



10:00-11:00

SESSION 5: A FOCUS IN THE WORK-UP OF CUSHING'S SYNDROME: THE ROLE OF IMAGING

Chairs: Francesco Briganti, Sossio Cirillo

10:00-10:15

THE IMAGING IN CUSHING'S DISEASE

Fabio Tortora

10:15-10:30

THE IMAGING IN ADRENAL CUSHING'S SYNDROME

Giovanni Vitale

10:30-10:45

THE IMAGING IN ECTOPIC CUSHING'S SYNDROME

Roberto Baldelli

10:45-11:00

Discussion



Roberto BALDELLI M.D., Ph.D.

**“Regina Elena” Italian National Cancer Institute
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THE SOURCE OF ACTH SECRETION



Roberto BALDELLI M.D. Ph.D.

In adults:

80% of CS is due to ACTH-dependent causes

20% due to adrenal causes

ACTH secreting neoplasms cause ACTH-dependent CS.

These are usually anterior ***pituitary microadenomas***, which result in the classic Cushing's disease.

Non-pituitary ectopic sources of ACTH, such as a small-cell lung carcinoma or carcinoid tumours, are the source of the remainder of ACTH-dependent disease. In the majority of patients presenting with clinical and biochemical evidence of CS, modern non-invasive imaging can accurately and efficiently provide the cause and the nature of the underlying pathology.



Considering that EAS accounts for 10% to 20% of all cases of Cushing's syndrome, these syndromes constitute a small but significant percentage of patients presenting with clinical overt hypercortisolism

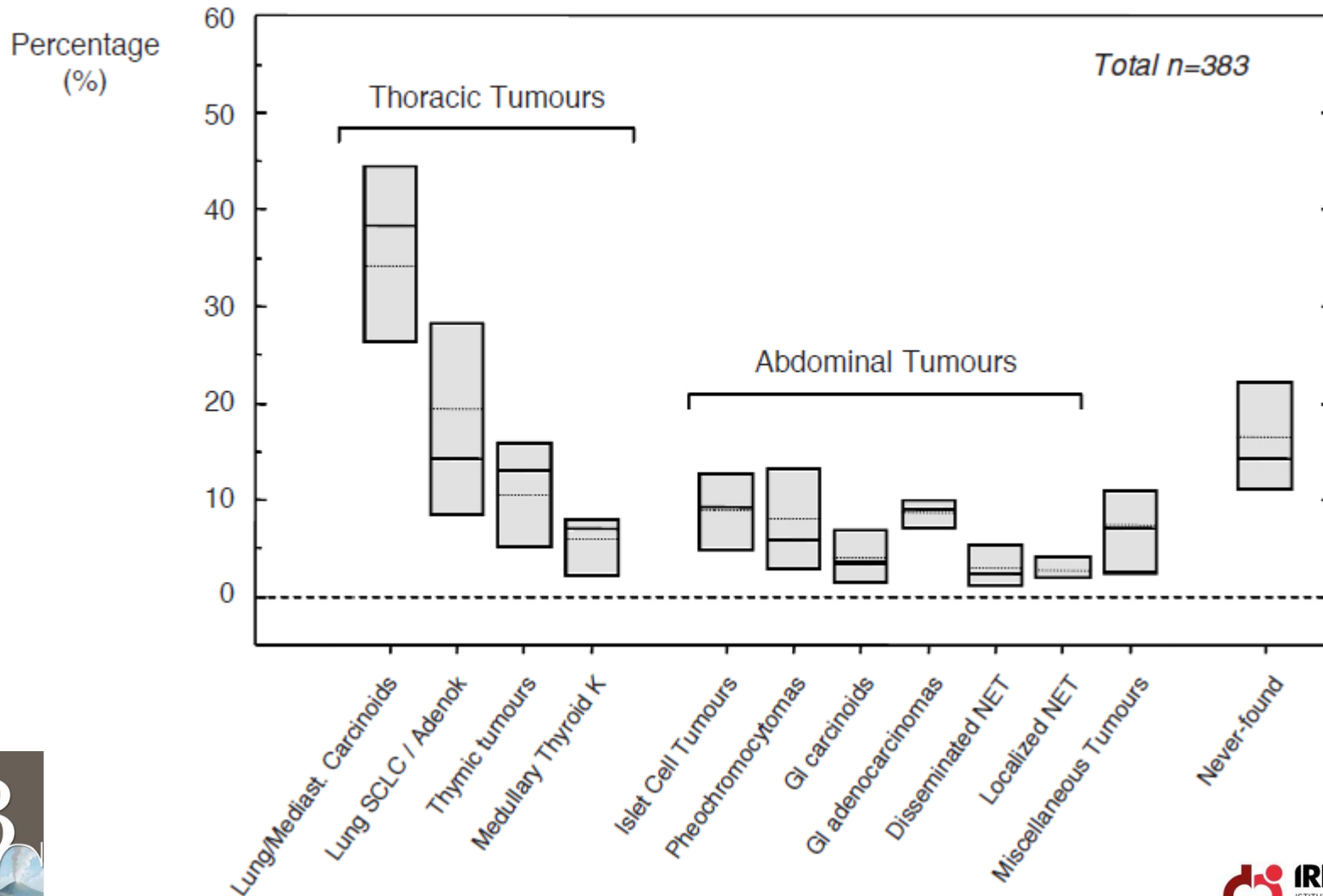


Ectopic Cushing and Other Paraneoplastic Syndromes in Thoracic Neuroendocrine Tumors

Diego Ferone, MD, PhD*, Manuela Albertelli, MD, PhD

- Overproduction of corticotropin by the pituitary gland or extrapituitary tumors leads to hypercortisolism or Cushing syndrome.
- Diagnosis of suspected Cushing syndrome involves 3 major steps: confirmation of hypercortisolism, differentiation between corticotropin-independent and corticotropin dependent causes of Cushing syndrome, and distinction between pituitary and ectopic corticotropin production.
- When ectopic corticotropin is produced by malignancies, circulating corticotropin and cortisol levels are extremely high, the duration of symptoms is shorter, and the clinical phenotype is atypical compared with pituitary-dependent Cushing disease.
- A definitive diagnosis of ectopic corticotropin secretion should require stringent criteria, including reversal of the clinical picture after resection of the tumor and/or demonstration of corticotropin immunohistochemical staining within the tumor tissue.
- Various neoplasms can produce corticotropin, especially those originating from neuroendocrine cells.

Prevalence of Tumours responsible of EAS



Ectopic ACTH Syndrome

ANDREA M. ISIDORI
ANDREA LENZI

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	FREQUENCY 1–3%	FREQUENCY ≤ 1%
Tumours associated with EAS	Ovarian carcinoma	Esophageal carcinoma
	Colonic/anal carcinoma	Kidney tumor
	Prostate	Hepatocarcinoma
	Uterine cervix carcinoma	Breast carcinoma
	Neuroblastoma	Salivary gland tumor
		Mesothelioma
		Lymphoma
	[Ectopic pituitary adenoma]	Melanoma
		Leydig cell tumor
		Larynx carcinoma
	Gallbladder tumours	

Ectopic Cushing and Other Paraneoplastic Syndromes in Thoracic Neuroendocrine Tumors

Diego Ferone, MD, PhD*, Manuela Albertelli, MD, PhD

- After small cell lung carcinoma (SCLC) and carcinoid tumors, the subsequent most commonly reported tumors causing ectopic corticotropin secretion are harbored in the thymus (11%) and pancreas (8%).
- The main conclusion that can be drawn from the most recently published series is that more than half of the tumors producing ectopic corticotropin secretion were found in the lung or in the thymus, whereas, including MTC and pheocromocytomas, two-thirds were in the thorax, neck, or adrenal glands.
- As in Cushing disease, major symptoms and signs include central obesity, primary or secondary amenorrhea in female patients, hirsutism, acne, violaceous skin striae, easy bruising, hypertension, glucose metabolism imbalance, fatigue, muscle weakness, mental changes or emotional disturbances, hyperpigmentation, and acanthosis nigricans.
- Due to the difficulties in differentiating the source of ectopic corticotropin and although the ectopic tumor represents the minority of all cases of Cushing syndrome, an accurate biochemical as well as radiological work-up is strongly recommended.

Ectopic Cushing and Other Paraneoplastic Syndromes in Thoracic Neuroendocrine Tumors

Diego Ferone, MD, PhD*, Manuela Albertelli, MD, PhD

- Surgery represents first-line treatment in these patients; however, differently from pituitary corticotropin-dependent Cushing, these cases are generally responsive to somatostatin analog therapy, at least in terms of clinical and biochemical control of the paraneoplastic syndrome.
- Hyponatremia is a common feature in patients with lung cancer.
- Ectopic acromegaly is rare, and since the discovery of growth hormone (GH)–releasing hormone (GHRH) approximately 30 years ago, only 74 cases have been reported in the literature.
- Carcinoid syndrome is a rare feature in bronchial carcinoid patients.
- Another rare cause of endocrine syndrome associated with carcinoid tumor is malignant hypercalcemia and includes ectopic production of parathormone (PTH) or PTH-related peptide (PTH-rp) by the tumor.

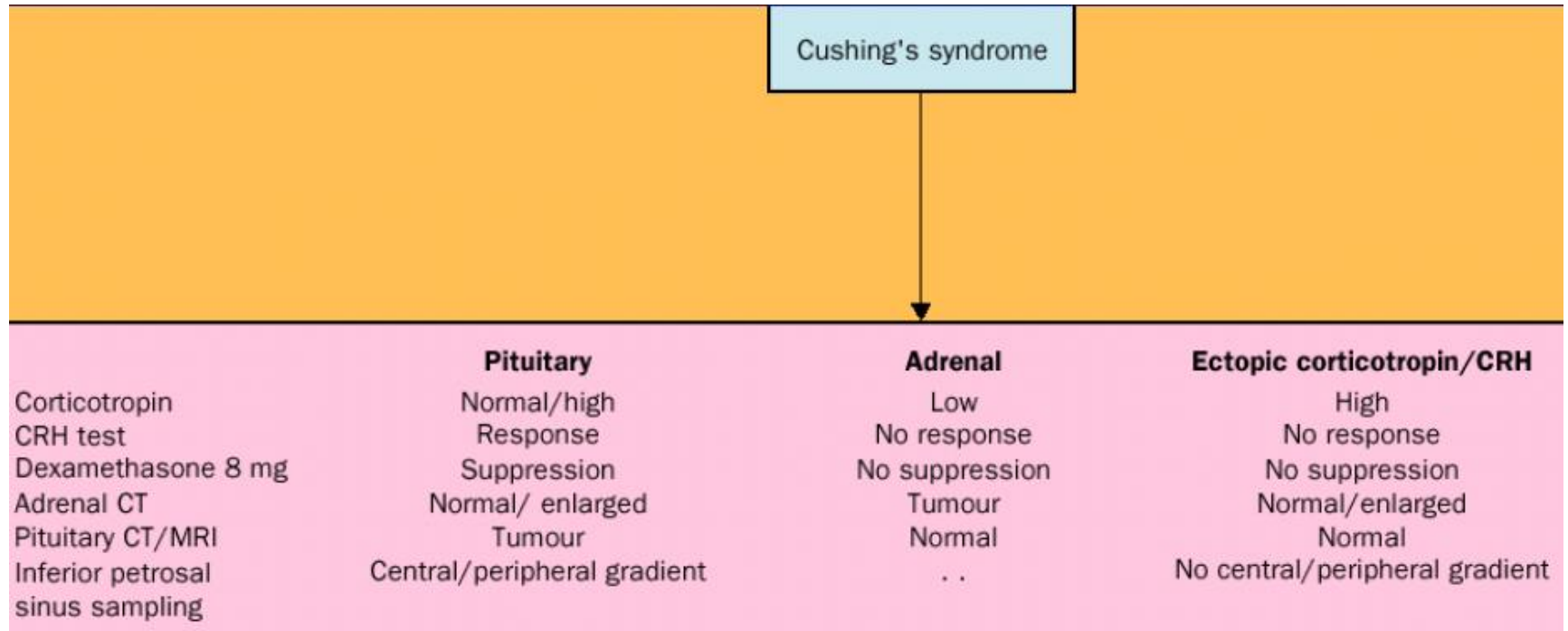


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DIAGNOSIS OF ECTOPIC ACTH SECRETION

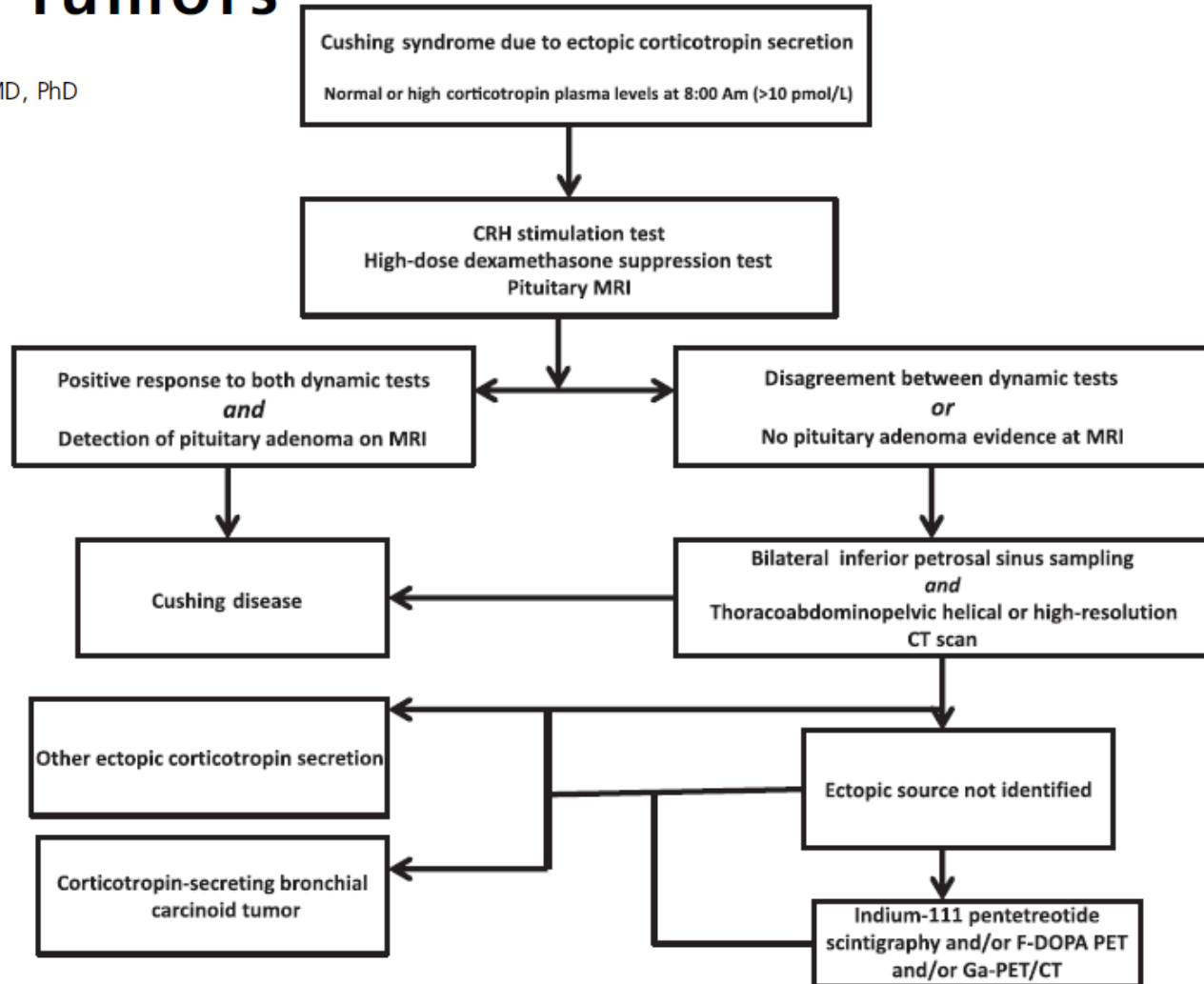


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Ectopic Cushing and Other Paraneoplastic Syndromes in Thoracic Neuroendocrine Tumors

Diego Ferone, MD, PhD*, Manuela Albertelli, MD, PhD



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Unità Operativa di Endocrinologia



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LOCALIZATION OF THE ACTH SECRETING TUMOUR



Roberto BALDELLI M.D. Ph.D.



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Identifying an ectopic ACTH source

1. Because the most likely site of ectopic corticotropin-producing tumors is thorax and these tumors are frequently bronchial carcinoids, accurate radiologic search is mandatory.
2. These lesions are mostly small, as well as slow growing, and conventional imaging studies, such as *CT and MRI scans, identify the tumor in only 50% of cases.*
3. Functional imaging studies, such as fludeoxyglucose F 18 (FDG) positron emission tomography (PET), and somatostatin receptor scintigraphy (SRS), are complementary imaging tools to detect carcinoids. SRS might be superior to FDG PET in detecting bronchial carcinoids.
4. FDG PET can distinguish highly active proliferative tumors, whereas bronchial carcinoids usually have a low proliferation index and are slow growing small lesions.

Identifying an ectopic ACTH source

Carcinoide ATIPICO

1. Because the most likely site of ectopic corticotropin-producing tumors is the thorax, these tumors are frequently bronchial carcinoids, accurate radiologic search is mandatory.

2. These lesions are more frequent than conventional imaging (chest X-ray, CT and MRI scans), identify the tumor in only 50% of cases.

3. Functional imaging studies include deoxyglucose F 18 (FDG) and somatostatin receptor scintigraphy (SRS), are complementary imaging tools to detect carcinoids. SRS might be more sensitive to FDG PET in detecting bronchial carcinoids.

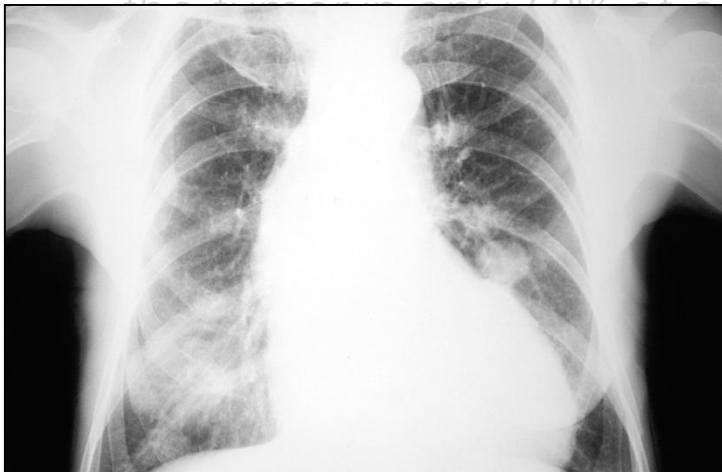
4. FDG PET can distinguish active proliferative tumors, whereas bronchial carcinoids have a low proliferation index and are slow growing lesions.



Identifying an ectopic ACTH source

1. Because the most likely site of ectopic corticotropin-producing carcinoma neuroendocrino a grandi cellule (LCNEC)

2. These lesions are mostly small, as well as slow growing, and conventional imaging studies, such as CT and MRI scans, identify them in only 50% of cases.



4. PET-CT can distinguish highly active proliferative tumors, whereas bronchial carcinoids usually have a low proliferation index and are slow growing small lesions.

Identifying an ectopic ACTH source

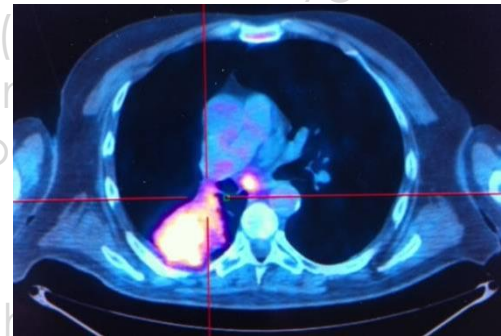
1. Because the most likely site of ectopic corticotropin-producing tumors is the thorax, a careful search is mandatory for all carcinoids, accurate radiologic search is mandatory.

Carcinoma neuroendocrino a grandi cellule (LCNEC)

2. These lesions are mostly small, as well as slow growing, and conventional imaging studies, such as CT and MRI scans, identify the tumor in only 50% of cases.



3. Functional imaging techniques, such as fludeoxyglucose F 18 (FDG) positron emission tomography (PET) and somatostatin receptor scintigraphy (SRS), are more sensitive than conventional imaging in detecting bronchial carcinoids.



4. FDG PET can distinguish high-grade, aggressive tumors, whereas bronchial carcinoids usually have a low proliferation index and are slow growing small lesions.

Identifying an ectopic ACTH source

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Other sources of excess ACTH production include neuroendocrine tumours of the thymus, bowel and pancreas, medullary carcinoma of the thyroid, phaeochromocytomas and mesotheliomas.

In approximately 12–20% of patients, despite repeated biochemical and radiological investigations, the source of the ectopic ACTH production remains undiscovered.

Occult ectopic ACTH syndrome has been defined as ACTH dependent hypercortisolism of greater than 6 months duration without emergence of an obvious cause or source.



Identifying an ectopic ACTH source

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1. After biochemical confirmation of EAS, optimal treatment includes localization and removal of the ACTH secreting tumour.
2. Early detection of ectopic corticotropin-producing tumours is crucial in the management of affected patients, as it can avoid adrenalectomy and help to reduce the risk of metastatic disease.
3. Localization of these tumours can occasionally be difficult and may require extensive long-term follow-up.
4. **The application of an imaging protocol with reliable and high sensitivity procedures is advised.**



Identifying an ectopic ACTH source

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1. Most **SCLCs** are detected using plain chest X ray, CT and/or MR imaging.
2. **Bronchial carcinoids** can be relatively small and thus be missed with conventional imaging; however, early application of 2–3 mm high-resolution CT chest scans, particularly with the new generation of multidetector CT, identifies the vast majority of such cases.
3. According to series from other specialized centres, serial CT and MRI scans fail to localize around 33% to 44% of ectopic corticotropin-producing tumours.



Identifying an ectopic ACTH source

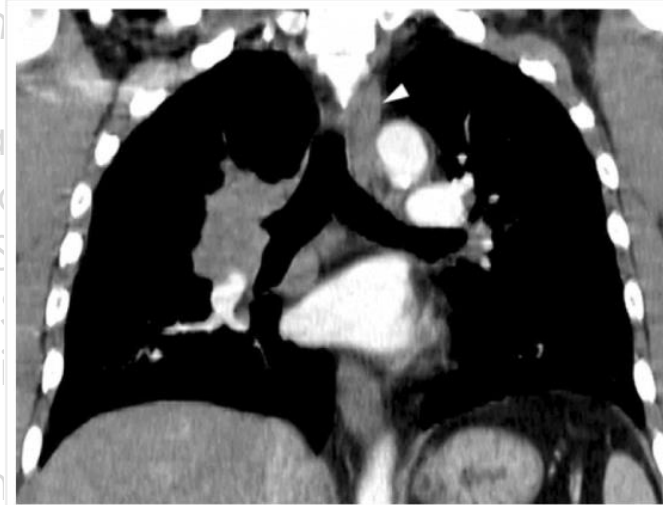
Carcinoma neuroendocrino a piccole cellule (SCLC)

1. Because the most likely site of ectopic corticotropin-producing carcinoids, accurate radiologic search is mandatory.

2. These lesions are mostly small, as well as slow growing, and conventional imaging studies, such as CT and MRI scans, identify the tumor in only

3. Functional imaging tools to detect bronchial carcinoids. Somatostatin receptor scintigraphy (SRS) and FDG PET in detecting

4. FDG PET can detect proliferative tumors, whereas bronchial carcinoids usually have a low proliferation index and are slow growing small lesions.



Identifying an ectopic ACTH source

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Imaging with ^{111}In -octreotide can identify true occult ACTH-secreting tumours in most patients with NETs, including bronchial carcinoid tumours, as bronchial lesions less than 1 cm in diameter have been identified with this technique.



Identifying an ectopic ACTH source

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1. However, there has been some concern whether it is really superior to CT scanning, as in a direct comparison ^{111}In -octreotide identified fewer lesions than CT, and did not identify any tumours that were not also seen with CT.
2. In the most recently published series, **scintigraphy with ^{111}In -octreotide did not add to the diagnostic information derived from other imaging techniques.**
3. There is currently no consensus as to whether ^{111}In -octreotide scintigraphy, after failing to establish the diagnosis at first presentation, should be included in the follow-up.
4. We believe that there is insufficient data to exclude this examination, as it can still provide useful diagnostic information for the management of these patients.

Identifying an ectopic ACTH source

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Pancreatic islet cell tumours and **MTCs** associated with EAS are usually large and have already metastasized to the liver by the time CS is diagnosed (CT scan and MRI are mandatory); no covert EAS secondary to an islet cell tumour was encountered in a relevant review.

Such tumours are usually functional and co-secrete other biologically active substances associated with characteristic clinical syndromes, and CS is only occasionally the main complaint on presentation.



Utility of Various Functional and Anatomic Imaging Modalities for Detection of Ectopic Adrenocorticotropin-Secreting Tumors

Marina S. Zemskova,* Bhaskar Gundabolu,* Ninet Sinaii, Clara C. Chen, Jorge A. Carrasquillo, Millie Whatley, Iffat Chowdhury, Ahmed M. Gharib, and Lynnette K. Nieman

1. Because ectopic ACTH-secreting (EAS) tumors are often occult, improved imaging is needed.
2. The objective was to evaluate the utility of [111In-DTPA-D-Phe]pentetretotide scintigraphy [octreotide (OCT)] imaging at 6 mCi [low OCT (LOCT)] and 18 mCi [high OCT (HOCT)], [18F]fluorodeoxyglucose (FDG)-positron emission tomography (PET) and [18F]L-3,4-dihydroxyphenylalanine (F-DOPA)-PET scans, computed tomography (CT), and magnetic resonance imaging (MRI).
3. The study was a prospective evaluating forty-one subjects participated, 30 with resected EAS tumors and 11 (three female) with occult EAS, based on inferior petrosal sinus sampling results and imaging studies.

Utility of Various Functional and Anatomic Imaging Modalities for Detection of Ectopic Adrenocorticotropin-Secreting Tumors

Marina S. Zemsanova,* Bhaskar Gundabolu,* Nishant J. Desai, Clara C. Chen, Jorge A. Carrasquillo, Millie Whatley, Iffat Chaudhry, Ahmed M. Gharib, and Lynnette K. Nieman



Intervention included every 6–12 months:

1. CT and MRI of neck, chest, abdomen,
2. LOCT (6 mCi) (with or without HOCT – 18 mCi)
3. FDG- PET
4. F-DOPA-PET

Utility of Various Functional and Anatomic Imaging Modalities for Detection of Ectopic Adrenocorticotropin-Secreting Tumors

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The author suggest that initial imaging of patients with presumed EAS include thoracic CT and MRI followed by LOCT. Further investigations in a larger population with different tumor types and amounts of tumor burden are necessary to confirm and extend these findings and determine the best imaging studies and/or their combinations for the detection of ectopic ACTH-producing tumors.

PT ID	CT	MRI	OCT	PET	Number detected by test combination
Patients with four studies performed (CT, MRI, OCT, PET)					
2, 3, 5, 6, 7, 9, 10, 12, 18, 29					10 patients: four tests positive
11, 23, 4, 13, 14, 16, 15, 17, 28, 8, 27			NEG (H) NEG (H) NEG (L)	NEG NEG NEG NEG ---NEG ---NEG NEG NEG ---	11 patients: two or three of four tests positive
Patient with CT, OCT, PET performed					
1					1 patient: 3/3 tests positive
Patients with CT, MRI, OCT performed					
19, 22					2 patients: 3/3 tests positive
20, 25, 21, 30	NEG		NEG (L) NEG (L) NEG (L) NEG (H)		4 patients: 1-2 of 3 tests positive
Patients with CT and MRI performed					
24					1 patient: both tests positive
26		NEG			1 patient: one test positive

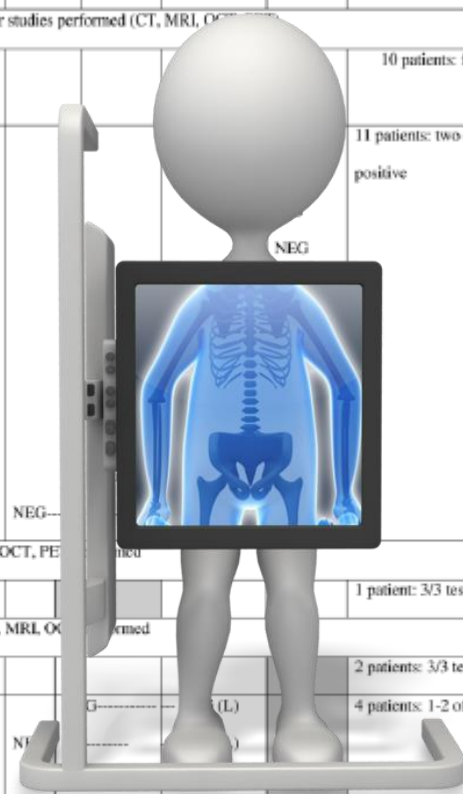


Utility of Various Functional and Anatomic Imaging Modalities for Detection of Ectopic Adrenocorticotropin-Secreting Tumors

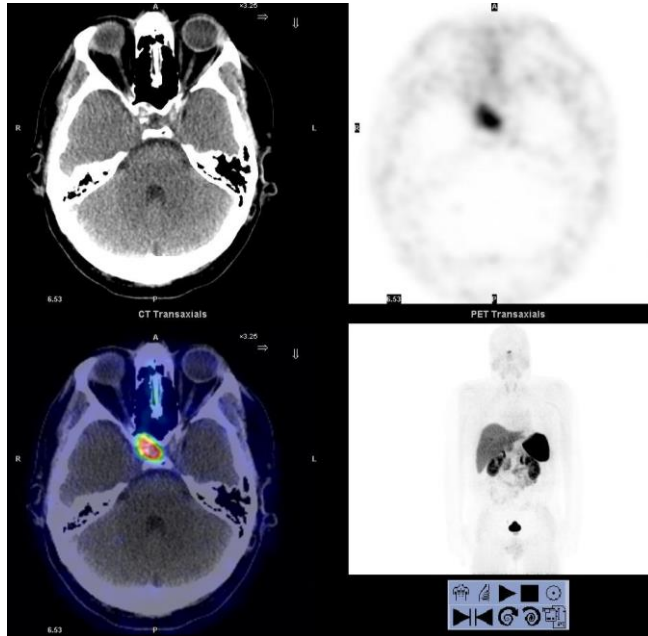
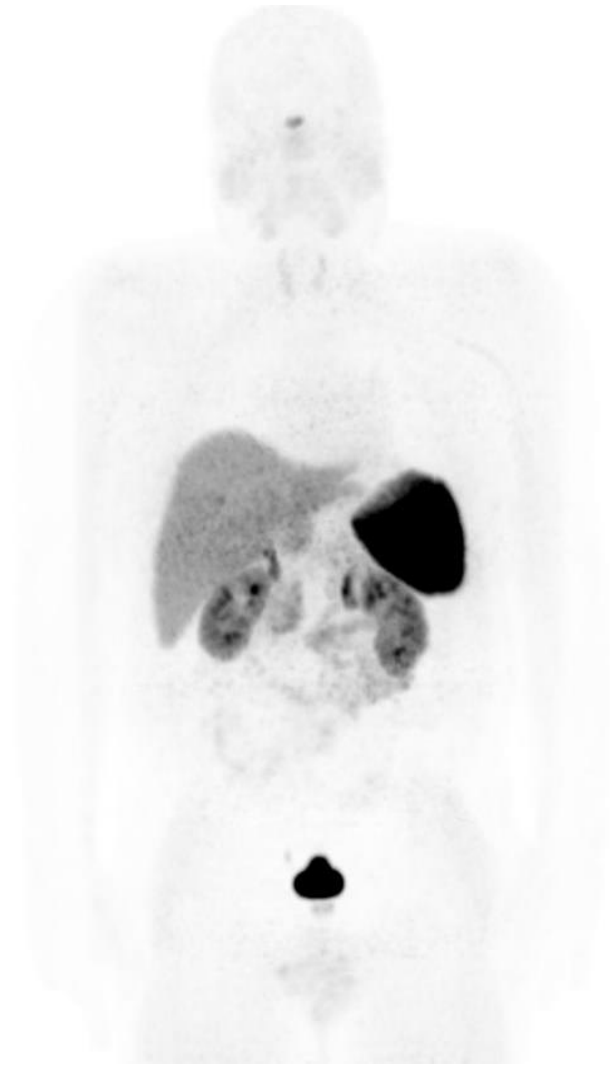
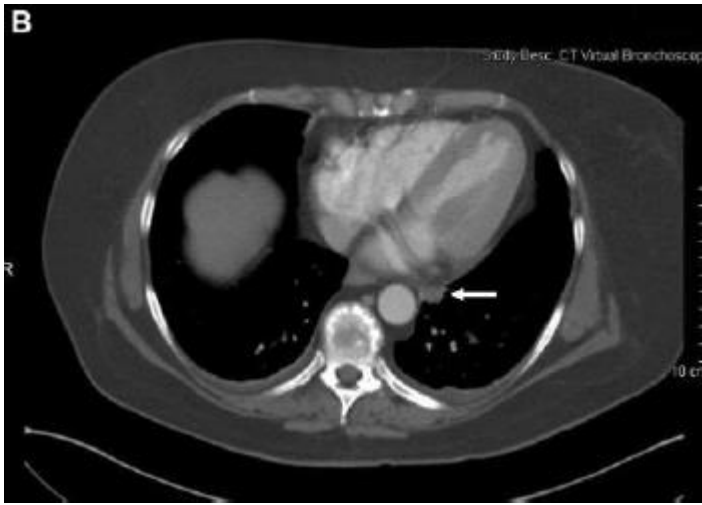
Marina S. Zemsanova,* Bhaskar Gundabolu,* Ninet Sinaii, Clara C. Chen, Jorge A. Carrasquillo, Millie Whatley, Iffat Chowdhury, Ahmed M. Gharib, and Lynnette K. Nieman

With **improved imaging techniques** over time, anatomical studies including CT (thinner 1.2 mm sections, chest) and MRI (3-T magnet) may better detect smaller tumors. This study did not address this question directly, because patients did not receive CT scans of different slice thickness at the same visit, and few had both 1.5- and 3-T MRI. Also, use of CT coregistration with PET may enhance its utility.

PT ID	CT	MRI	OCT	PET	Number detected by test combination
Patients with four studies performed (CT, MRI, OCT, PET)					
2, 3, 5, 6, 7, 9, 10, 12, 18, 29					10 patients: four tests positive
11, 23, 4, 13, 14, 16, 15, 17, 28, 8, 27				NEG	11 patients: two or three of four tests positive
Patient with CT, OCT, PET performed					
1					1 patient: 3/3 tests positive
Patients with CT, MRI, OCT performed					
19, 22					2 patients: 3/3 tests positive
20, 25, 21, 30					4 patients: 1-2 of 3 tests positive
Patients with CT and MRI performed					
24					1 patient: both tests positive
26		NEG			1 patient: one test positive



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Paziente di 48 anni

Aprile 2013 esegue una ecografia dell'addome in seguito ad una colica biliare.

In **anamnesi patologica remota** tonsillectomia, pregressa polmonite nel 2012 e sindrome depressiva.

Non allergie specifiche.

Non assume farmaci.

In **anamnesi familiare** storia di carcinoma della prostata con ripetizioni ossee (nonno materno).





Aprile 2013 (Viterbo)

Ecografia addominale: fegato con steatosi di grado medio. Nel VI segmento si rileva una formazione rotondeggiante con orletto ipoecogeno di 16 mm di diametro che merita ulteriori accertamenti. Nella colecisti formazione litiasica di 10 mm. Coledoco e vie biliari regolari.

Non alterazioni della vena porta e vena cava.

Pancreas, milza, aorta nella norma.

Reni regolari.

Prostata nei limiti.



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Aprile 2013 (Viterbo)



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Aprile 2013 (Viterbo)

Ecografia con mdc (CEUS) dell'addome superiore: dopo iniezione e.v. di Sonovue 1 f si documenta, in fase arteriosa, un intenso omogeneo "enhancement", seguito da rapido "wash out" in fase portale (durante la quale è evidente un sottile bordo di "enhancement"). La lesione si mantiene ipoecogena in fase tardiva.

CONCLUSIONI: Lesione ipervascolare del V-VI segmento epatico.



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Aprile 2013 (Viterbo)



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Maggio 2013 (Viterbo)

TC total body con/senza mdc: nella norma eccetto per la confermata alterazione focale ipodensa a carico del VI segmento di 13 mm di DM, del IV segmento di 4 mm e del VII segmento di 3 mm di DM. Colecisti litiasica (calcolo di circa 15 mm). Inoltre calcolo di 4 mm in sede caliceale media.



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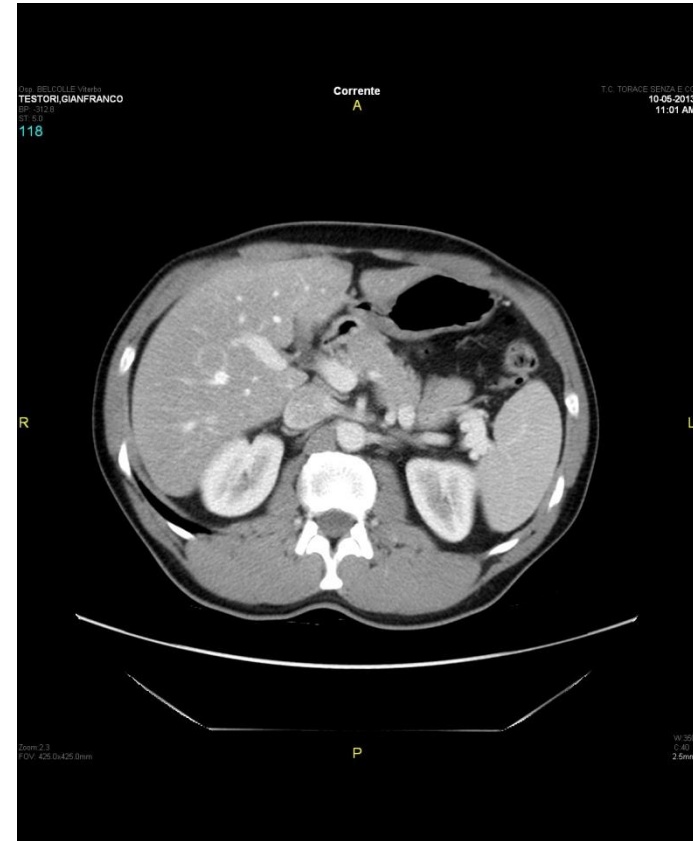
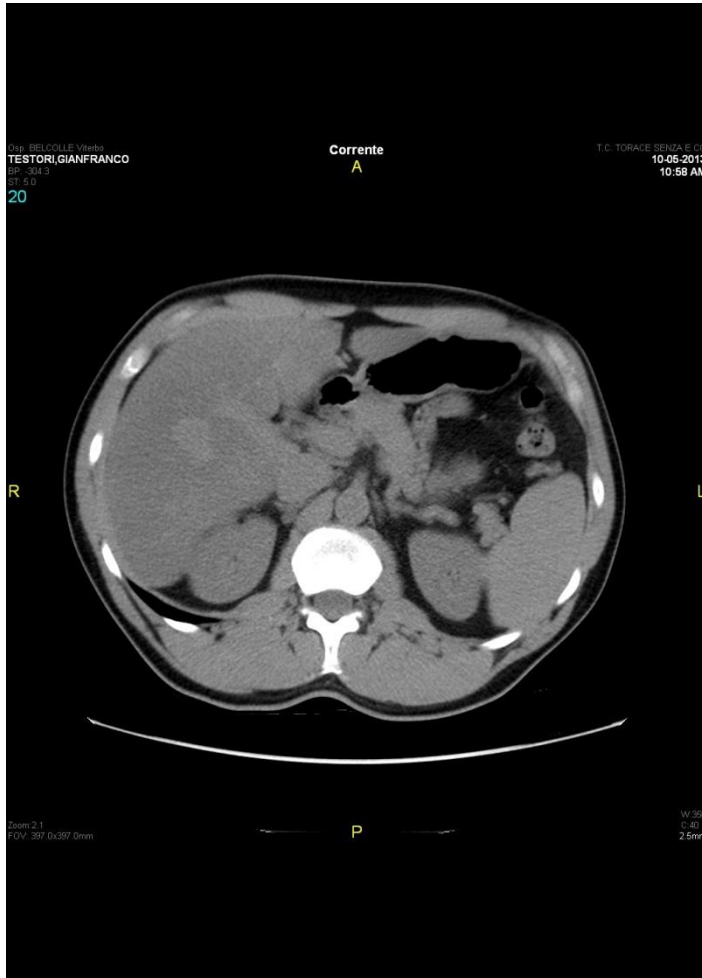
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Maggio 2013 (Viterbo)



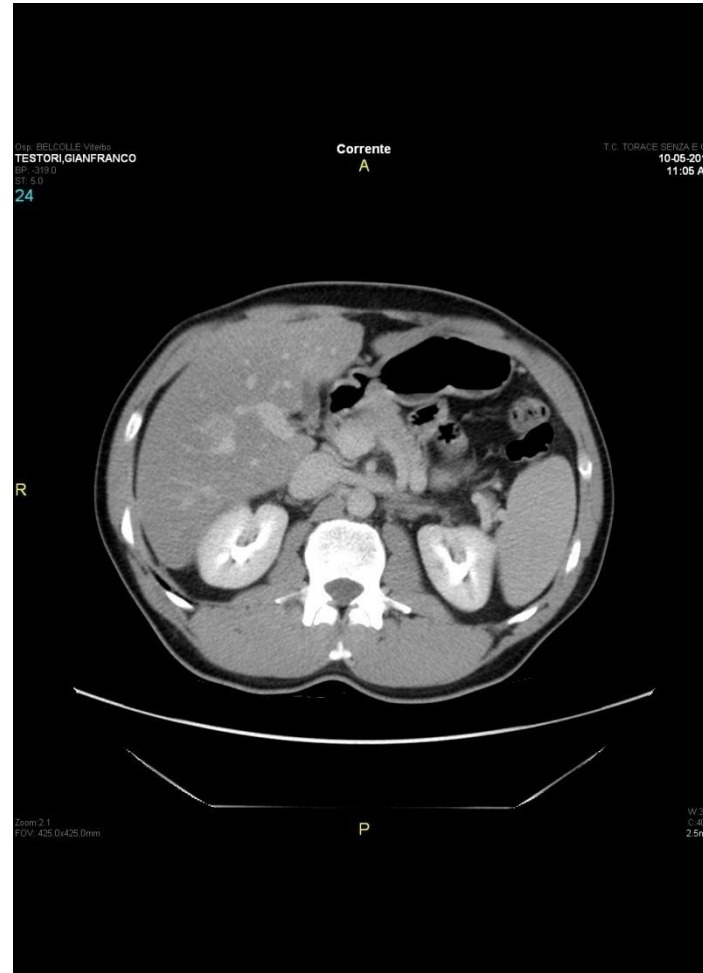
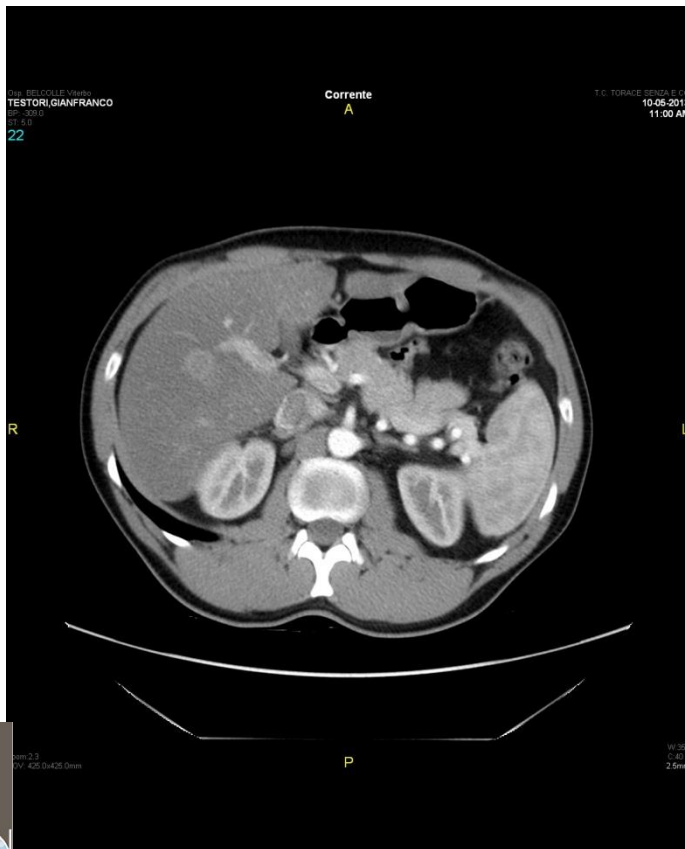
TC total body con/senza mdc



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Maggio 2013 (Viterbo)

TC total body con/senza mdc



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Maggio 2013 (Viterbo)



TC total body con/senza mdc



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Maggio 2013 (Viterbo)

Biopsia nodulo epatico di circa 2 cm con diagnosi istopatologia di frammenti tessuto epatico commisti a neoplasia con architettura trabecolare-acinare, CDx2 +, sinaptofisina +, cromogranina +, CK7 -, CK20 -. Aspetto immunomorfologico compatibile con neoplasia neuroendocrina.



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Maggio 2013

(1° visita endocrinologica Istituto Nazionale Tumori “Regina Elena”)

Revisione vetrini



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Maggio 2013

(1° visita endocrinologica Istituto Nazionale Tumori “Regina Elena”)

Revisione vetrini: materiale citologico incluso in paraffina costituito da minuti aggregati di epatociti tipici commisti ad aggregati di cellule con caratteristiche morfologiche ed immunofenotipiche (cromogranina +, sinaptofisina +) compatibili con tumore neuroendocrino ben differenziato (assenza di mitosi e di cellule ciclanti, **Ki 67 -**, nelle rare cellule presenti nel campione in esame).



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Giugno 2013 (IRE)



Cg A	14 ng/mL,
5-HIAA	11.9 mg/24h (<8),
NSE	10.8 ng/mL

Clinicamente

Habitus cushingoide

ACTH	450 pg/ml
cortisolo	600 mcg/dl



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Giugno 2013

Octreoscan total body (PTV) (111 In-Octreotide-111MBq e.v): non chiara iperfissazione focale del tracciante a carico della lesione clinicamente nota localizzata al VI segmento epatico; moderato iperaccumulo focale del radiofarmaco a livello del tratto di passaggio tra testa e corpo del pancreas; due ulteriori aree di lieve iperfissazione del radiofarmaco sono inoltre apprezzabili una in sede addominale (regione periombelicale) e l'altra in sede pelvica (regione ipogastrica) con incerta attribuzione delle sedi anatomiche corrispondenti (linfadenopatie mesenteriali? Anse intestinali?).



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Giugno 2013

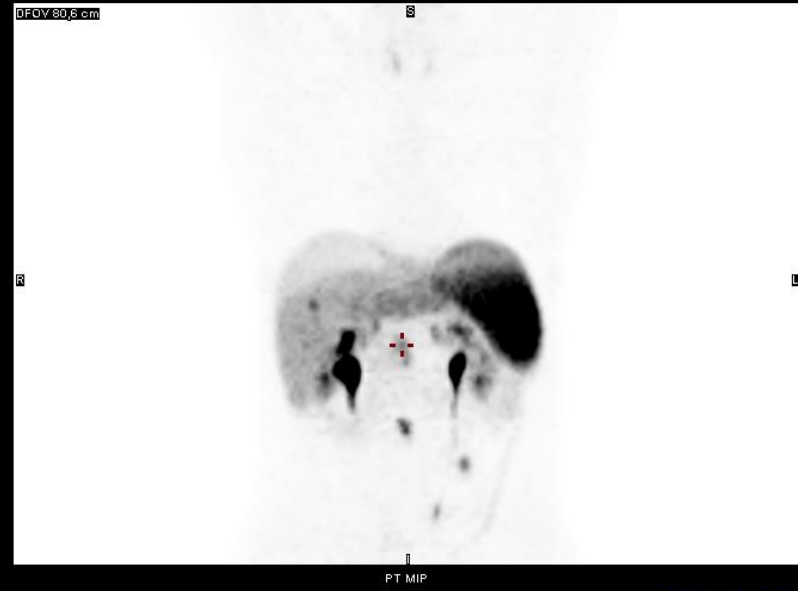
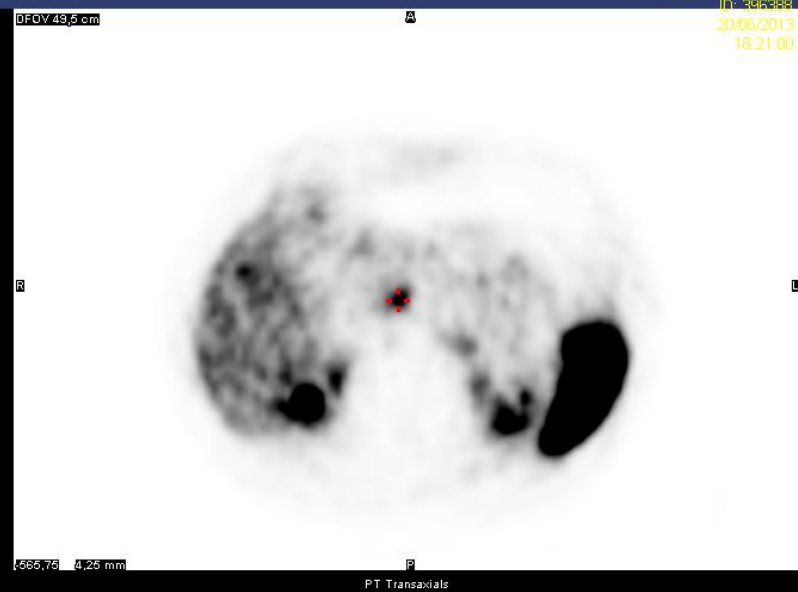
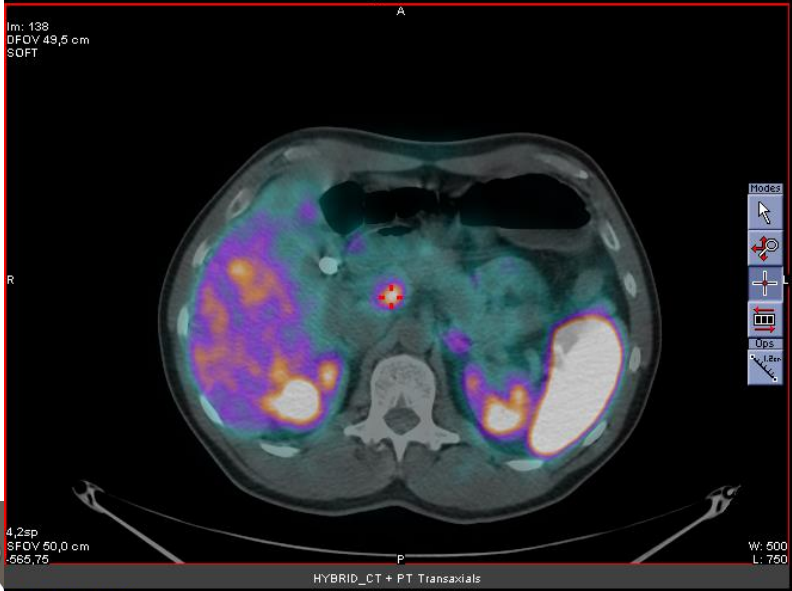
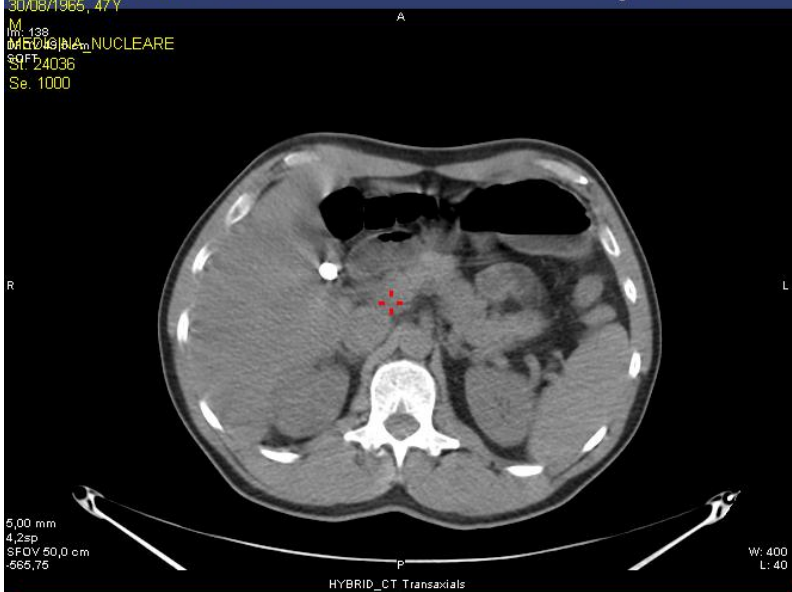
PET gallio 68 DOTATATE (IRST-IRCCS Meldola): presenza di lesioni esprimenti recettori per la somatostatina a livello epatico e adenopatico in sede interaortocavale in paziente con dubbia fissazione a livello pancreatico.



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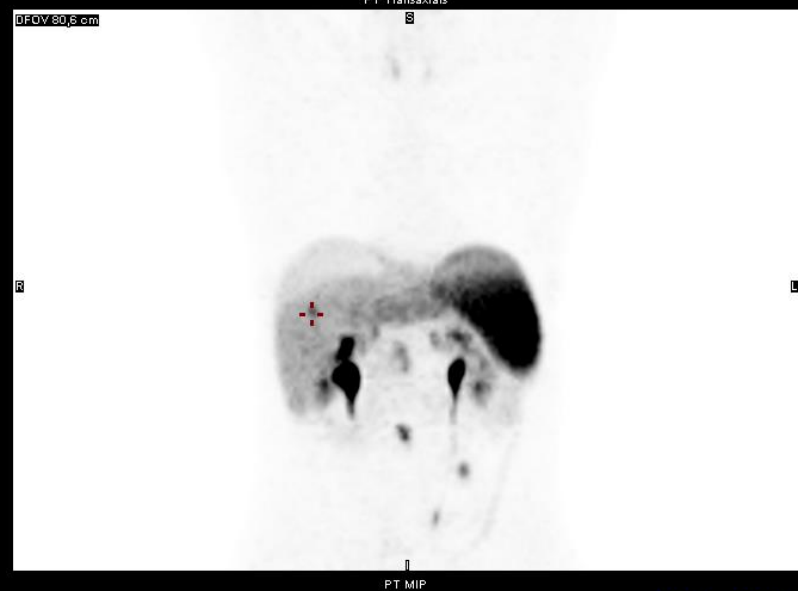
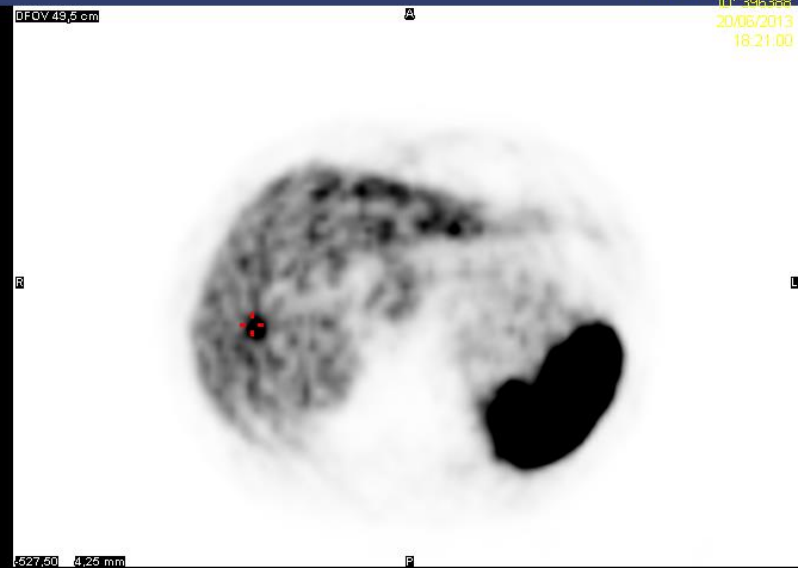
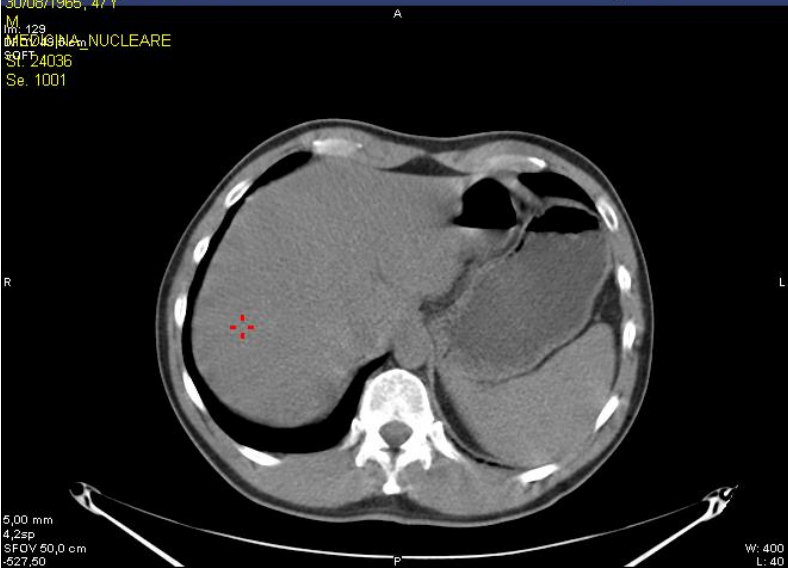


TESTORI GIANFRANCO
30/08/1965, 47Y
M
Im: 129
DFOV 49,5 cm
SFOV 50,0 cm
SFT
Se. 1001

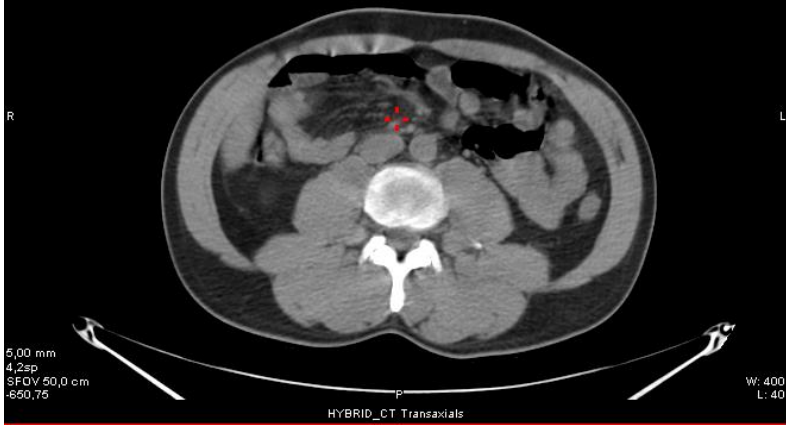
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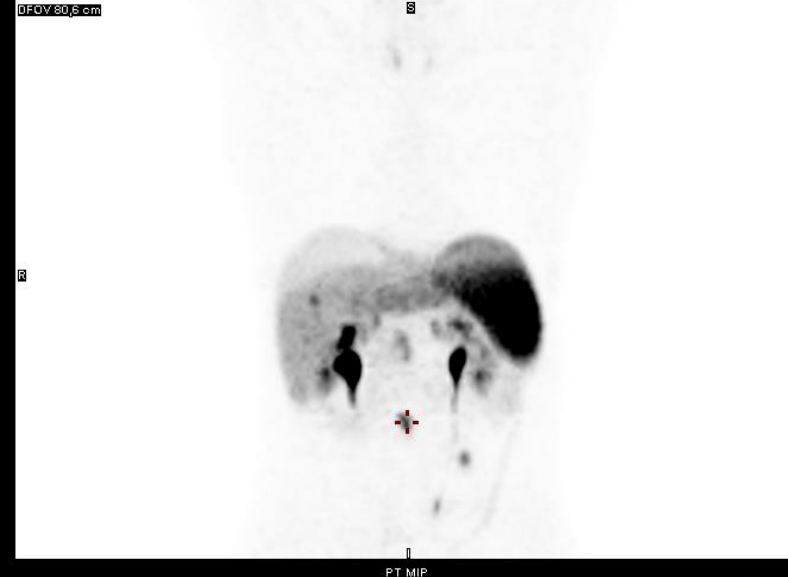
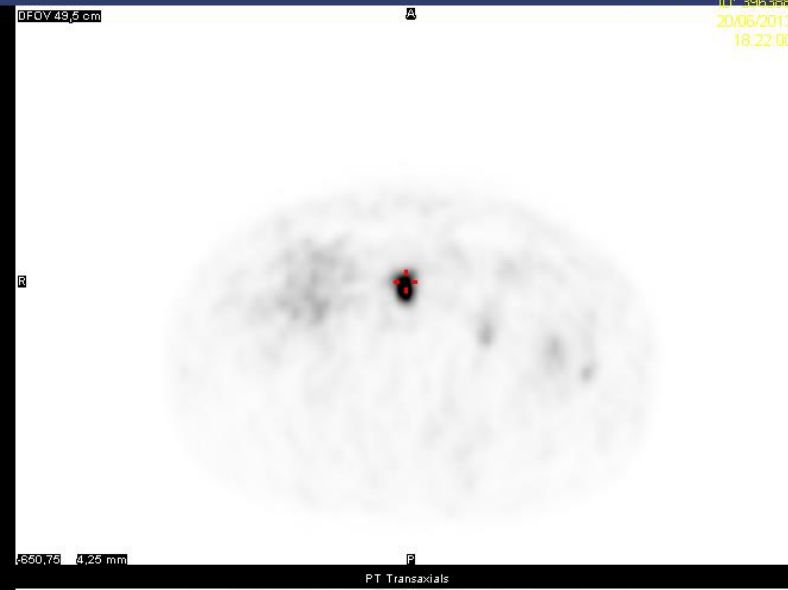
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Altogether to Beat Cushing's Syndrome
ABC
Viaggio alla (ri)scoperta della Sindrome di Cushing
Quarta Edizione
Napoli, 5-7 maggio 2015
Rodi S. Lucia



Pixel size : 100%



L = 128 W = 256

Altogether to Beat Cushing's Syndrome

ABC

Viaggio alla (ri)scoperta della Sindrome di Cushing
Quarta Edizione

Napoli, 5-7 maggio 2015
Rovelli S. Lucia





Ecoendoscopia (26 luglio 2013 – Ospedale Fatebenefratelli)

Colelitiasi con formazione litiasica di circa 13 mm, due piccole linfadenomegalie (circa 4-5 mm) in prossimità del confluente portale e assenza di significative alterazioni a carico del parenchima pancreatico.

Rettosigmoidocolonscopia (28 giugno 2013 – Ospedale S. Andrea)

Diverticoli al colon sx, alcuni anche all'ascendente.



Roberto BALDELLI M.D. Ph.D.



Unità Operativa di Endocrinologia ISTITUTO DI RICOVERO E CURA A CARATTERE SCIENTIFICO



PET 18 FDG

(10 luglio 2013 – Istituto Nazionale Tumori “Regina Elena”)

Modestissimo ipermetabolismo di natura da determinare a livello di testa/corpo del pancreas, non evidenza scintigrafica di tessuto ad elevata attività metabolica a livello epatico.



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Videocapsula (7 agosto 2013 – Istituto Nazionale Tumori “Regina Elena”)

..... a livello dell'ileo distale è presente una neoformazione depressa al centro ed estesa per circa $\frac{1}{4}$ della circonferenza del lume

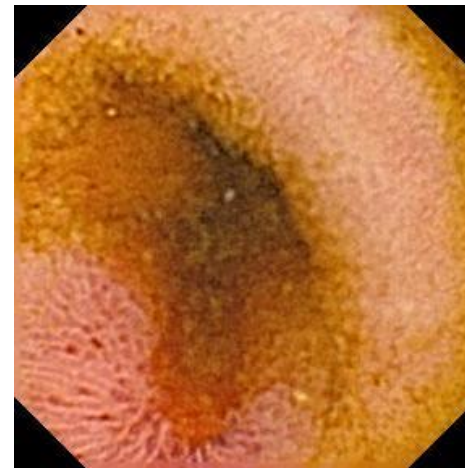
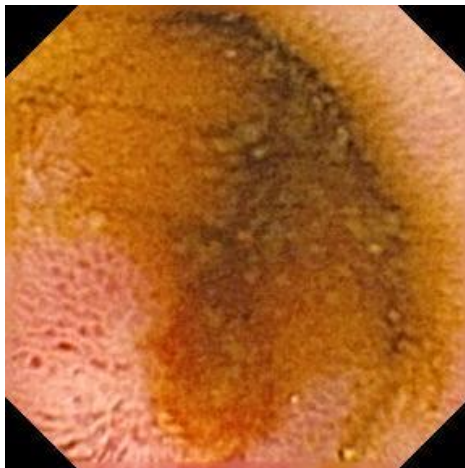
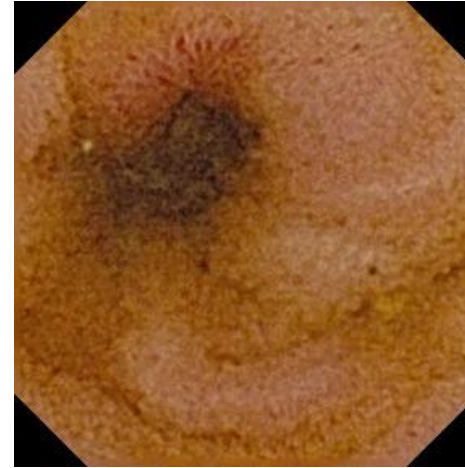


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Videocapsula (7 agosto 2013 – Istituto Nazionale Tumori “Regina Elena”)



TC addome completo con/senza mdc (entero-TC)

(10 settembre 2013 Istituto Nazionale Tumori

“Regina Elena”)



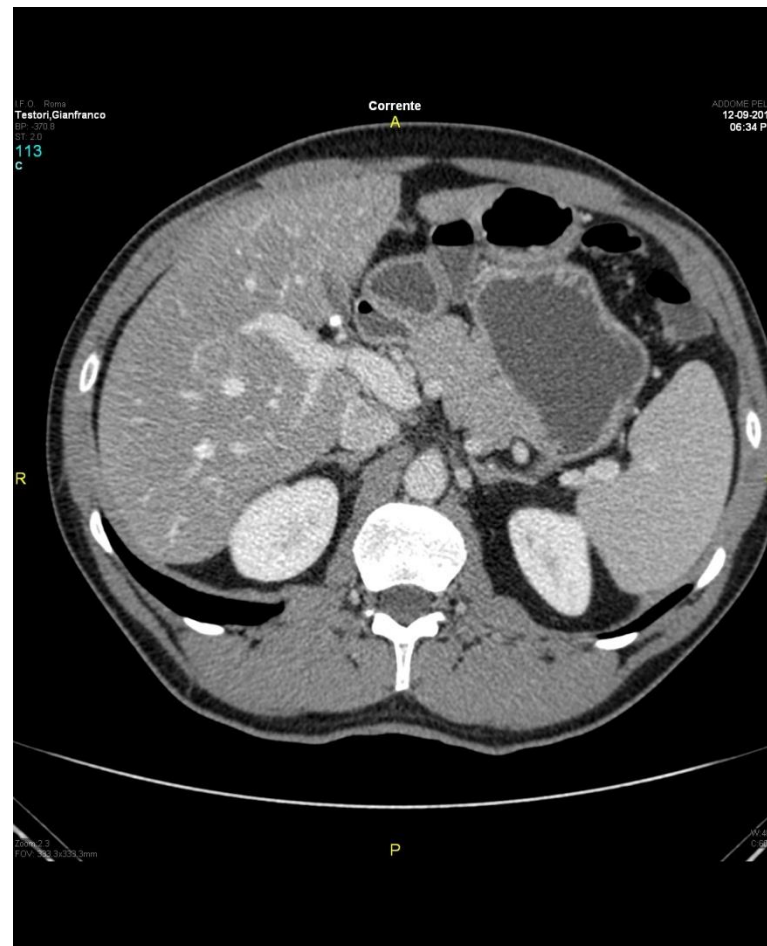
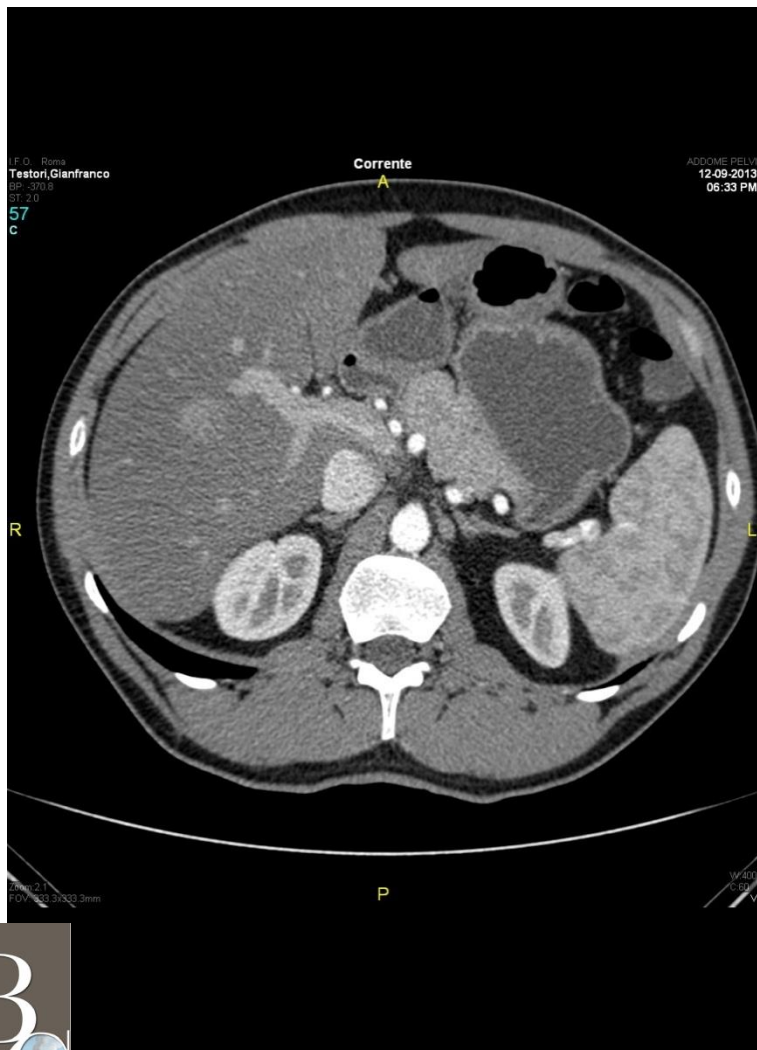
In corrispondenza di una delle ultime anse ileali ispessimento a manicotto di circa 2 cm iperdenso dopo mdc in fase tardiva, sospettato con la video capsula. Altro piccolo ispessimento è apprezzabile a livello dell'ultima ansa ileale. Piccoli linfonodi in sede mesenterica e lungo la catena di drenaggio del colon dx di circa 7 mm di DM. Al VII segmento epatico area di circa 2 cm che aumenta i valori di densità prevalentemente in fase portale per poi presentare un anello periferico di iperdensità con al centro un'area di iso-ipodensità. Apprezzabili piccole formazioni ipodense a livello epatico di presumibile significato cistico. Calcolosi della colecisti e nefrolitiasi a livello caliceale inferiore del rene dx. Surreni aumentati di volume in assenza di lesioni nodulari.



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TC addome completo con/senza mdc (entero-TC)

(10 settembre 2013 Istituto Nazionale Tumori "Regina Elena")



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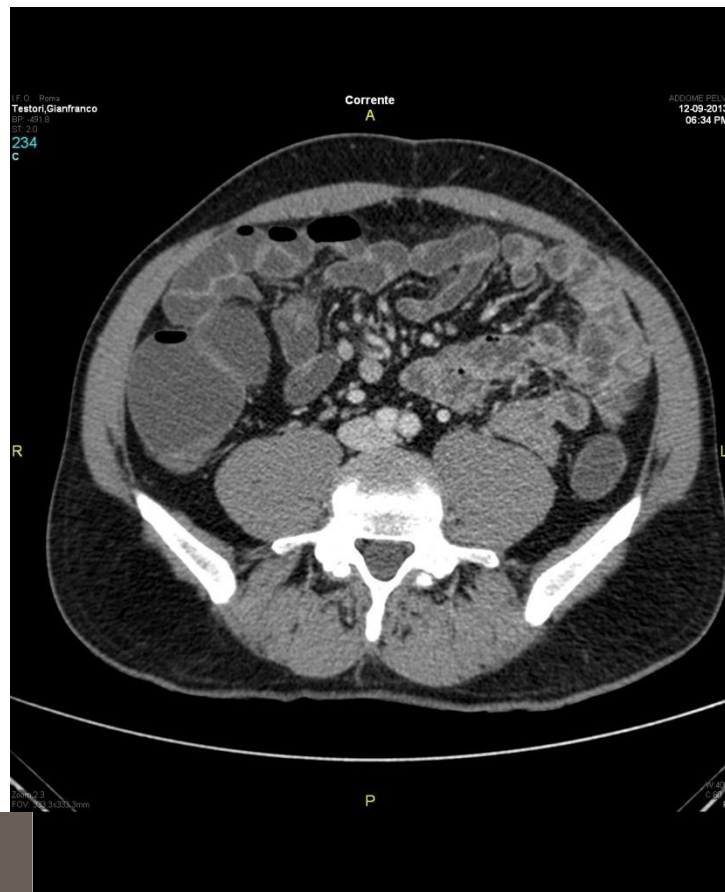
TC addome completo con/senza mdc (entero-TC)

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TC addome completo con/senza mdc (entero-TC)

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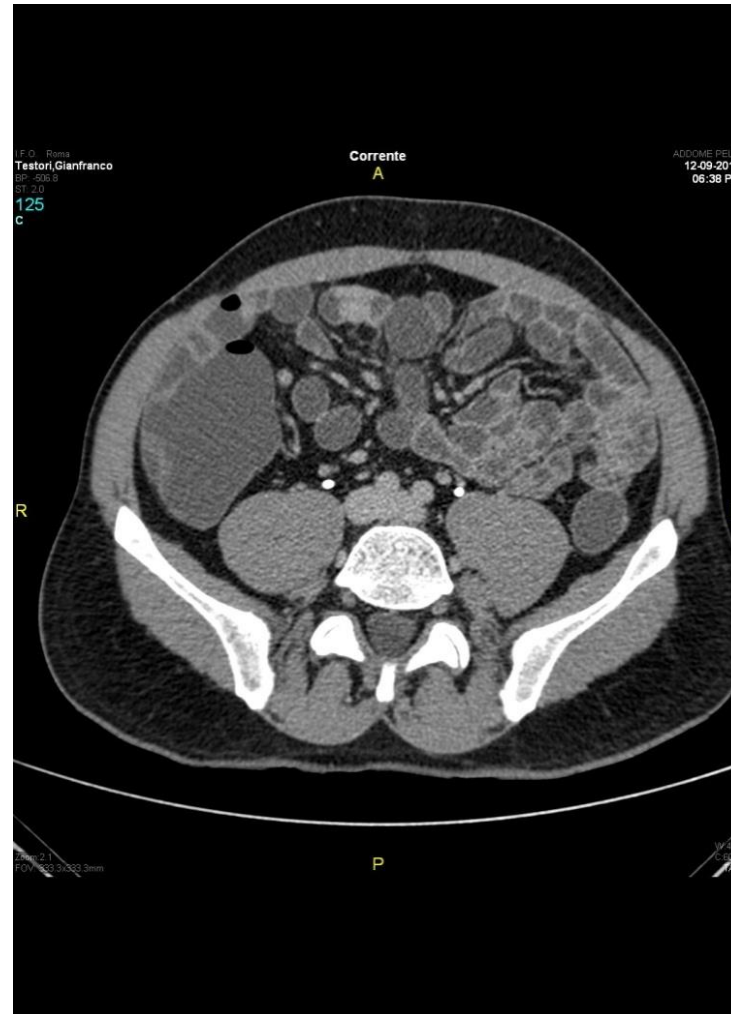
TC addome completo con/senza mdc (entero-TC)

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TC addome completo con/senza mdc (entero-TC)

(10 settembre 2013 Istituto Nazionale Tumori “Regina Elena”)



Roberto BALDELLI M.D. Ph.D.

EAS

(X-ray chest)
CT scan



Morpho-functional imaging
(for treatment)

MRI





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