

66 Year old AAM with Amiodarone Induced Thyroiditis (AIT)

**Endorama
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Fellow**

HPI:

- ❖ 66 year old AAM with PMH of HTN, HLD, Afib, CHF (EF 10-15%) on amiodarone, stage V CKD, DMII, CAD s/p CABG
- ❖ Was in is usual state of health until 3 months ago.
- SOB + CP + palpitation and found to in Afib with RVR + CHF exacerbation
- Hypotensive and hypoxic, admitted to the ICU on mechanical ventilation and vasopressors.
- TFT >> TSH 0.02 (0.4 – 5.5), FT4 3.4 (0.8-1.8), FT3 5.1 (1.8 – 4.6)
- Bedside Ultrasound >> homogeneous gland with no increase in vascularity, no nodules.
- Amiodarone was d/c and started on hydrocortisone iv + Methimazole + Lopressor IV PRN.
- 3 days later patient developed vasculitic reaction and Methimazole was d/c

Discharge after 3 weeks on the following medications:

Propranolol 20 mg po Q8hrs, Prednisone 40 mg daily

1 month later

- **Admitted again to the same hospital for CP/SOB (missed dialysis)**
- **TFT showed TSH 0.03, FT4 3.9, FT3 4.9**
- **HR was controlled with BB**
- **Still on prednisone 40 mg daily**

This admission:

➤ Same (SOB, CP) >> pulmonary edema, hypotension, Afib with RVR >>> intubated + vasopressors

ROS: Non obtainable

PMH:

- ✓ HTN
- ✓ CAD (s/p CABG 2005)
- ✓ ESRD on HD
- ✓ Afib
- ✓ CHF (EF 10-15%)
- ✓ DM type II
- ✓ HLD

Family History:

- ✓ CAD >> father, maternal grandmother.
- ✓ Breast Ca mother
- ✓ DM (mother)

Surgical history:

- ✓ CABG

Social history

- ✓ Quit smoking 7 years ago, no alcohol, no illicit drugs.

Home medications

- ✓ Lisinopril 20 mg daily
- ✓ propranolol 20 mg q8hrs
- ✓ Lantus 30 units daily
- ✓ Novolog 8 units with meals + MDSSI (1-5)
- ✓ Warfarin 5 mg po daily
- ✓ Prednisone 40 mg daily

Current medications

- ✓ Dopamine + NE + Epi drip
- ✓ Hydrocortisone 100 mg Q8hrs

On Examination

- **Vitals:** BP 100/52 | Pulse 85, no fever, RR 18, BMI 28.2
- **General:** sedated intubated on mechanical ventilator
- **HEENT:** normocephalic , no exophthalmos,
- **Neck:** supple, no LN enlargement, I don't appreciate any thyromegaly
- **CVS/Pulm:** **inspiratory crackles bilateral**, S1 + S2, irregular pulse, systolic murmur at the aortic area.
- **Abd:** soft lax, diminished bowel sounds.
- **Skin:** warm, no rash
- **Neuro:** difficult to assess

Outside labs

Test/results	3/2013	4/2013
WBC	14,200	
HB	10.7	
Plt	140	
K	3.7	
Carbon Dioxide	17	
BUN	78	
Cr	6.8	
ALP	150	101
ALT	2100	467
AST	1780	397
<i>TSH (0.4 – 5.0)</i>	0.022	0.03
<i>FT4 (0.7 – 1.8)</i>	3.4	3.9
<i>FT3 (1.8 – 4.6)</i>	5.1	4.9

Admission labs

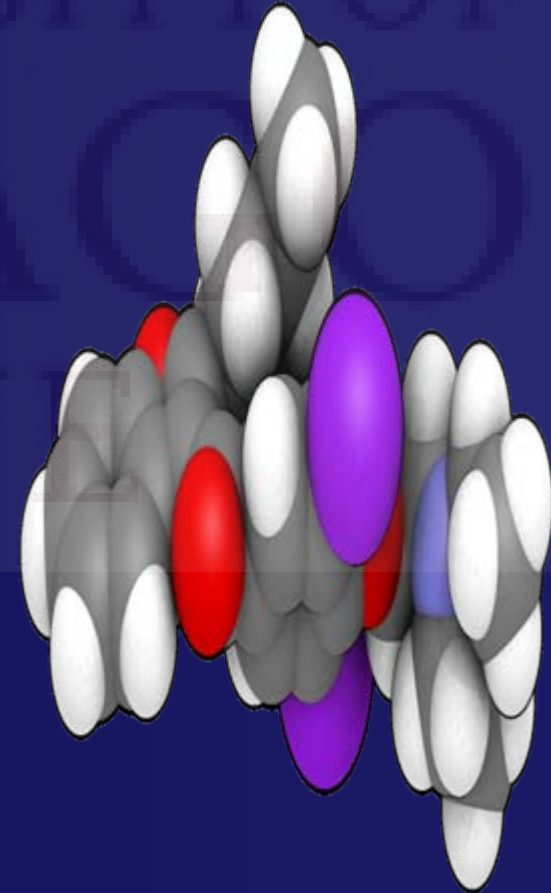
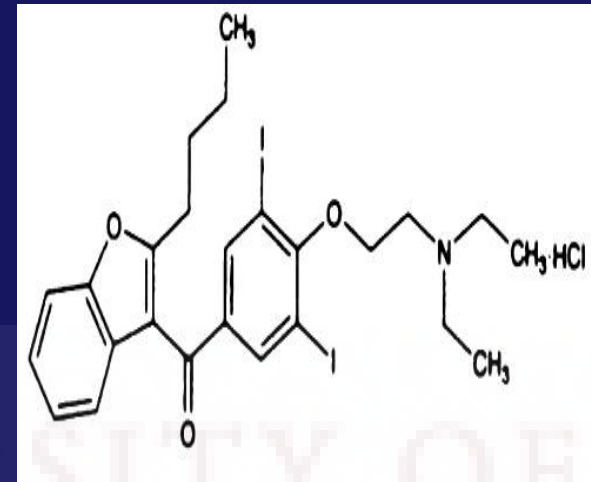
	6/13/2013	6/13/2013	
WBC	16,300		
Hb	10.1		
Plt	97		
K	4.4		
Carbon dioxide	14		
BUN	81		
Cr	8.6		
ALP	175		
ALT	3179		
AST	2231		
TSH (0.4 – 5.0)	0.011	TSI	- ve
FT4 (0.7 – 1.8)	5.7	TPO Ab	- ve
FT3 (1.8 – 4.6)	8.5	Tg Ab	- Ve

Clinical Qs

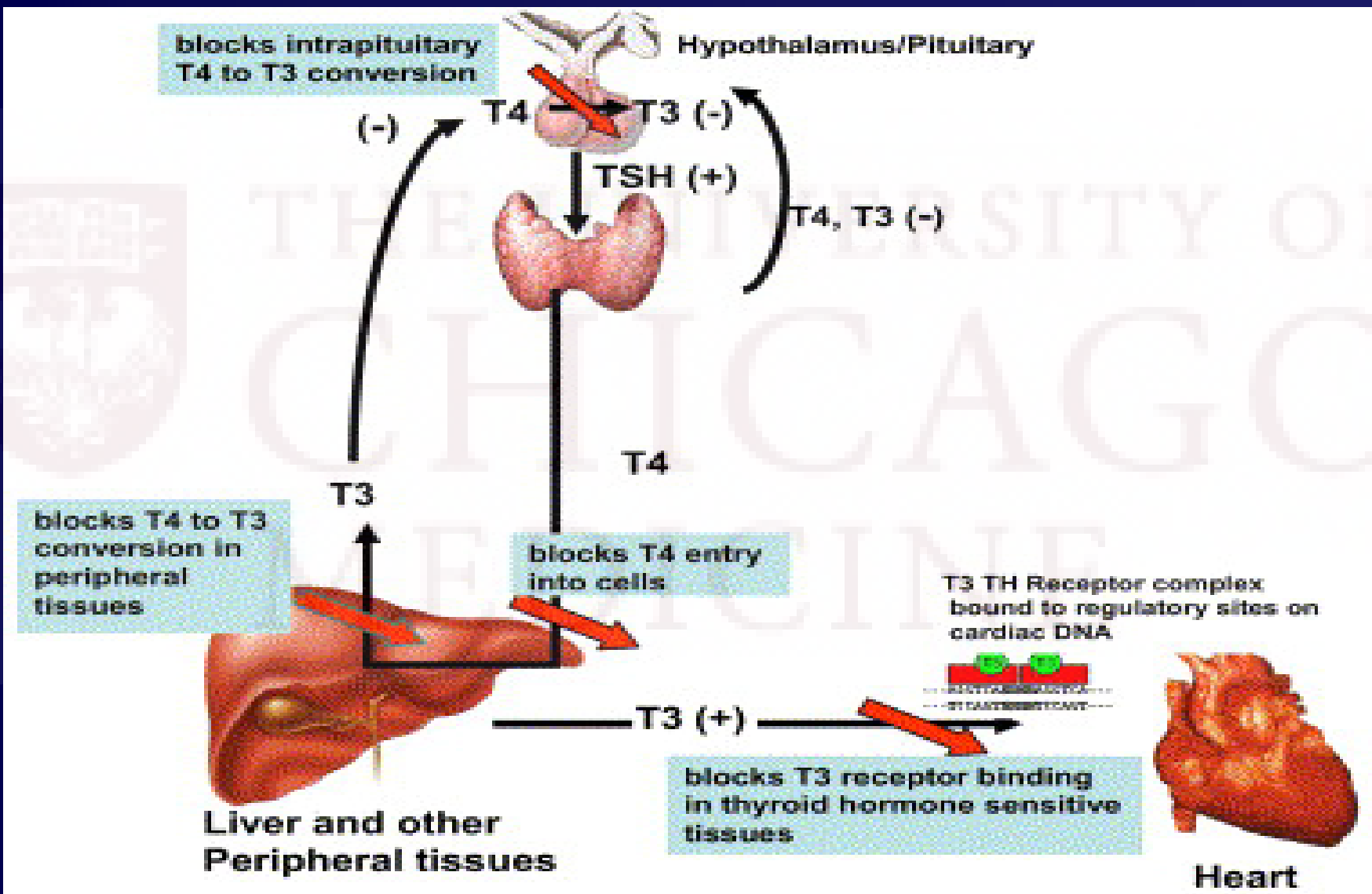
- 1). What is prevalence of AIT?
- 2). What is the treatment for AIT?
- 3). Do we need to discontinue Amiodarone?
- 4). Is there any role for Doppler US to predict therapeutic response?

Properties of amiodarone

- Contains **37%** iodine by weight.
- Extensively stored in multiple tissues, including adipose tissue—1/2 life of weeks to several months.
- Amiodarone concentrations in the thyroid have been found to be as much as **20 times** the blood concentrations.



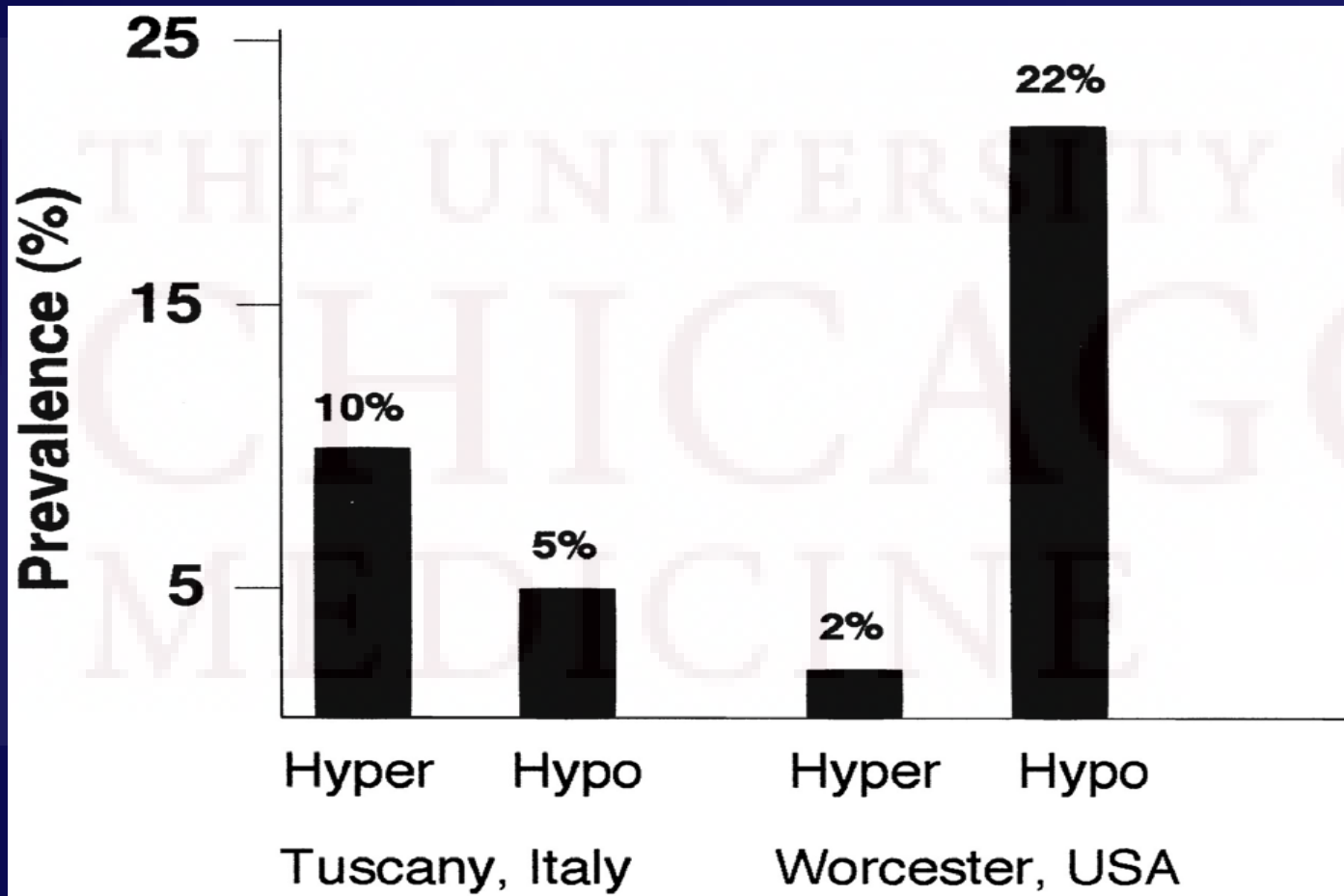
Amiodarone and the Thyroid



Type 1 vs Type 2

	Type 1	Type 2
Goiter	++	+/-
Reactive iodine uptake	+/-	-
IL-6	-	++
CFDS	Normal/increase	Decrease
Response to steroid	-	+
Response to thionamide	+	-

Prevalence of amiodarone-induced hyperthyroidism and hypothyroidism in an iodine-deficient area of Northern Tuscany, Italy, and in an iodine-sufficient area of Worcester, Massachusetts



Treatment of amiodarone-induced thyrotoxicosis, a difficult challenge: results of a prospective study.

Bartalena L, Brogioni S, Grasso L, Bogazzi F, Burelli A, Martino E.

Source

Istituto di Endocrinologia, University of Pisa, Italy. 1996 Aug;81(8):2930-3

24 AIT patients, 12 type I and 12 type II, were evaluated prospectively. Sex, age, severity of thyrotoxicosis, and cumulative amiodarone doses were similar.

Type 1 received methimazole (30 mg/day) +/- perchlorate (0.5 gms BID). IL-6 and FT3 were normalized within 4 weeks

Type 2 received prednisone (40 mg/day, tapered over 3 months). IL-6 and FT3 with marked reduction after 2-5 days. Type II had higher serum IL-6.

Suggested mixed form (both Type 1 and Type 2) of AIT

Conclusion

Type I; treat with MMI and perchlorate

Type II; treat with prednisone.

Mixed AIT; may require MMI, perchlorate, and prednisone.

Four Type 1 patients did not respond to MMI and perchlorate. Three of these had prompt resolution “within a few days” with prednisone. One patient was resistant to all treatments

Do we need to discontinue Amiodarone?

Successful Treatment of Amiodarone-Induced Thyrotoxicosis

Faizel Osman, MB, MRCP; Jayne A. Franklyn, MD, PhD, FRCP; Michael C. Sheppard, PhD, FRCP; Michael D. Gammage, MD, FRCP. 2002, 105:1275-1277, UK

- 28 patients median age 64.1 with AIT in the U.K. (14 type I and 14 type II)
- 16 patients continue amiodarone (mainly with VT) and 12 stop amiodarone (mainly with SVT)
- All pts treated with Carbimazole

Conclusion:

- The total dose of CBZ required to induce euthyroidism was no different if amiodarone was continued or stopped.
- The rate of improvement in thyroid function tests no different (at 6 weeks: FT4 25.6 pmol/L in patients in whom amiodarone was continued versus 22.2 pmol/L in those in whom it was stopped.
- Relapse rate, **two (13%)** of those who continued amiodarone had relapse of AIT, compared with **3 (25%)** who stopped.

Using color flow Doppler in predicting therapeutic response.

Heterogeneity of amiodarone-induced thyrotoxicosis: evaluation of color-flow Doppler sonography in predicting therapeutic response.

Wong R, Cheung W, Stockigt JR, Topliss DJ.

Department of Endocrinology and Diabetes, Alfred Hospital, Melbourne, Victoria, Australia

24 patients with AIT in an iodine-replete environment

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graph TD; A[24 patients with AIT in an iodine-replete environment] --> B[13 patients showed CFDS 0]; A --> C[11 patients with CFDS I-III]; B --> D[7 of 13 (58%) were prednisolone-responsive]; C --> E[1 of 7 (14%) was prednisolone-responsive.]; D --> F[Euthyroidism was achieved twice as rapidly in patients with CFDS 0 than those with CFDS I-III];
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13 patients showed CFDS 0

11 patients with CFDS I-III

7 of 13 (58%) were prednisolone-responsive

1 of 7 (14%) was prednisolone-responsive.

Euthyroidism was achieved twice as rapidly in patients with CFDS 0 than those with CFDS I-III

Back to my patient

- Unfortunately, patient died 4 hrs after arrival (V-tach >>> V-Fib)

AAACE recommendations

A. Clinically euthyroid:

- Observation or thionamide alone.

B. Mild to moderate symptoms:

1. Consider CFDS, if available, and base initial treatment accordingly (Glucocorticoids for CFDS 0 and Thionamides for CFDS I-III.)
2. Begin thionamide, if no response within 1 month
Consider adding glucocorticoids.
3. Begin thionamide and glucocorticoid. If no clear decline in thyroid hormones at 2-3 weeks, taper steroids and continue thionamide.

C. Severe symptoms (clinically unstable)

- Begin thionamides and glucocorticoids.

- Withdraw amiodarone based on clinical relevance of amiodarone to the treated disease **not the degree of hyperthyroidism.**
- If steroids are used and appear to be effective, taper slowly over 2-3 months.
- Patient with no response to the medical management, **thyroidectomy may be considered.**

References

- 1 Martino E, Bartalena L, Bogazzi F & Braverman LE. The effects of amiodarone on the thyroid. *Endocrine Reviews* 2001 22 240–254.
- 2 O'Sullivan AJ, Lewis M & Diamond T. Amiodarone-induced thyrotoxicosis: left ventricular dysfunction is associated with increased mortality. *European Journal of Endocrinology* 2006 154 533–536.
- 3 Bogazzi F, Bartalena L, Tomisti L, Dell'Unto E, Cosci C, Sardella C, Tanda ML, Lai A, Gasperi M, Aghini-Lombardi F & Martino E
Potassium perchlorate only temporarily restores euthyroidism in patients with amiodarone-induced hypothyroidism who continue amiodarone therapy. *Journal of Endocrinological Investigation* 2008 31 515–519.
- 4 Bogazzi F, Bartalena L, Gasperi M, Braverman LE & Martino E. The various effects of amiodarone on thyroid function. *Thyroid* 2001 11 511–519.
- 5 Bogazzi F, Dell'Unto E, Tanda ML, Tomisti L, Cosci C, Aghini-Lombardi F, Sardella C, Pinchera A, Bartalena L & Martino E. Long-term outcome of thyroid function after amiodarone-induced thyrotoxicosis, as compared to subacute thyroiditis. *Journal of Endocrinological Investigation* 2007 29 694–699.



Thank you

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