7° CONGRESSO NAZIONALE

FIMMG® Federazione Italiana Medici di Famiglia

Metis® DI MEDICINA GENERALE SOCIETÀ SCIENTIFICA DEI MEDICI

La Medicina Generale oltre la pandemia

5-10 ottobre 2020-Villasimius (CA)

GIOVEDÍ 19 DICEMBRE 2019

Ore 10.30

Giovanna è una vostra paziente di 65 anni

Dal 04/06/2013 affetta da DM2

-Nel 2005 intervento di QUART successiva radioterapia e terapia con anastrazolo terminato nel 2010

- Da quando aveva 55 anni affetta da disturbo d'ansia generalizzato

- Storia di coliche renali bilateralmente

La sua terapia al domicilio è la seguente:

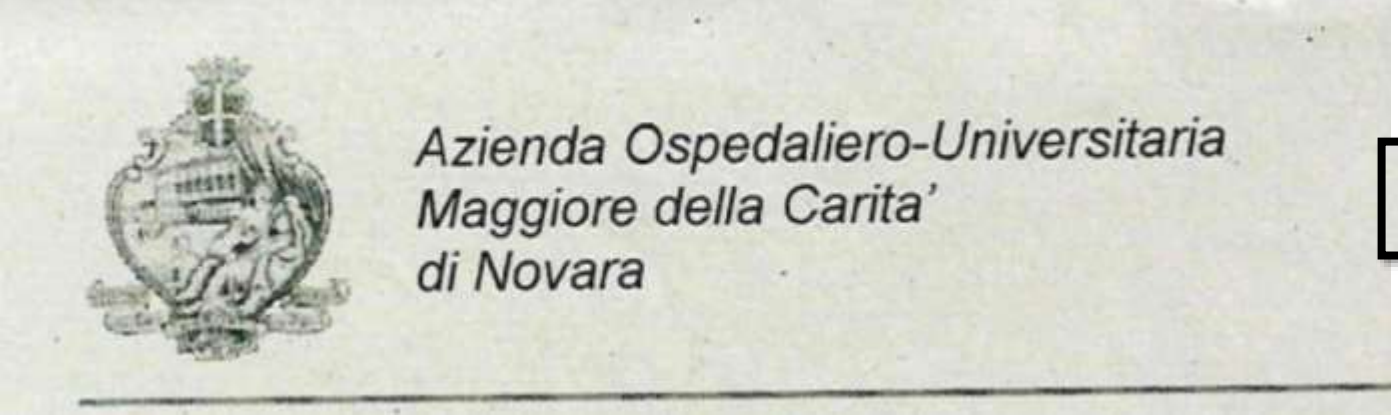
-Slowmet 1000 mg 1/die
-Paroxetina 20 mg 1/die
-Simvastatina 20 mg 1/die
-Idroclorotiazide 25 mg 1/die

Peso: 100Kg

Altezza: 153

BMI: 42,7

Circonferenza vita: 131 cm



Azienda Ospedaliero-Universitaria
Maggiore della Carita'
di Novara

Novara, 19-03-2019

Giovanna che permane libera da malattia. riferisce calo ponderale di 5 kg che francamente non sono in grado di valutare.
obbiettivamente permane importante obesità addominale che rende impossibile la valvìutazione di fegato e milza o la presenza di eventuali masse addominali. riferisce comunque alvo regolare.

ho preso visione di una MOC patologica ma è già programmata una visita presso il centro per osteoporosi.

VISITA ONCOLOGICA DI CONTROLLO

Please answer the questions below to calculate the ten year probability of fracture with BMD.

Country: Italy Name/ID: GIOVANNA About the risk factors

Questionnaire:

1. Age (between 40 and 90 years) or Date of Birth

   Age: 65 Date of Birth: Y: 1955 M: 04 D: 10

2. Sex — ○ Male ● Female

3. Weight (kg) — 100

4. Height (cm) — 153

5. Previous Fracture — ● No ○ Yes

   Fractured Hip — ● No ○ Yes

   t Smoking — ● No ○ Yes

   orticoids — ● No ○ Yes

9. Rheumatoid arthritis — ● No ○ Yes

10. Secondary osteoporosis — ● No ○ Yes

11. Alcohol 3 or more units/day — ● No ○ Yes

12. Femoral neck BMD (g/cm^2)

T-Score -1.3

Clear Calculate

BMI: 42.7

The ten year probability of fracture (%)

with BMD

Major osteoporotic	4.3
Hip Fracture	0.6

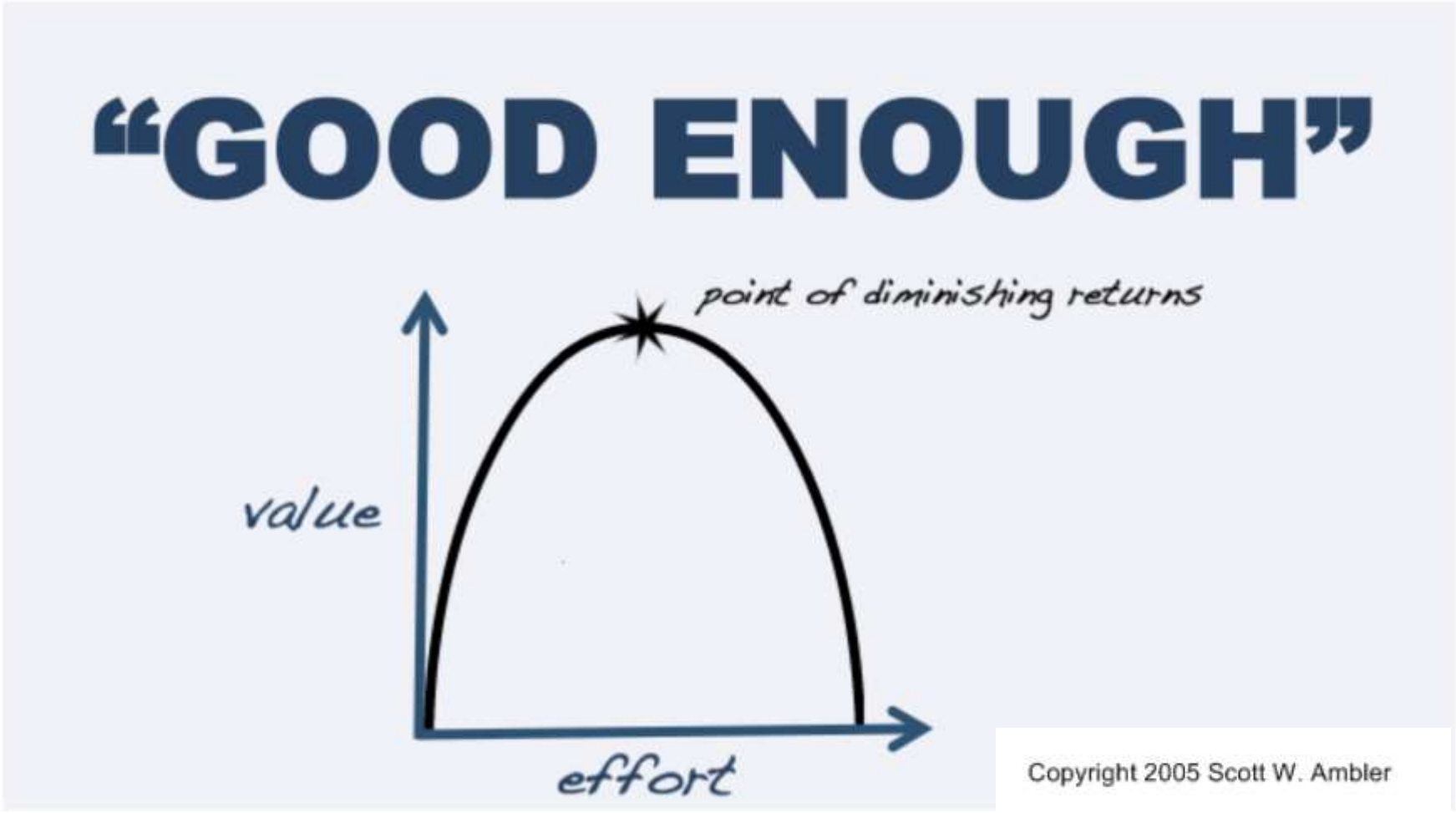
If you have a TBS value, click here: Adjust with TBS

ESAMI EMATOCHIMICI

Glicemia	**170 mg/dL**
Glicata	**7,5% (58 mmol/mol)**

“GOOD ENOUGH”

The Trouble with "Good Enough"



Good enough isn't necessarily a bad thing. In many areas of life, chasing perfection is a fool's errand, or at least a poor use of our time. We don't need to spend hours taste-testing every mustard on the gourmet shelf to find the absolute best; a good enough brand will suffice for our sandwich.

ESAMI EMATOCHIMICI

Trigliceridi **191 mg/dL**

Dieta

ESAMI EMATOCHIMICI

Acido urico **9 mg/dL**

ESAMI EMATOCHIMICI

Calcemia	**12 mg/dL**

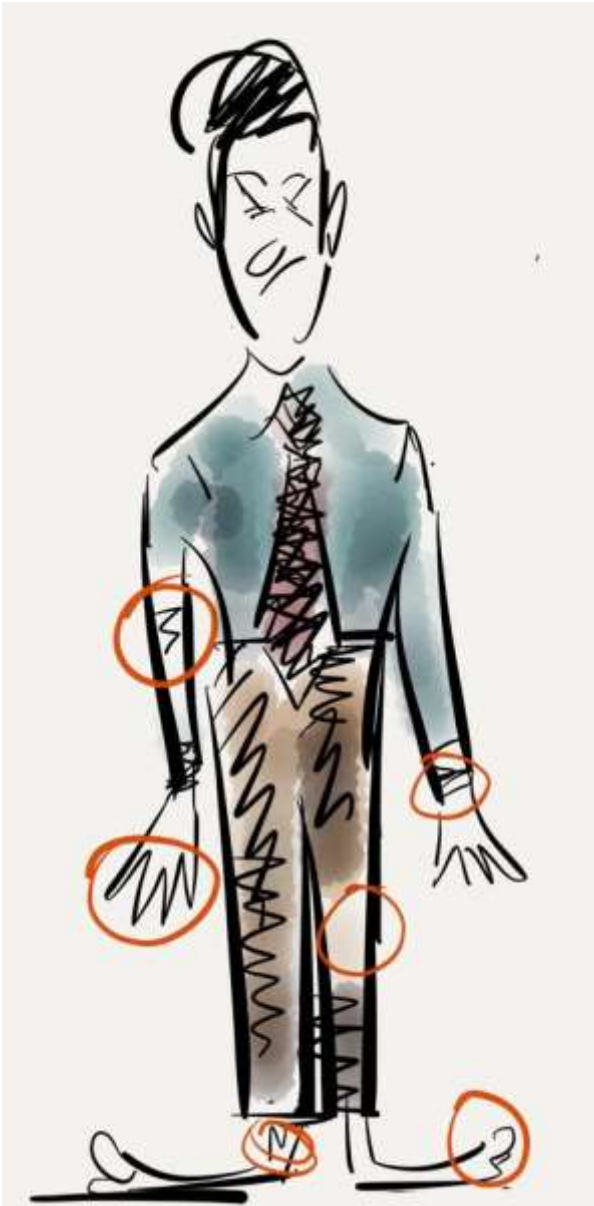
Riassumendo…

1) Diabete
2) Ipercalcemia asintomatica
3) Iperuricemia asintomatica

3 PROBLEMI CLINICI DA RISOLVERE

Procedendo dalla fine…

IPERURICEMIA ASINTOMATICA



DynaMed

Hyperuricemia

Updated: Aug 31, 2018
Author: James W Lohr, MD
Chief Editor: Vecihi Batuman, MD, FASN

Medical Care

Asymptomatic hyperuricemia

Most patients with asymptomatic hyperuricemia never develop gout or stones. Pharmacologic treatment for asymptomatic hyperuricemia carries some risk, is not considered beneficial or cost-effective, and generally is not recommended. However, these patients can be advised on lifestyle changes such as changes in diet, reduction in alcohol intake, and exercise, which may lower uric acid levels.[29] The exception to this is in an oncologic setting, in which patients receiving cytolytic treatment may receive prophylaxis against acute uric acid nephropathy.

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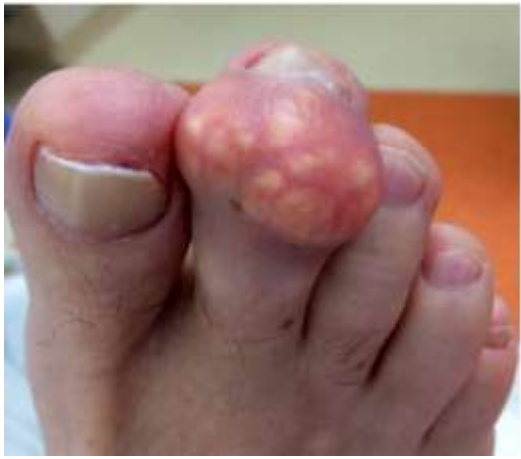
Images in topic (5) View all



Asymptomatic hyperuricemia

Authors: Michael A Becker, MD, David B Mount, MD
Section Editor: Nicola Dalbeth, MBChB, MD, FRACP
Deputy Editor: Paul L Romain, MD

Contributor Disclosures

All topics are updated as new evidence becomes available and our peer review process is complete.

Literature review current through: **Aug 2019.** | This topic last updated: **Feb 06, 2019.**

Causes of hyperuricemia due to decreased uric acid clearance

Clinical disorders	Drug- or diet-induced
Chronic renal insufficiency of any form	Diuretics (thiazides and loop diuretics)
Lead nephropathy (saturnine gout)	Cyclosporine and tacrolimus
Effective volume depletion (eg, fluid losses, heart failure)	Low-dose salicylates
Diabetic or starvation ketoacidosis	Ethambutol
Lactic acidosis	Pyrazinamide
Preeclampsia	Ethanol
Obesity	Levodopa
Hyperparathyroidism	Methoxyflurane
Hypothyroidism	Laxative abuse (alkalosis)
Sarcoidosis	Salt restriction
Chronic beryllium disease	

Con quale farmaco sostituisco il tiazidico?

- In patients with persistent asymptomatic hyperuricemia requiring medical therapy for another condition, we avoid medications for the coexisting condition that may promote hyperuricemia (eg, thiazide diuretics for hypertension), and we prefer the use of medications for coexisting conditions that reduce serum urate levels and/or decrease the risk for incident gout when these choices are acceptable therapeutic alternatives. (See 'General measures' above.)
- When there are acceptable alternatives for the management of diseases accompanying hyperuricemia, we prefer the use of medications that reduce serum urate levels and/or decrease the risk for incident gout and the avoidance of medications/additives promoting hyperuricemia. Examples of the former include the use of fenofibrate for hyperlipidemia [77] and losartan [38,78] or calcium channel blockers [38] for hypertension. Likewise, agents that may promote hyperuricemia/incident gout that should be avoided, when possible, include thiazide or loop diuretics, angiotensin-converting enzyme (ACE) inhibitors, non-losartan angiotensin II receptor blockers, and beta blockers [38]. (See "Lifestyle

LOSARTAN O CALCIO ANTAGONISTI

Diuretic-induced hyperuricemia and gout

Author: Michael A Becker, MD
Section Editor: Richard H Sterns, MD
Deputy Editor: John P Forman, MD, MSc

Contributor Disclosures

All topics are updated as new evidence becomes available and our peer review process is complete.

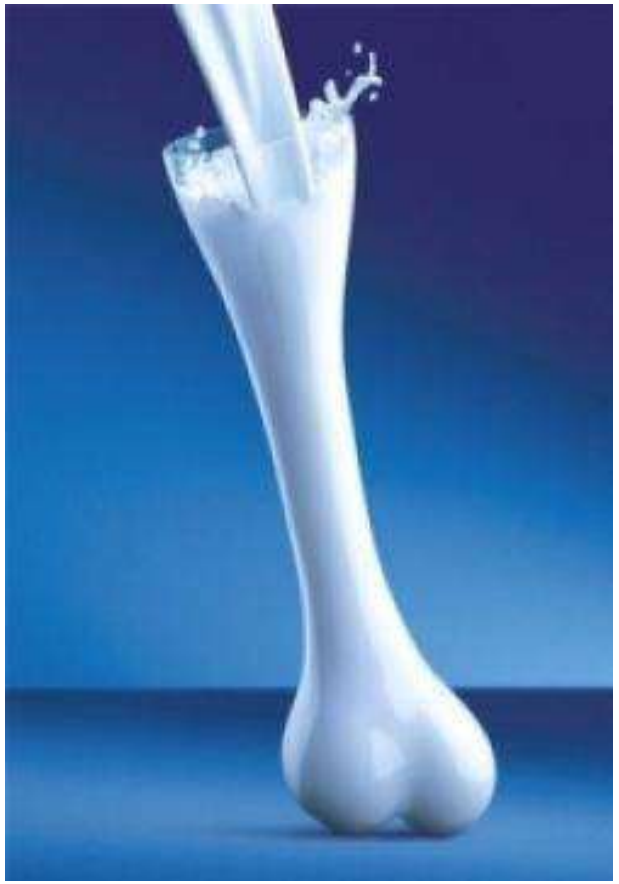
Literature review current through: **Aug 2019.** | This topic last updated: **Nov 07, 2017.**

Procedendo dalla fine…

2

TWO

IPERCALCEMIA ASINTOMATICA



Reperti Anamnestici utili

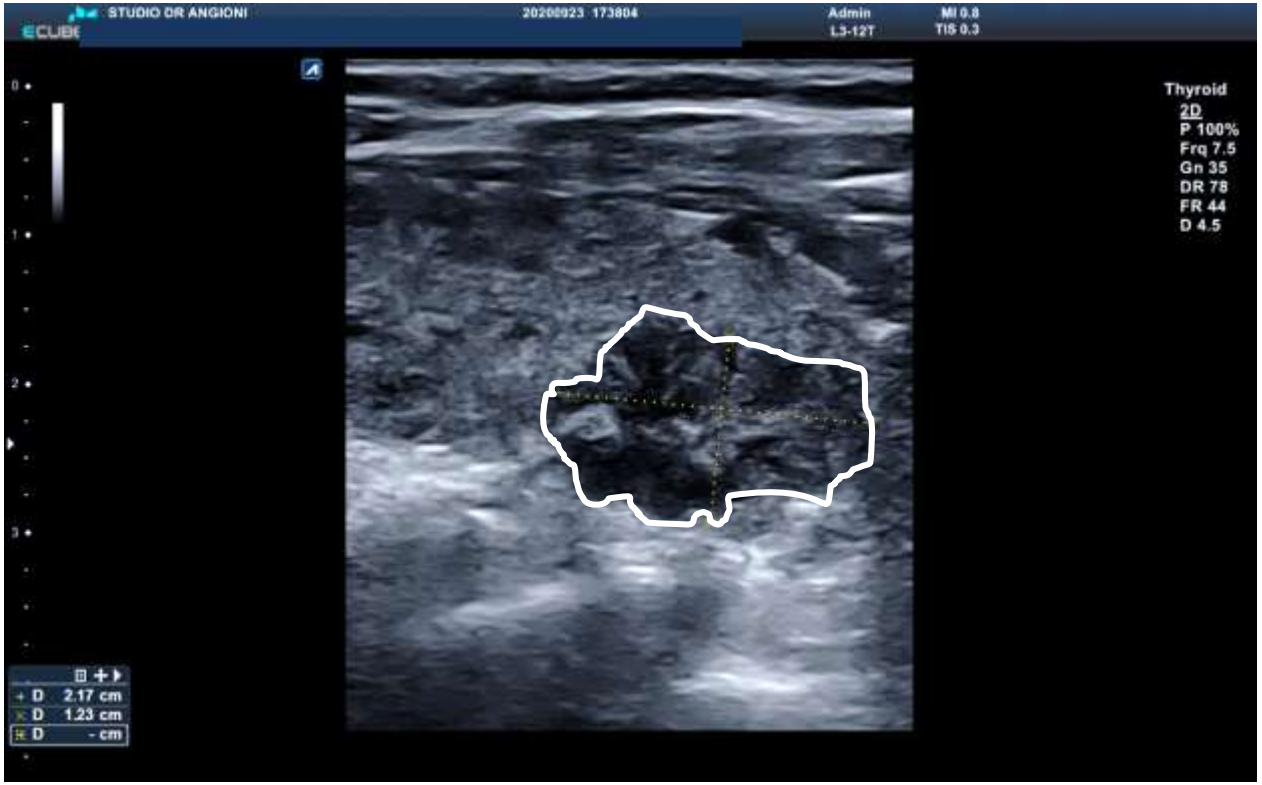
- Storia di coliche renali
- Uso di diuretici tiazidici
- Osteoporosi
- Ipercalcemia

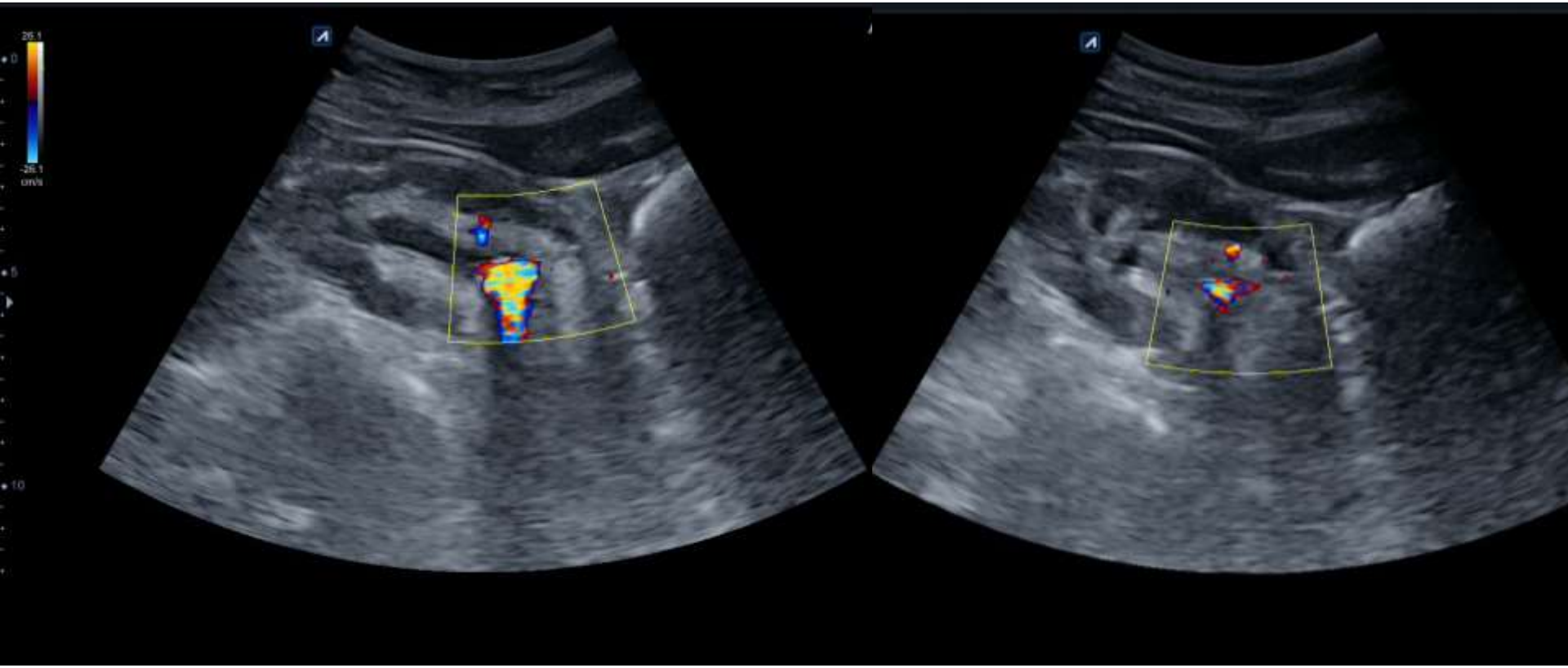
COSA FARE??

Reperti Anamnestici utili

- Storia di coliche renali
- Uso di diuretici tiazidici
- Osteoporosi
- Ipercalcemia

Ecografia…


STUDIO DR ANGIONI
20200923 173804
Admin
L3-12T
MI 0.8
TIS 0.3
Thyroid
2D
P 100%
Frq 7.5
Gn 35
DR 78
FR 44
D 4.5
D 2.17 cm
D 1.23 cm
D - cm


26.1
0
-26.1
cm/s
5
10

Twinkling=Scintillio

- **Artefatti**= interpretazioni false di echi da parte della macchina, non corrispondente a realtá anatomica
- Possono essere usati come strumenti diagnostico

PTH 110 pg/ml

Diagnosis

Making the Diagnosis

- suspect primary hyperparathyroidism in patients with hypercalcemia [2,3]
- diagnosis of primary hyperparathyroidism is based on persistent hypercalcemia in presence of elevated or inappropriately normal (not suppressed) parathyroid hormone (PTH) [1,2,3]
  - serum intact PTH typically > 28 pg/mL (3 pmol/L) in primary hyperparathyroidism [3]
  - PTH in the lower range of normal (< 25-30 pg/mL, measured by first- and second-generation intact assays) may be inappropriate in the setting of hypercalcemia and may indicate 1 or both of [1]
    - presence of PTH fragments that are not detectable by these assays, leading to falsely low PTH measurement in patients with true primary hyperparathyroidism
    - an early form of primary hyperparathyroidism
  - repeated measurements of albumin-corrected calcium levels usually required for accurate diagnosis [1]
- diagnosis of normocalcemic hyperparathyroidism is based on inappropriately high PTH levels in presence of normal total and ionized calcium levels after excluding causes of secondary hyperparathyroidism [1]

Overview and Recommendations

Background

- Primary hyperparathyroidism is an endocrine disorder caused by autonomous overproduction of parathyroid hormone (PTH) by abnormal parathyroid glands and characterized by hypercalcemia without appropriate suppression of plasma parathyroid hormone levels; it is the most common cause of hyperparathyroidism and of hypercalcemia.

- It is most common in adults > 50 years old and 3-4 times more common in females compared to males. Possible risk factors include exposure to ionizing radiation, long-term lithium therapy, hereditary endocrine syndromes such as multiple endocrine neoplasia, reduced physical activity, and low calcium intake.

Evaluation

- Most patients (about 80%) are asymptomatic, and the disease is detected by an incidental finding of hypercalcemia on a laboratory test.

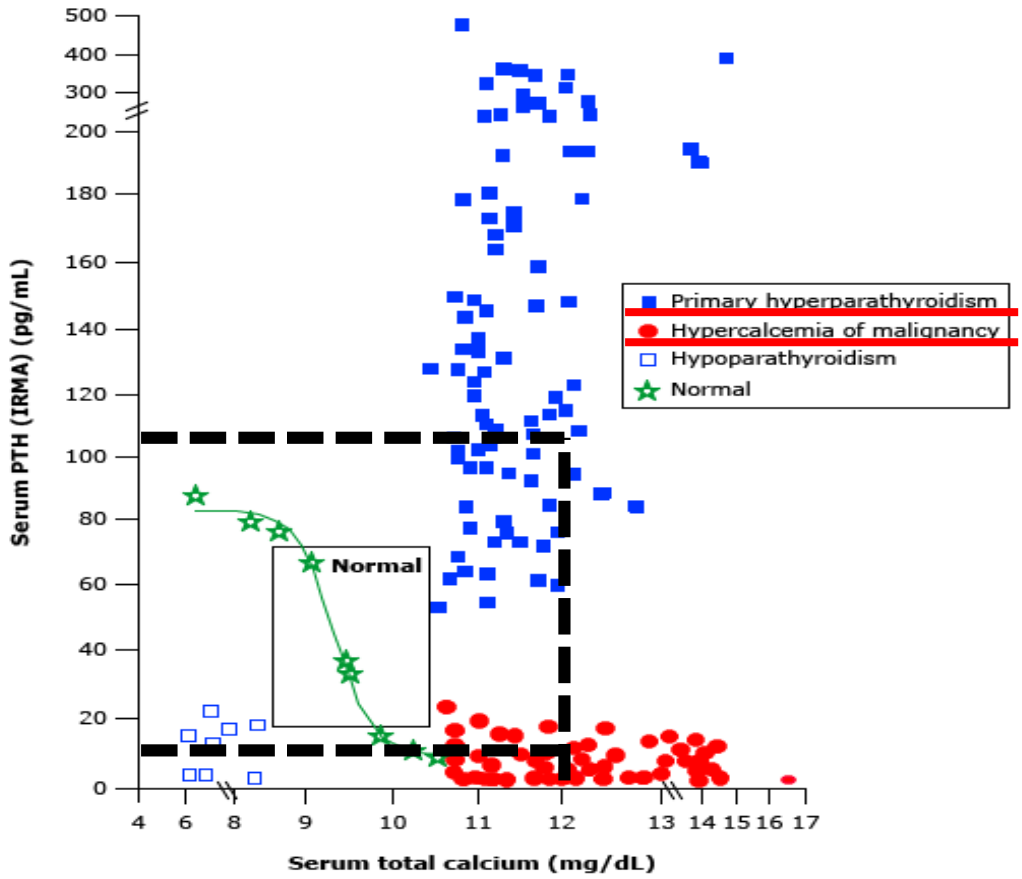
Past Medical History (PMH)

- ask about history or symptoms suggestive of other causes of hypercalcemia, including
  - 1,25-dihydroxyvitamin D overproduction [1]
  - osteolytic metastases [1]
  - sarcoidosis
  - excess calcium ingestion [1]
  - malignancies [1]
  - familial hypocalciuric hypercalcemia [1]
  - prolonged immobilization [1]
  - excessive vitamin A intake (West Indian Med J 2014 Jan;63(1):105 full-text)
- ask about history or symptoms suggestive of other causes of parathyroid hormone elevation, including [1,2]
  - renal insufficiency or renal calcium leak
  - vitamin D deficiency
  - malabsorption syndromes
- ask about complications of primary hyperparathyroidism such as anemia, gastroesophageal reflux disease, peptic ulcer disease, and acute pancreatitis [1]

Family History (FH)

- ask about personal and family history in patients with suspected primary hyperparathyroidism (AAES Strong recommendation, Moderate-quality evidence) [1]
- ask about family history of inherited syndromes such as [1,3]
  - multiple endocrine neoplasia type 1, multiple endocrine neoplasia type 2A, and multiple endocrine neoplasia type 4
  - familial hyperparathyroidism-jaw tumor syndrome
  - familial isolated hyperparathyroidism
  - familial hypocalciuric hypercalcemia

Serum parathyroid hormone (PTH) concentrations in hypercalcemia and hypocalcemia



Serum PTH concentrations according to the serum total calcium concentration in various disease states. The normal range is shown in the white box. The sigmoidal curve (green stars) is derived from a calcium citrate infusion protocol administered to 38 normal subjects. Serum PTH and calcium values are low in hypoparathyroidism (open blue boxes) and high in primary hyperparathyroidism (blue squares). The serum calcium concentration is high and serum PTH is appropriately low in patients with non-PTH-induced hypercalcemia of malignancy (red circles).

PTH: parathyroid hormone.

Data from: Haden ST, Brown EM, Hurwitz S, et al. The effects of age and gender on parathyroid hormone dynamics. Clin Endocrinol 2000; 52:329.

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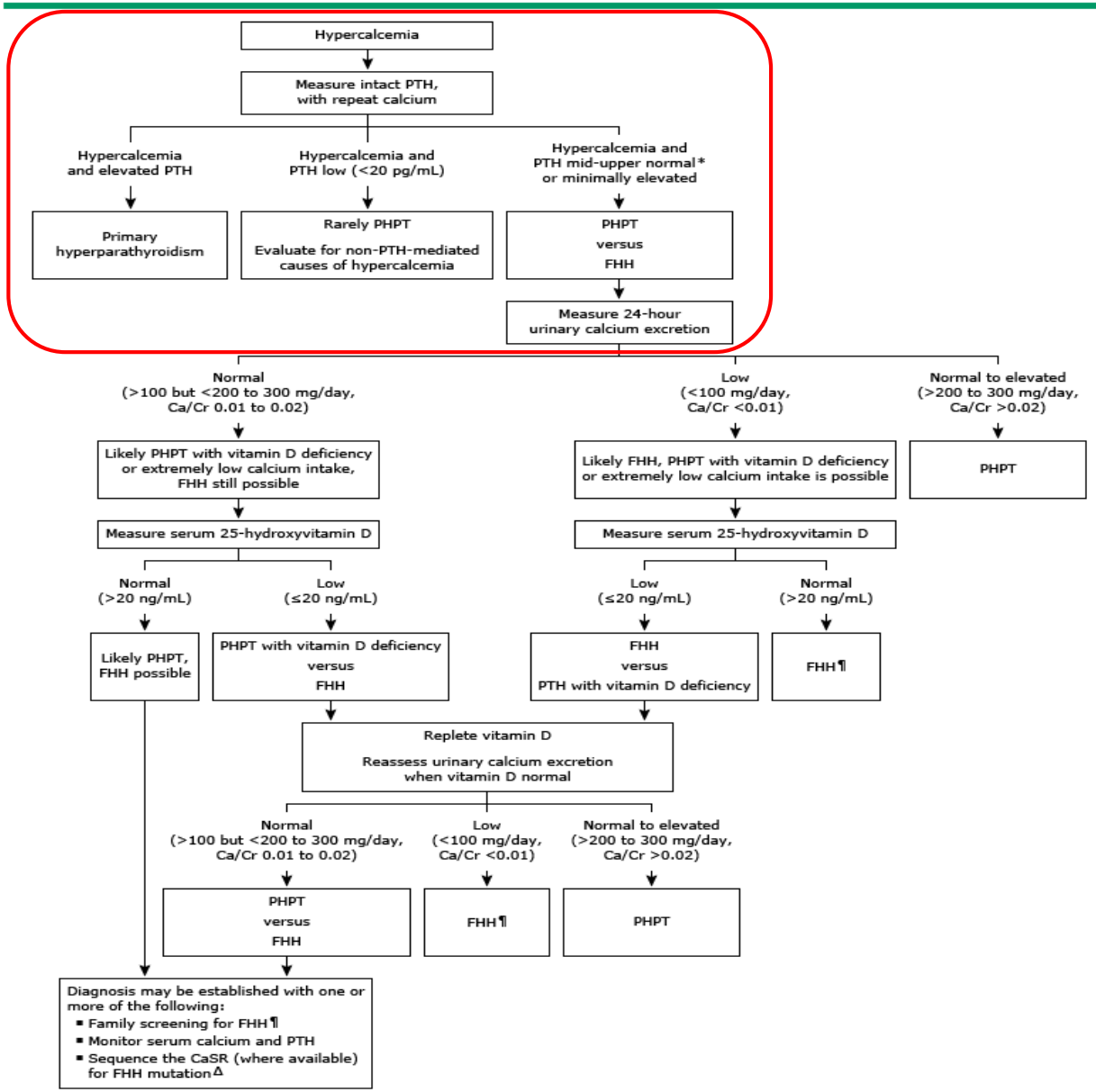
  - familial isolated hyperparathyroidism
  - familial hypocalciuric hypercalcemia

Diagnosis of primary hyperparathyroidism



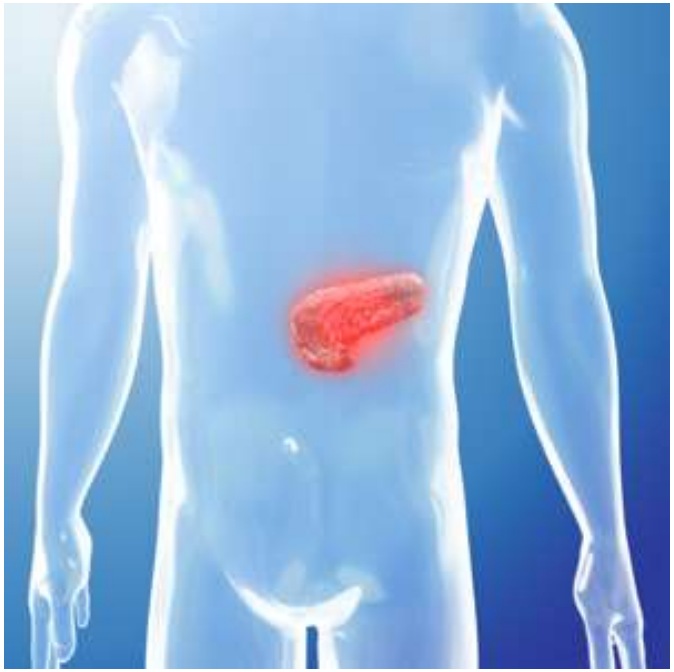
PTH: parathyroid hormone; PHPT: primary hyperparathyroidism; FHH: familial hypocalciuric hypercalcemia; Ca/Cr: calcium/creatinine ratio; CaSR: calcium-sensing receptor.

* Inappropriately normal given hypercalcemia

¶ Assess for a family history of asymptomatic hypercalcemia, especially in young children.

Δ Refer to UpToDate topic on hyperparathyroidism for details.

Procedendo dalla fine…



DIABETE DI NUOVO RISCONTRO

Facile…..

Aumento Slowmet a 2000 mg/die

Ed esegue Hb glicata tra 6 mesi…

A febbraio 2020 Giovanna esegue paratiroidectomia..


LOCKDOWN

VENERDÍ 24 LUGLIO 2020
Ore 18.45

‘’Buongiorno dottore sono tornata a portare il risultato della glicata, intanto le faccio vedere che ho perso peso, mi sono impegnata’’

✓	18/11/2019	100	153	42,72 - Obesità di classe...

Data: 24/07/2020 (1)

✓	**BMI**	BMI: 38,45, Peso: 90, Altezza: 153

Giovanna

Provenienza. :
Codice Fisc. :
Campione del :
Stampa del :

Esame	Risultato	Unita' Di Misura	Valori di Riferimento
Monociti #valore assoluto	0.35	x10^3/ microL	0.00 - 0.80
Eosinofili #valore assoluto	0.13	x10^3/ microL	0.00 - 0.45
Basofili #valore assoluto	0.03	x10^3/ microL	0.00 - 0.20
CONTEGGIO ERITROBLASTI			
Eritroblasti	0.0	%	
Eritroblasti #valore assoluto	0.00	x10^3/ microL	

Valori di riferimento in base alle indicazioni della letteratura nazionale ed internazionale e commisurati alla popolazione di pazienti afferenti al Laboratorio

BIOCHIMICA

GLICEMIA	* 451	mg/dL	70 - 100

Valori normali a digiuno: 70-100 mg/dl
Alterata glicemia a digiuno: 101-125 mg/dl

EMOGLOBINA GLICATA	* 15.6	%	4.2 - 6.2
EMOGLOBINA GLICATA	* 147.0	mmol/mol	22.4 - 44.3

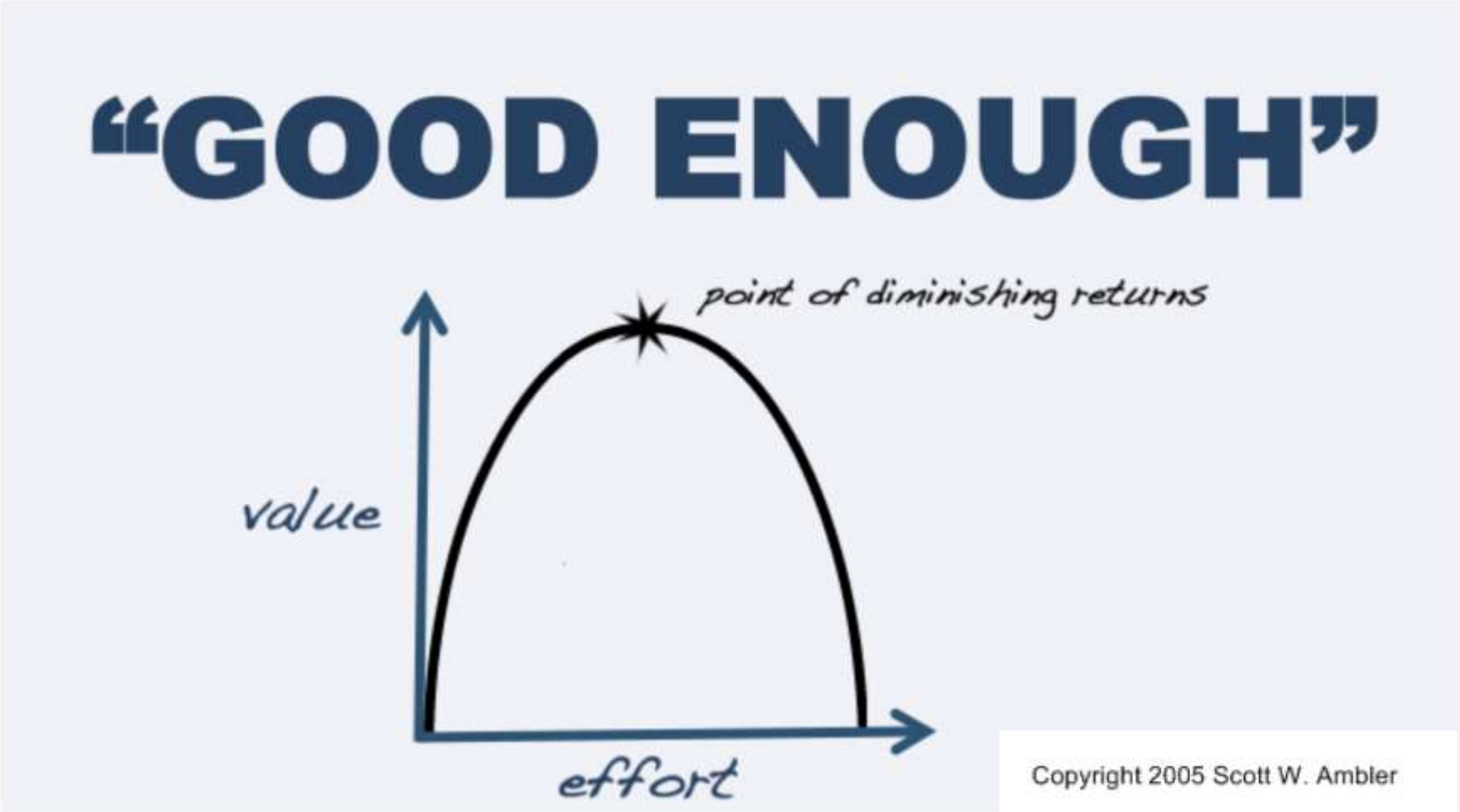
(Metodica capillare standardizzata IFCC)

CREATININA	0.69	mg/dL	0.60 - 1.10
Filtrato glomerulare stimato (eGFR)	92		

Formula CKD-EPI
mL/min/1,73m2
normalità GFR > 90
lieve riduzione GFR 60 - 89
moderata riduzione GFR 30 - 59
marcata riduzione GFR 15 - 29
severa riduzione GFR < 15
Nota: eGFR non applicabile in gravidanza, negli estremi di massa corporea e nei soggetti con patologie multiple.
Se il paziente è afroamericano il valore della formula CKD-EPI deve essere moltiplicato per 1,15

ACIDO URICO	4.9	mg/dL	2.6 - 6.0

The Trouble with "Good Enough"



Cosa fare?

- Quale farmaco è piú adatto per Giovanna?
- È giusto sospendere la metformina?
- Giovanna potrebbe usare anche i nuovi farmaci?

PAZIENTI DIVERSI STESSA TERAPIA?

Savina 85 anni

1) DM2
2) Ipertesa
3) IRC stadio 3
4) Osteoporosi
5) Osteoartrosi

Glicata 6,4%
(prima del cambio di Target e Terapia)

Assume:
-Metformina 1000 mg
-Paracetamolo 1000 mg AB
-Vitamina D
-Ramipril 5 mg

Giovanna 65 anni

1) DM2
2) Ipertesa
3) Obesa
4) Sdr. Ansioso depressiva
5) Osteoporosi
6) Pregresso k mammella
7) Pregresso adenoma parat.

Glicata 15,6 %

Assume:
-Slowmet 2000 mg
-Paroxetina 20 mg
-Simvastatina 20 mg
-Losartan 100 mg
-Alendronato 70 mg + vit D