

Complicanze cardiache dell' ipertiroidismo: gestione nei diversi setting assistenziali

Coordinatore: V. Giammarco

Moderatori: V. Giammarco, P. Limone



Roma,
9-11 novembre 2012

Il parte - 50 min (tolleranza fino a 55)

Endocrinologo e Cardiologo: ipertiroidismo e dolore toracico

Conduce V. Giammarco -

V. Triggiani - relazione introduttiva - **10 min**

Discussants: C. Cappelli, F. Tiratterra

Take-home messages 10 min (tolleranza fino 15 min) - P. Limone

presentazione caso clinico 1 - V. Giammarco 20 min

• 18/11/2011 Donna di 31aa (codice giallo al TRIAGE) ricoverata in UTIC per "*senso di oppressione intenso alla regione sternale*" e rilievo di lieve aumento della troponina rimasto costante nei controlli seriali delle successive 24 ore. *Non fa uso di estroprogestinici*. Riferisce di assumere Tapazole (1c) da alcune settimane per ipertiroidismo

Recenti dosaggi ormonali

• **TSH 0,02 FT4 4,8 (vn 0,6-1,12 ng/ml) FT3 13,3 pg/ml (vn 2,5-3,9)**

□ **domanda Tiratterra:** il valore diagnostico del DOLORE TORACICO, ECG , TROPONINA e loro combinazione

Risposta Tiratterra



- Problema centrale: distinguere se è espressione di una patologia importante oppure no.
- La valutazione diagnostica del dolore toracico acuto ha tre cardini: l'anamnesi, l'ecg, il dosaggio dei marcatori di danno miocardico (troponine).
- L'anamnesi rimane la tecnica più importante per poter distinguere tra le molte cause di dolore toracico (E. Braunwald).

- In caso di sovraslivellamento del tratto ST in almeno 2 derivazioni nel 75% dei casi IMA.
- In caso di sottoslivellamento del tratto ST o inversione dell' onda T in almeno 2 derivazioni nel 20% dei casi IMA.
- ECG seriati.

Markers di necrosi miocardica

- Sono proteine rilasciate nel sangue dal tessuto muscolare cardiaco necrotico.
- Individuano lesioni dei miociti indipendentemente dalla causa.
- Mioglobina, CK-MB massa, Troponine.

Troponine



- Cardiospecificità molto elevata.
- Aumenta dopo circa 6 ore.
- Raggiunge il picco dopo circa 24 ore.
- Può rimanere elevata per 10-14 giorni.

Troponine



Cause non cardiache di aumento della troponina:

- *Insufficienza renale cronica.*
- *Eventi cerebrovascolari acuti (emorragia subaracnoidea).*
- *Embolia polmonare.*
- *Broncopneumopatia cronica ostruttiva.*
- *Malattie acute non cardiache.*

S. Agewall Troponin elevation in coronary vs non coronary disease *European Heart Journal* (2011) 32, 404-411.

A. Tanindi Troponin elevation in conditions other than acute coronary syndromes *Vascular Health and Risk Management* 2011;7; 597-603



Universal definition of myocardial infarction

Kristian Thygesen, Joseph S. Alpert and Harvey D. White on behalf of the Joint ESC/ACCF/AHA/WHF Task Force for the Redefinition of Myocardial Infarction

Criteria for acute myocardial infarction

The term myocardial infarction should be used when there is evidence of myocardial necrosis in a clinical setting consistent with myocardial ischaemia. Under these conditions any one of the following criteria meets the diagnosis for myocardial infarction:

- Detection of rise and/or fall of cardiac biomarkers (preferably troponin) with at least one value above the 99th percentile of the upper reference limit (URL) together with evidence of myocardial ischaemia with at least one of the following:
 - Symptoms of ischaemia;
 - ECG changes indicative of new ischaemia [new ST-T changes or new left bundle branch block (LBBB)];
 - Development of pathological Q waves in the ECG;
 - Imaging evidence of new loss of viable myocardium or new regional wall motion abnormality.

- 18/11/2011 Donna di 31aa (codice giallo al TRIAGE) ricoverata in UTIC per *"senso di oppressione intenso alla regione sternale"* e rilievo di lieve aumento della troponina rimasto costante nei controlli seriali delle successive 24 ore. *Non fa uso di estroprogestinici*. Riferisce di assumere Tapazole da alcune settimane per ipertiroidismo
Recenti dosaggi ormonali
- TSH 0,02 FT4 4,8 (vn 0,6-1,12 ng/ml) FT3 13,3 pg/ml (vn 2,5-3,9)

Viene richiesta

***"CONSULENZA ENDOCRINOLOGICA
per nulla osta a coronarografia in
paziente con ipertiroidismo"***

▫ **domanda a Cappelli**

è appropriato richiedere la consulenza endocrinologica?

Risposta Cappelli



- Valutare rischio nell' utilizzare mezzo di contrasto iodato
- Ottimizzare la terapia tireostatica
- B-bloccante?

consulenza endocrinologo

-da 4 mesi ipertiroidismo di grado elevato da M. di Basedow con lieve oftalmopatia in OS, non attiva
- **Il "dolore", motivo del ricovero in UTIC, può rientrare nella sintomatologia correlata all'ipertiroidismo**

□ **Domanda a Cappelli:** è motivata questa affermazione?



Case Report

• Open Access •

Hyperthyroidism-associated coronary spasm: A case of non-ST segment elevation myocardial infarction with thyrotoxicosis

Xiao-Hu Kuang¹, Shu-Yang Zhang²

¹Chinese Academy of Medical Sciences & Peking Union Medical College & Peking Union Medical College Hospital, Beijing 100730, China

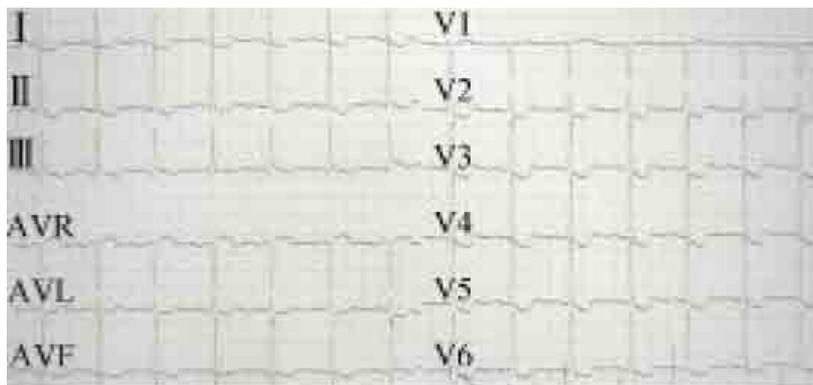
²Department of Cardiology, Peking Union Medical College Hospital, Shuai Fu Yuan 1, Beijing 100730, China

Abstract

Hyperthyroidism is associated with many heart diseases. Thyrotoxic state has a relationship with coronary spasm. We present a case of a non-menopausal woman with hyperthyroidism who complained of chest pain. The diagnosis of coronary spasm was confirmed by coronary angiography (CAG). She is treated well with anti-thyrotoxicosis and anti-anginal medication. We recommend not use CAG as the first diagnostic choice among the patients with medication-uncontrolled hyperthyroidism and chest pain.

J Geriatr Cardiol 2011; 8: 258–259. doi: 10.3724/SP.J.1263.2011.00258

Keywords: hyperthyroidism; coronary spasm; coronary angiography; beta-blocker



Continua CC 1



consulenza endocrinologo

-da 4 mesi ipertiroidismo di grado elevato da M. di Basedow con lieve oftalmopatia in OS, non attiva
- Il "dolore", motivo del ricovero in UTIC, può rientrare nella sintomatologia correlata all'ipertiroidismo

- **Stessa domanda a Tiratterra:** è azzardata questa affermazione?

Risposta Tiratterra



- Quanto è alta la possibilità che la paziente sia affetta da cardiopatia ischemica?

Conclusione consulenza endocrinologica

- *“....Comunque al momento è sconsigliabile la somministrazione di mezzo di contrasto iodato per eventuale coronarografia”*
- domanda a Cappelli: é giusta questa precauzione

SHORT COMMUNICATION

Risk of iodine-induced thyrotoxicosis after coronary angiography: an investigation in 788 unselected subjects

Gerhard Hintze, Oliver Blombach, Heike Fink¹, Ute Burkhardt² and Johannes Köbberling²

Department of Internal Medicine, Stormarn Hospital, Teaching Hospital of Luebeck University, Bad Oldesloe, Germany, ¹Division of Endocrinology, Department of Medicine, University of Essen, Essen, Germany and ²Department of Internal Medicine, Ferdinand-Sauerbruch-Klinik, Teaching Hospital of Duesseldorf University, Wuppertal, Germany

(Correspondence should be addressed to G Hintze, Department of Internal Medicine, Hospital Stormarn Schützenstrasse 55, 23843 Bad Oldesloe, Germany)

Abstract

In this study, the risk of iodine-induced thyrotoxicosis in unselected patients from an iodine-deficient area was investigated. The patients were consecutively enrolled. Thyroid hormone values and urinary iodine excretion were determined before, as well as 1, 4 and 12 weeks after iodine contamination by coronary angiography. Two of 788 unselected patients developed hyperthyroidism within 12 weeks. The two patients did not belong to a risk group for iodine-induced thyrotoxicosis (i.e. old people, patients with goiter or possible thyroid autonomy, low TSH). Both patients had normal TSH levels at baseline and ultrasound of the thyroid was without evidence of nodules. The study shows that in euthyroid unselected patients from an iodine-deficient area short-term iodine contamination by contrast media rarely leads to hyperthyroidism. On account of these facts, prophylactic therapy, e.g. by perchlorate or thiamazole, is not generally recommended, because the risk of side-effects is perhaps even greater than the risk of iodine-induced thyrotoxicosis.

0021-972X/04/15.00/0
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doi: 10.1210/jc.2004-0728

Scintigraphy for Risk Stratification of Iodine-Induced Thyrotoxicosis in Patients Receiving Contrast Agent for Coronary Angiography: A Prospective Study of Patients with Low Thyrotropin

EVA FRICKE, HARALD FRICKE, ELKE ESDORN, ANNETT KAMMEIER, OLIVER LINDNER, KNUT KLEESIEK, DIETER HORSTKOTTE, AND WOLFGANG BURCHERT

Institute of Molecular Biophysics, Radiopharmacy, and Nuclear Medicine (E.F., H.F., E.E., A.K., O.L., W.B.); Institute for Laboratory and Transfusion Medicine (K.K.); and Department of Cardiology (D.H.), Heart and Diabetes Center North Rhine-Westphalia, D-32545 Bad Oeynhausen, Germany

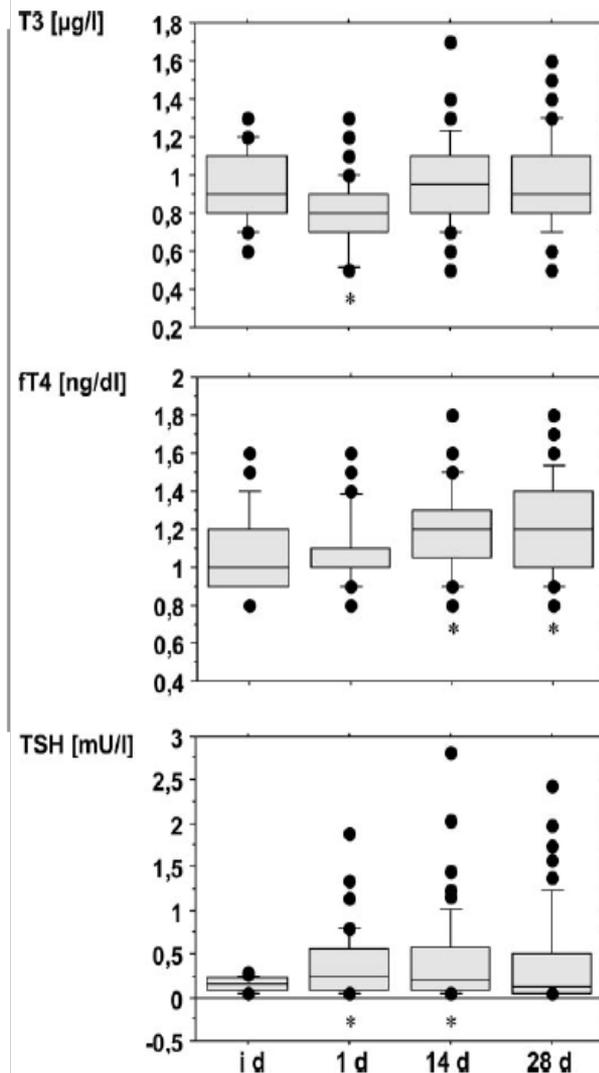
The risk of iodine-induced thyrotoxicosis in euthyroid patients receiving iodine-containing contrast agents is known to be low, but data on this risk in patients with latent hyperthyroidism are scarce. We investigated the role of thyroid scintigraphy using Tc-99m preceding the application of iodine-containing contrast material to estimate the risk of iodine-induced thyrotoxicosis in patients with low levels of TSH.

In a prospective study on 91 patients, thyroid scintigraphy was performed before coronary angiography (CA). In patients with technetium thyroid uptake (TCTU) less than 1%, CA was done without prophylactic drugs (n = 56). Patients with TCTU

greater than 1% were treated either with 900 mg of perchlorate or, depending on the autonomous volume, combined with 20 to 60 mg thiamazole.

In the 56 patients with TCTU less than 1%, no case of iodine-induced hyperthyroidism occurred within 4 wk after CA. In the patients who received prophylactic drugs, two cases of mild thyrotoxicosis were observed.

Our data suggest that in patients with low levels of TSH, the risk of hyperthyroidism after application of iodine-containing contrast media is negligible if TCTU is less than 1%. In these patients, CA can be performed without administration of prophylactic drugs. (*J Clin Endocrinol Metab* 89: 6092-6096, 2004)



Continua CC 1

Consulenza endocrinologica: indicazioni terapeutiche

- *Per raggiungere al più presto il blocco della elevata produzione ormonale: TAPAZOLE 4 COMPRESSE SUBITO INSIEME A DELTACORTENE 50 MG da ridurre fino a sospendere in tempi brevi*
- **domanda a Cappelli:** sei d'accordo su questa impostazione terapeutica?

Continua CC 1

risposta Cappelli

Diagnostic criteria for thyroid storm*

Thermoregulatory dysfunction		Cardiovascular dysfunction	
Temperature (°F °C)		Tachycardia	
99 to 99.9 37.2 to 37.7	5	99 to 109	5
100 to 100.9 37.8 to 38.2	10	110 to 119	10
101 to 101.9 38.3 to 38.8	15	120 to 129	15
102 to 102.9 38.9 to 39.4	20	130 to 139	20
103 to 103.9 39.4 to 39.9	25	≥140	25
≥104.0 >40.0	30	Atrial fibrillation	10
Central nervous system effects		Heart failure	
Mild	10	Mild	5
Agitation		Pedal edema	
Moderate	20	Moderate	10
Delirium		Bibasilar rales	
Psychosis		Severe	15
Extreme lethargy		Pulmonary edema	
Severe	30	Precipitant history	
Seizure		Negative	0
Coma		Positive	10
Gastrointestinal-hepatic dysfunction			
Moderate	10		
Diarrhea			
Nausea/vomiting			
Abdominal pain			
Severe	20		
Unexplained jaundice			

* A score of 45 or more is highly suggestive of thyroid storm; a score of 25 to 44 supports the diagnosis; and a score below 25 makes thyroid storm unlikely.
Adapted from: Burch HB, Wartofsky L. Life-threatening thyrotoxicosis. Thyroid storm. *Endocrinol Metab Clin North Am* 1993; 22:263.



4/8/2011 **Pronto Soccorso**

• Donna di 33 aa di origine dominicana (da alcuni aa lavora in Italia come infermiera) viene visitata in PS con codice giallo al TRIAGE ("palpitazione e dolore toracico oppressivo"). Recente diagnosi di tireotossicosi non ancora in trattamento. Non fa uso di estroprogestinici

CONSULENZA CARDIOLOGICA in PS

- *lamenta dolore toracico puntorio alla regione sottoscapolare sx variabile con atti respiratori;*
- *ECG tachicardia sinusale (133 min) con anomalie della ripolarizzazione diffuse, più evidenti in sede posteriore*
- *EcoCG: assenza di evidenti anomalie della cinesi; sezioni sinistre nei limiti, sezioni destre dilatate con IT medio-moderata. **PAPS (pulmonary artery systolic pressure) 45 mmHg**. In corso markers di necrosi miocardica.*
 - **(PAPS valore normale <25 mmHg)**

domanda a Tiratterra



- *che ne pensi di questo quadro clinico e del valore dell'ECG ed ecocardiogramma*

(valutazione complessiva della consulenza cardiologica tenendo conto del valore diagnostico dei dati disponibili in contemporanea: caratteristica del dolore toracico, ECG, Ecocardiogramma, PAPS elevata)

risposta Tiratterra



- Dolore puntorio variabile con gli atti respiratori.
- ECG
- Ecocardiogramma:
 - sezioni sinistre nei limiti
 - sezioni destre dilatate
 - insufficienza tricuspидale medio-moderata
 - PAPS 45 mm Hg

Embolia polmonare ?

Continua CC 2



Ricovero UTIC

Pervengono risultati ESAMI LABORATORIO eseguiti in PS

- TROPONINA e MIOGLOBINA normali
- **D DIMERO 4225 NG/ML (vn <500)**
- **TSH 0,01 FT4 5,6 (vn 0,6-1,12 ng/ml) FT3 21,3 pg/ml (vn 2,5-3,9)**

□ **domanda Tiratterra** *utilità diagnostica del D Dimero*

D-Dimer Testing in Laboratory Practice

Armando Tripodi^{1*}

Tabella 1

Condizioni caratterizzate da aumentate concentrazioni di D-dimero

Età avanzata	Ictus
Periodo neonatale	Arteriopatia periferica
Gravidanza	Aneurismi
Ospedalizzazione	Insufficienza cardiaca congestizia
Disabilità	Emolisi (anemia falciforme)
Infezioni	Emorragie
Tumori	Sindrome acuta respiratoria
Chirurgia recente	Epatopatie e malattie renali
Traumi, ustioni	Malattie infiammatorie intestinali
Coagulazione intravascolare disseminata	Terapia trombolitica
Tromboembolismo venoso	Dissezione aortica
Cardiopatía ischemica	

risposta Tiratterra

Utilità diagnostica del D-Dimero

- I D-dimeri sono un prodotto finale della lisi della fibrina e rappresentano un marker di attivazione della coagulazione.
- E' utile nel sospetto di tromboembolismo venoso grazie al suo alto valore predittivo negativo.

Tre giorni fa in reparto

	ETA'	D-dimero	diagnosi
• In 6 pazienti era stato misurato il D-dimero in Pronto Soccorso.	68 aa	4267	Diabete mellito scompensato
	84 aa	2081	Cardiopatía ischemica
• V.n. 0-550 ng/ml	75 aa	1815	BPCO riacutizzata
	83 aa	4540	sepsi
	87 aa	3858	Insufficienza cardiaca
	68	2737	Emorragia digestiva

domanda a Cappelli



- *Quali rapporti fra ipertiroidismo e ipertensione polmonare?*

SPECIAL FEATURE

Clinical Case Seminar

Pediatric Pulmonary Arterial Hypertension and Hyperthyroidism: A Potentially Fatal Combination

Christine M. Trapp, Robert W. Elder, Adrienne T. Gerken, Aviva B. Sopher, Shulamit Lerner, Gaya S. Aranoff, and Erika B. Rosenzweig

Divisions of Pediatric Endocrinology, Diabetes, and Metabolism (C.M.T., A.B.S., S.L., G.S.A., A.T.G.) and Pediatric Cardiology (R.W.E., E.B.R.), Department of Pediatrics, Children's Hospital of New York Presbyterian, Columbia University College of Physicians and Surgeons, New York, New York 10032

Context: Patients with pulmonary arterial hypertension (PAH) who develop hyperthyroidism are at risk for acute cardiopulmonary decompensation and death.

Cases and Setting: We present a series of eight idiopathic PAH/heritable PAH pediatric patients who developed hyperthyroidism between 1999 and 2011. Institutional Review Board approval was obtained; informed consent was waived due to the retrospective nature of the series. All eight patients were receiving iv epoprostenol; five of the eight patients presented with acute cardiopulmonary decompensation in the setting of hyperthyroidism. In the remaining three patients, hyperthyroidism was detected during routine screening of thyroid function tests. The one patient who underwent emergency thyroidectomy was the only survivor of those who presented in cardiopulmonary decline.

Evidence Synthesis: Aggressive treatment of the hyperthyroid state, including emergency total thyroidectomy and escalation of targeted PAH therapy and β -blockade when warranted, may prove lifesaving in these patients. Prompt thyroidectomy or radioactive iodine ablation should be considered for clinically stable PAH patients with early and/or mild hyperthyroidism to avoid potentially life-threatening cardiopulmonary decompensation.

Conclusions: Although the association between hyperthyroidism and PAH remains poorly understood, the potential impact of hyperthyroidism on the cardiopulmonary status of PAH patients must not be ignored. Hyperthyroidism must be identified early in this patient population to optimize intervention before acute decompensation. Thyroid function tests should be checked routinely in patients with PAH, particularly those on iv epoprostenol, and urgently in patients with acute decompensation or symptoms of hyperthyroidism. (*J Clin Endocrinol Metab* 97: 2217–2222, 2012)

REVIEW ARTICLE

Hyperthyroidism and Pulmonary Hypertension: An Important Association

*Sailaja Vallabhajosula, MD, Saba Radhi, MD, Cihan Cevik, MD, Raed Alalawi, MD,
Rishi Raj, MD and Kenneth Nugent, MD*

Abstract: Pulmonary hypertension is a complex disorder with multiple etiologies. The World Health Organization Group 5 (unclear multifactorial mechanisms) includes patients with thyroid disorders. The authors reviewed the literature on the association between hyperthyroidism and pulmonary hypertension and identified 20 publications reporting 164 patients with treatment outcomes. The systolic pulmonary artery (PA) pressures in these patients ranged from 28 to 78 mm Hg. They were treated with antithyroid medications, radioactive iodine and surgery. The mean pretherapy PA systolic pressure was 39 mm Hg; the mean posttreatment pressure was 30 mm Hg. Pulmonary hypertension should be considered in hyperthyroid patients with dyspnea. All patients with pulmonary hypertension should be screened for hyperthyroidism, because the treatment of hyperthyroidism can reduce PA pressures, potentially avoid the side-effects and costs with current therapies for pulmonary hypertension and limit the consequences of untreated hyperthyroidism. However, the long-term outcome in these patients is uncertain, and this issue needs more study. Changes in the pulmonary circulation and molecular regulators of vascular remodeling likely explain this association.

Key Indexing Terms: Graves' disease; Hyperthyroidism; Pulmonary hypertension. [Am J Med Sci 2011;342(6):507–512.]

[J Thromb Haemost. 2010 Oct;8\(10\):2176-81. doi: 10.1111/j.1538-7836.2010.03993.x.](#)

Increased risk of pulmonary embolism among patients with hyperthyroidism: a 5-year follow-up study.

[Lin HC](#), [Yang LY](#), [Kang JH](#).

School of Health Care Administration, Taipei Medical University, Taiwan.

Abstract

BACKGROUND: Although studies have indicated that hyperthyroidism is associated with hypercoagulability, most such studies have focused only on examining the incidence of venous thrombosis. As far as we know, no study has attempted to explore the risk of pulmonary embolism (PE) among patients with hyperthyroidism.

OBJECTIVE: Using a nationwide population-based dataset, this study was aimed at estimating the risk of PE among hyperthyroidism patients during a 5-year period, as compared with non-hyperthyroidism patients during the same period.

METHODS: Data sourced from the Taiwan Longitudinal Health Insurance Database were analyzed. The study included 8903 patients with hyperthyroidism as a study cohort and 44,515 randomly selected patients without hyperthyroidism as a comparison cohort. Stratified Cox proportional hazard regressions were used to compute the 5-year PE-free survival rate between these two cohorts.

RESULTS: Of the total of 53,418 patients, 41 patients (0.08%) were identified as having PE during the follow-up period, 14 from the study cohort (0.16% of the hyperthyroidism patients) and 27 comparison patients (0.06% of patients from the comparison cohort). After adjustment for geographic region, monthly income, hypertension, diabetes, hyperlipidemia, peripheral vascular disease, coronary heart disease, cancer, recent surgery, recent fracture, pregnancy and the use of anticoagulants, the risk of having PE during the 5-year follow-up period was 2.31 times greater (95% confidence interval 1.20-4.45, $P=0.012$) for patients with hyperthyroidism than for patients in the comparison cohort.

CONCLUSION: We found an increased risk of PE in patients with hyperthyroidism. Clinicians should be aware of this increased risk.

Richiesta consulenza endocrinologica urgente per nulla osta ad esecuzione di scintigrafia polmonare

Consulenza endocrinologica *Conclusione*

- *ipertiroidismo da gozzo diffuso autoimmune con oftalmopatia di grado lieve in fase non attiva*
 - *Ritengo che la sintomatologia cardio vascolare sia del tutto giustificata dall'ipertiroidismo.*
- **Domanda a Cappelli** *è azzardata questa affermazione?*

Risposta Cappelli



- *Si, ritengo che seppur “giustificata” sia azzardata.*
- *Il sospetto rimane; la possibilità di embolia polmonare che non è stata ancora esclusa.*
- *La diagnosi di ipertensione polmonare da ipertiroidismo RIMANE una diagnosi di esclusione*

Domanda a Tiratterra



Roma,
9-11 novembre 2012

- *la diagnostica dell' embolia polmonare*

- La valutazione clinica (sia che si avvalga del giudizio clinico empirico o di criteri predittivi convalidati) consente di valutare la probabilità che sia presente embolia polmonare.
- Anamnesi
- Esame obiettivo
 - Rx torace
 - ECG
 - EGA

WELLS Score (PE)

• Clinical signs and symptoms compatible with DVT	3
• PE judged to be the most likely diagnosis	3
• Surgery or bedridden for more than 3 days during past 4 weeks	1.5
• Previous DVT or PE	1.5
• Heart rate > 100 min ⁻¹	1.5
• Hemoptysis	1
• Active cancer (treatment ongoing or within previous 6 months, or palliative treatment)	1

≤ 4 : **LOW** (or "PE Unlikely") pretest probability

4.5 - 6 : **MODERATE** pretest probability

> 6 : **HIGH** pretest probability

Wells PS, et al. *Thromb Haemost* 2000; 83: 416-20

Kearon C, et al. *Ann Intern Med* 2006; 144: 812-21

Risposta Tiratterra



- L' angioTAC “multislice” è attualmente considerata il gold standard per la valutazione dell' embolia polmonare acuta
(J Thor Imaging, 2012; 27(2): w28-31).
- La scintigrafia ventilo-perfusoria continua ad essere una valida e consolidata metodica nei casi di embolia polmonare.

Guidelines on the diagnosis and management of acute pulmonary embolism European Heart Journal (2008) 29, 2276-2315.

Continua CC 2

- *Iniziare Tapazole 2cx2/die. In attesa dell'efficacia terapeutica del tireostatico (non prima di 15 gg), controllare la tachicardia con Tenormin.*
- 30/9/2011 CONTROLLI AMBULATORIALI DOPO 1 MESE (endocrinologo e cardiologo)
- **Endocrinologo** Dopo iniziale miglioramento clinico e biochimico, è stato necessario aumentare il Tapazole 3x2c die per ripresa dell' ipertiroidismo
- **Cardiologo (+EcoCardio):** non disturbi cardio vascolari; PAPS 42 mmHG
- Scintigrafia polmonare e Doppler venoso arti inferiori: **NEGATIVI**

Domanda a Cappelli



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- *ci sono problemi alla esecuzione della scintigrafia polmonare e angio TAC nel corso di ipertiroidismo*

Risposta Cappelli



Per quanto ho letto tutti i mezzi di contrasto contengono iodio (anche quelli non iodati).

Quindi ...

controlli endocrinologici successivi

- Nei controlli endocrinologici successivi per resistenza al trattamento con tireostatici somministrata terapia radioisotopica 2 volte
- 13/6/2012 (dopo 10 mesi dal 1° ricovero)
 - normalizzazione della funzione tiroidea
 - controllo EcoCardio: PAPs 25 mmHg
- Attualmente in remissione

