (TR), presented very interesting data on the urological treatment of renal stones. More in particular the speaker talked about the relationship between the individual clinical expertise, the evidence based medicine, the patient's values and expectation and the best available clinical evidence. Going deeper in his lecture, Prof. Sarica presented the 2017 European association of Urology guidelines on pain management due to renal

colic. In the main part of his lecture, the speaker talked about the renal stones management, indications and methods for their removal. More in particular the speaker presented very interesting data on the shock wave lithotripsy, percutaneous nephrolithotomy, retrograde renal surgery and on the laparoscopic approach. Prof. Sarica talked also about the correlation between obesity and stones and the best methods to apply in these patients. The speaker presented very interesting clinical cases of symptomatic patients affected by non-lower pole stone, staghorn stones, symptomatic and asymptomatic calyceal stones. In the second part of his lecture, Prof. Sarica talked about ureteral stones, their treatment based on the proximal and the distal ureteral



Staghorn stones

Clinical Principle

- Should be removed if attendant comorbidities do not preclude treatment
 - Risk for deterioration of renal function
 - Loss of kidney
 - ESRD
 - Infectious complications
 - Mortality
- Older series more infection stones
- Newer series more metabolic stones



stone position and their size. More in particular the speaker presented data on the stone removal with the extra-corporeal SWL vs the ureteroscopic management, the medical expulsive therapy and on the residual fragments management. Finally, Prof. Sarica talked about stones in special conditions, in pregnancy, in transplanted kidney and in pediatric patients. In conclusion, the speaker pointed out the future stone treatment will be characterized by a personalized approach.

Residual Fragments



Moderate Recommendation
Evidence Level Grade C



- Residual fragments are NOT clinically insignificant
- Should offer endoscopic treatment to render pts stone free
 - Especially if infection stones suspected

Natural History, Complications and Re-Intervention Rates of Asymptomatic Residual Stone Fragments after Ureteroscopy: a Report from the EDGE Research Consortium

Ben H. Chew,*† Hilary L. Brotherton,‡ Roger L. Sur,‡ An Qi Wang,‡ Bodo E. Knudsen,‡ Courtney Yong,‡ Tracy Marien,‡ Nicole L. Miller,‡ Amy E. Krambeck,‡ Cameron Charchenko† and Mitchell R. Humphreys||

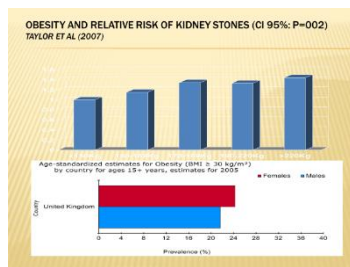


- What's about the effects of NSAIDs and Opioids for the treatment of renal colic pain management, based on the data presented by the speaker?
- What's about the treatment algorithm for renal stone treatment presented by the speaker?
- What's about the staghorn stones management for the speaker point of view?
- When to consider nephrectomy based on the data presented by the speaker?
- What is the best treatment for the asymptomatic non-obstructing calyceal stones?
- What's about the main indications for the symptomatic Lower Pole stones from the speaker point of view?
- What is the treatment algorithm for the ureteral stones therapy?
- What's about the residual fragments management from the speaker point of view?

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the cooperation between urologists and nephrologists

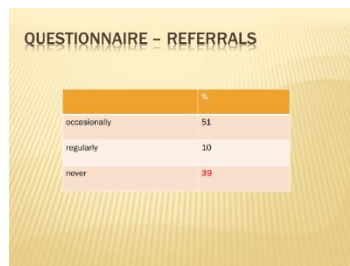


hyperoxaluria, nephrolithiasis and oxalate nephropathy. In the main part of his lecture, Prof. Buchholz presented very interesting data on the role of diet and life style in the treatment of kidney stones and on the environmental risk factors and spoke about

The main topic of Prof. Buchholz presentation, was the cooperation between urologists and nephrologists. The speaker, coming from Dubai (UEA), presented very interesting data on the new world-wide trends in kidney stone composition and its relationship with obesity. Going deeper in his lecture, Prof. Buchholz talked about bariatric surgery and its effects on bone and renal metabolism leading to

the main problems linked with investigation, diagnosis and treatment of the Ca stone formers. In the second part of his talk, the speaker presented very interesting data on a questionnaire running among urologists and highlighted that the results are indicative for the need to grow the collaboration between urologists and nephrologists for a better management of the stone former patients.

- "IDIOPATHIC" CA NEPHROLITHIASIS: CONDITIONS TO BE RULED OUT
1. Hyperparathyroidism
 2. Hyperthyroidism
 3. Sarcoidosis
 4. Vitamin D excess
 5. Calcium supplements
 6. immobilization
 7. Clinical evidence of BD
 8. Malignant neoplasms
 9. Distal RTA
 10. MSK
 11. Primary hyperoxaluria
 12. Enteric hyperoxaluria
 13. Bowel disease
 14. Chronic pancreatitis
 15. Vitamin C supplements
 16. Chronic diarrhea
 17. Lithogenic drugs
 18. Urinary infection
 19. Gouty diathesis
 20. Cystinuria
- © Nephrol Dial 2007; 16(2): 155-159



- How to investigate a calcium stone former from the speaker point of view?
- What the physicians have to do in advising stone former patients on prevention or treatment?
- How to make a correct diagnosis on the stone chemical composition?
- What are the main mechanisms of the coronary microvascular dysfunction presented by the speaker?
- What are the main characteristics of a CaOx stone former from the speaker point of view?
- What about the questionnaire to urologists presented by the speaker?

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These are only some of the topics addressed in the congress's sections

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