中华医学会核医学分会
技术与继续教育学组

中华医学会核医学分会第十一届委员会
技术与继续教育学组
系列专家讲座

亚急性甲状腺炎

Subacute Granulomatous Thyroiditis

董萍
天津医科大学第二医院核医学科
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亚急性甲状腺炎

（Subacute granulomatous thyroiditis，SAT）

- 亚急性甲状腺炎（简称亚甲炎），又称病毒性甲状腺炎、DeQuervain甲状腺炎、肉芽肿性甲状腺炎或巨细胞性甲状腺炎等，1904年由DeQuervain首先报道；
- 本病近年来逐渐增多，临床变化复杂，可有误诊及漏诊，且易复发，但常为自限性（病程3-6个月），多数患者可得到痊愈；
- 本病可因季节或病毒流行而有人群发病的特点。

病因及流行病学

- 发病前患者常有上呼吸道感染史，发病常随季节变动、且具有一定的流行性；
- 患者血中有病毒抗体存在（抗体的效价高度和病期相一致），最常见的是柯萨奇病毒抗体，其次是腺病毒抗体、流感病毒抗体、腮腺炎病毒抗体等；
- 中国人、日本人的亚急性甲状腺炎与HLA-Bw35有关，提示对病毒的易感染性具有遗传因素；
- 多见于中年妇女。

病程特点

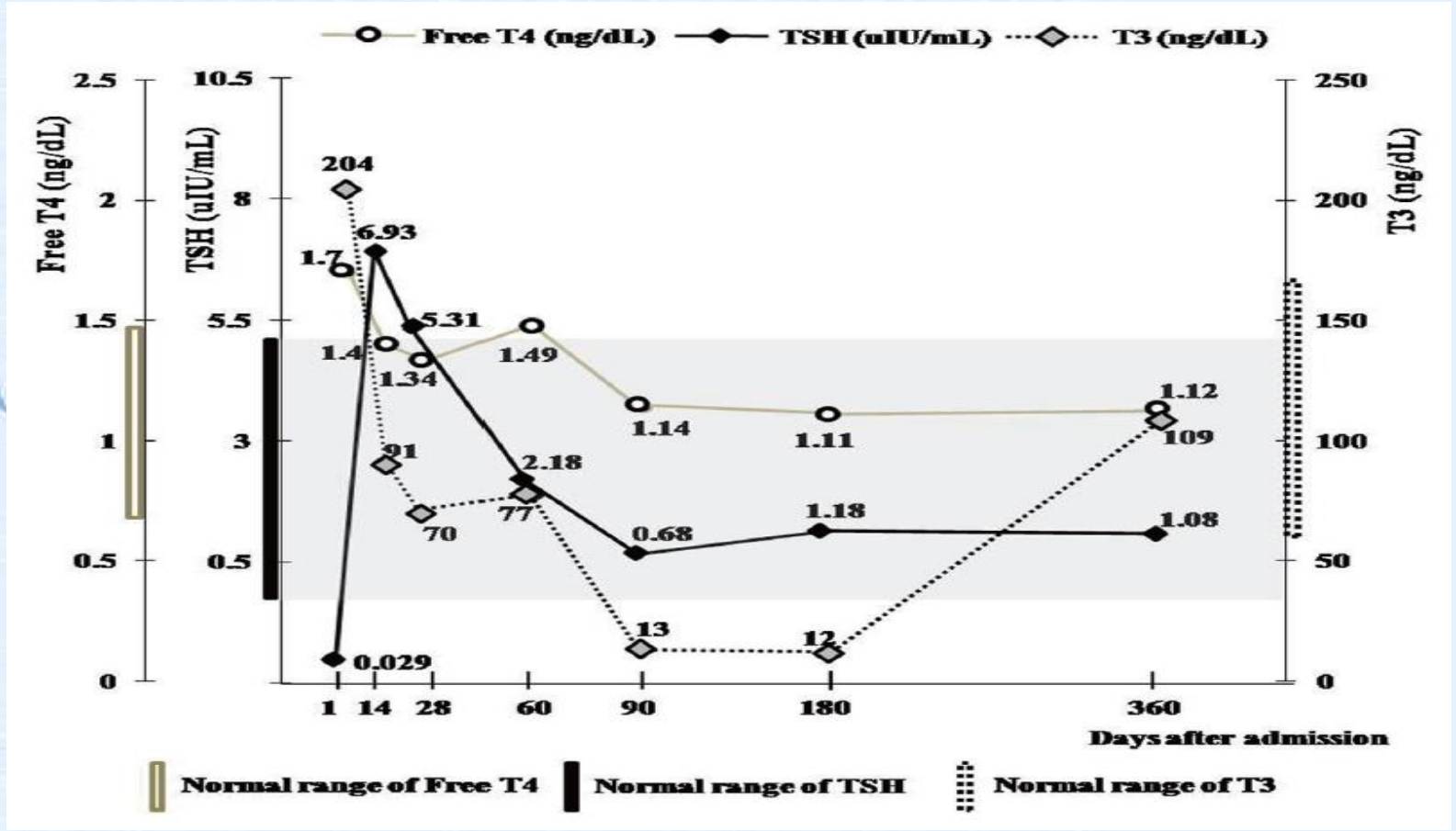
- 典型者病程可分为三期：

  ①早期：甲状腺机能亢进期

  ②中期：甲状腺机能减退期

  ③恢复期

亚甲炎的甲状腺激素变化过程



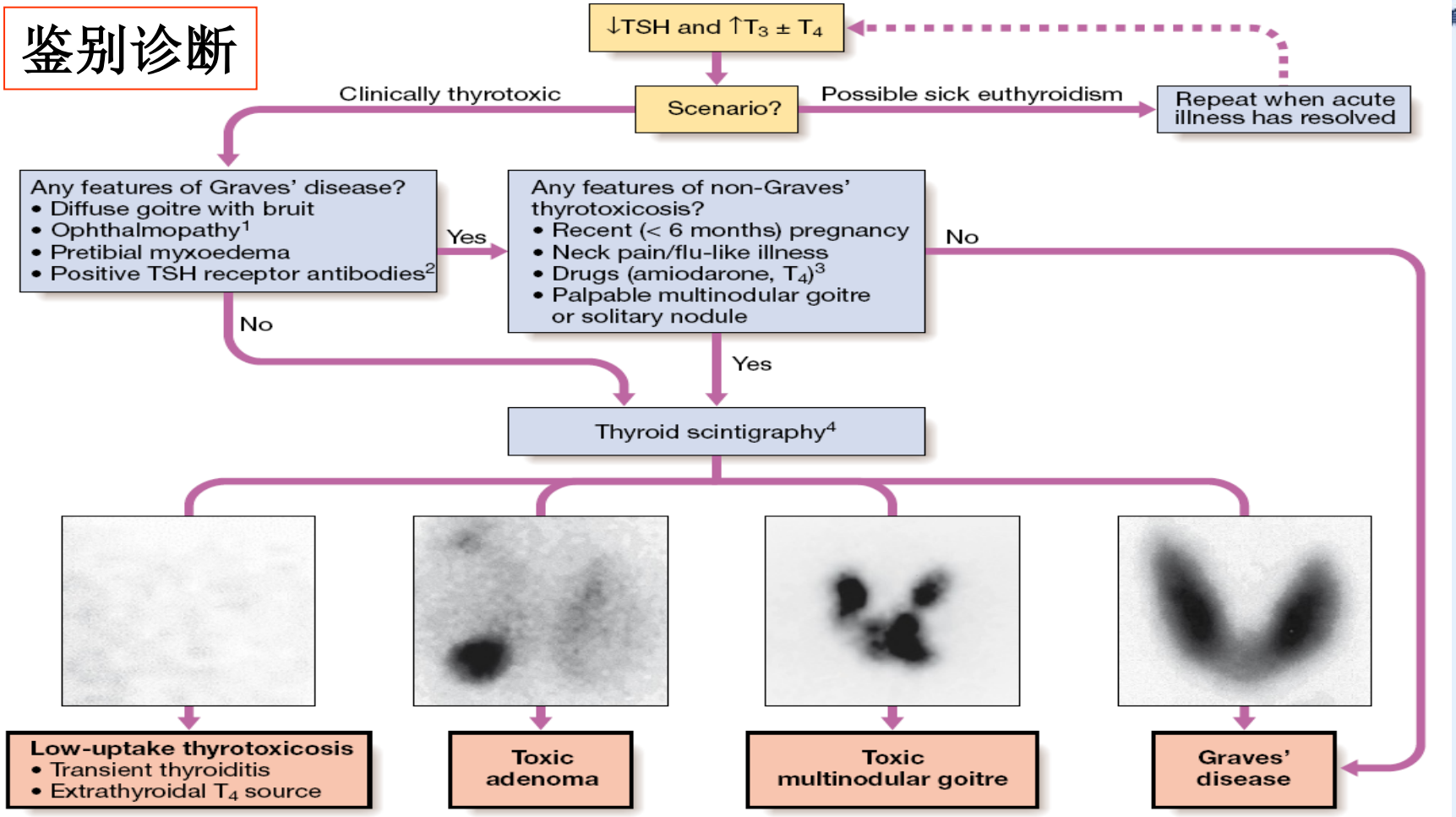
诊断标准

- 甲状腺肿大、疼痛、质硬、触痛，常伴上呼吸道感染的症状和体征：发烧、乏力、食欲不振、颈部淋巴结肿大等
- 血沉加快
- 一过性甲亢表现
- 甲状腺摄^{131}I取率下降
- 甲状腺自身抗体阴性或低滴度
- 甲状腺穿刺或活检，有多核巨细胞或肉芽肿改变

符合以上其中4条可以诊断。

Iitaka M，Momotuni N，Ishii J，et al. Incidence of subacute thyroiditis recurrences after a prolonged latency：24-year survey. J Clin Endocrinol Metab，1996，81：466-469.

鉴别诊断


↓TSH and ↑T_3 ± T_4
Scenario?
Clinically thyrotoxic
Possible sick euthyroidism
Repeat when acute illness has resolved
Any features of Graves' disease?
• Diffuse goitre with bruit
• Ophthalmopathy[1]
• Pretibial myxoedema
• Positive TSH receptor antibodies[2]
Yes
No
Any features of non-Graves' thyrotoxicosis?
• Recent (< 6 months) pregnancy
• Neck pain/flu-like illness
• Drugs (amiodarone, T_4)[3]
• Palpable multinodular goitre or solitary nodule
No
Yes
Thyroid scintigraphy[4]
Low-uptake thyrotoxicosis
• Transient thyroiditis
• Extrathyroidal T_4 source
Toxic adenoma
Toxic multinodular goitre
Graves' disease

鉴别诊断

Hashimoto's thyroiditis

First described by Hakura Hashimoto in 1912, Hashimoto's thyroiditis is usually asymptomatic and requires no treatment.[9] As with other forms of thyroiditis, inflammation may lead to a temporary thyrotoxicosis, but euthyroidism returns in the majority, with only 20% developing hypothyroidism. Hashimoto's disease is usually painless, but compression symptoms may occur, often disproportionate to the size of the goitre. Clinical examination reveals a non-tender, firm goitre. Diffuse lymphocytic infiltration, atrophic follicles, germinal centres and fibrosis characterise the histopathology. Thyroid hormone replacement is required when hypothyroidism develops. Surgery may be indicated for compression symptoms and when there is suspicion of co-existing malignancy.

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Amiodarone-Induced Thyrotoxicosis

胺碘酮导致高甲状腺激素血症

Clinical Course and Predictors of Outcome

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Peter Ammann, MD,* Beat Schaer, MD,* Christian Sticherling, MD,* Beat Muller, MD,†
Stefan Osswald, MD, FACC, FESC*

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Results Eighty-four patients were included in the present analysis; 27 patients received prednisone for AIT. There was no difference in time to normalization of free thyroxine between those receiving and those not receiving prednisone. Long-term follow-up showed high morbidity and mortality; 47 patients (56%) reached the primary end point. Patients receiving prednisone had a worse outcome than those not receiving prednisone ($p = 0.003$). Although patients received prednisone for 84 ± 65 days, curves started to separate only 12 months after the initial diagnosis.

Conclusions Patients with AIT have a high event rate during follow-up. Prednisone had no effect on time to normalization of thyroxine levels and was associated with an increased event rate. Importantly, AIT-related problems must be expected late, at a time when thyroid function is under control. (J Am Coll Cardiol 2007;49:2350–5) © 2007 by the American College of Cardiology Foundation

Amiodarone-associated thyrotoxicosis

Amiodarone is an iodine-containing drug (37% by molecular weight), whose use frequently results in abnormal tests of thyroid function in euthyroid patients, specifically a slight rise in free T4, a slight fall in free T3, and a transient increase in TSH, largely because of effects on peripheral deiodination of T4 to T3.[132] Although these changes are common they do not show thyroid dysfunction. However, amiodarone-associated thyrotoxicosis occurs in 6–10% of people given the drug and is more common in iodine-deficient areas.[133] This complication is serious and is associated with more than doubling of adverse cardiac events[134] and worse outcomes than in Graves' or toxic nodular hyperthyroidism, especially in patients with left ventricular dysfunction.[135]

For a diagnosis of amiodarone-associated thyrotoxicosis free T4 and free T3 must be increased and TSH must be suppressed. Two types are described: type 1 is iodine-induced thyrotoxicosis and occurs in people predisposed to thyroid autoimmunity or with pre-existing thyroid nodules; type 2 is destructive thyroiditis due to toxic effects of amiodarone on thyroid cells. Various ways to distinguish the types have been suggested, although distinction is often not possible because patients have features of both. The most specific test is colour flow doppler ultrasonography which detects increased vascularity in type 1, a finding absent in type 2.[136]

Type 1 amiodarone-associated thyrotoxicosis is best treated with carbimazole or methimazole. Doses of at least 40 mg daily are often required. Addition of potassium perchlorate can be helpful,[137] but this drug is not widely available. Type 2 is best treated with prednisolone (40 mg per day), which typically leads to rapid resolution,[136,137] although use of prednisolone can worsen outcomes from cardiovascular events.[138] When a clear distinction between types 1 and 2 is not possible, a combination of thionamides and glucocorticoids should be given until biochemical improvement occurs, at which point drugs can be gradually withdrawn. Discontinuation of amiodarone might be contraindicated because of serious underlying heart disease and requires joint decisions between the endocrinologist and cardiologist. Because of the very long drug half-life (100 days), discontinuation of amiodarone is largely ineffective.[133] Furthermore, control or resolution of thyrotoxicosis is achievable while amiodarone is being given.[139] Iodine uptake is low in both types of thyrotoxicosis because of the iodine load in the drug, so radioiodine treatment is unfeasible for at least for 6–12 months after amiodarone withdrawal. Thyroidectomy has been used in patients resistant to other therapies, but is associated with risk of morbidity and mortality.[140] Thyroidectomy under local anaesthesia has also been used.[141]

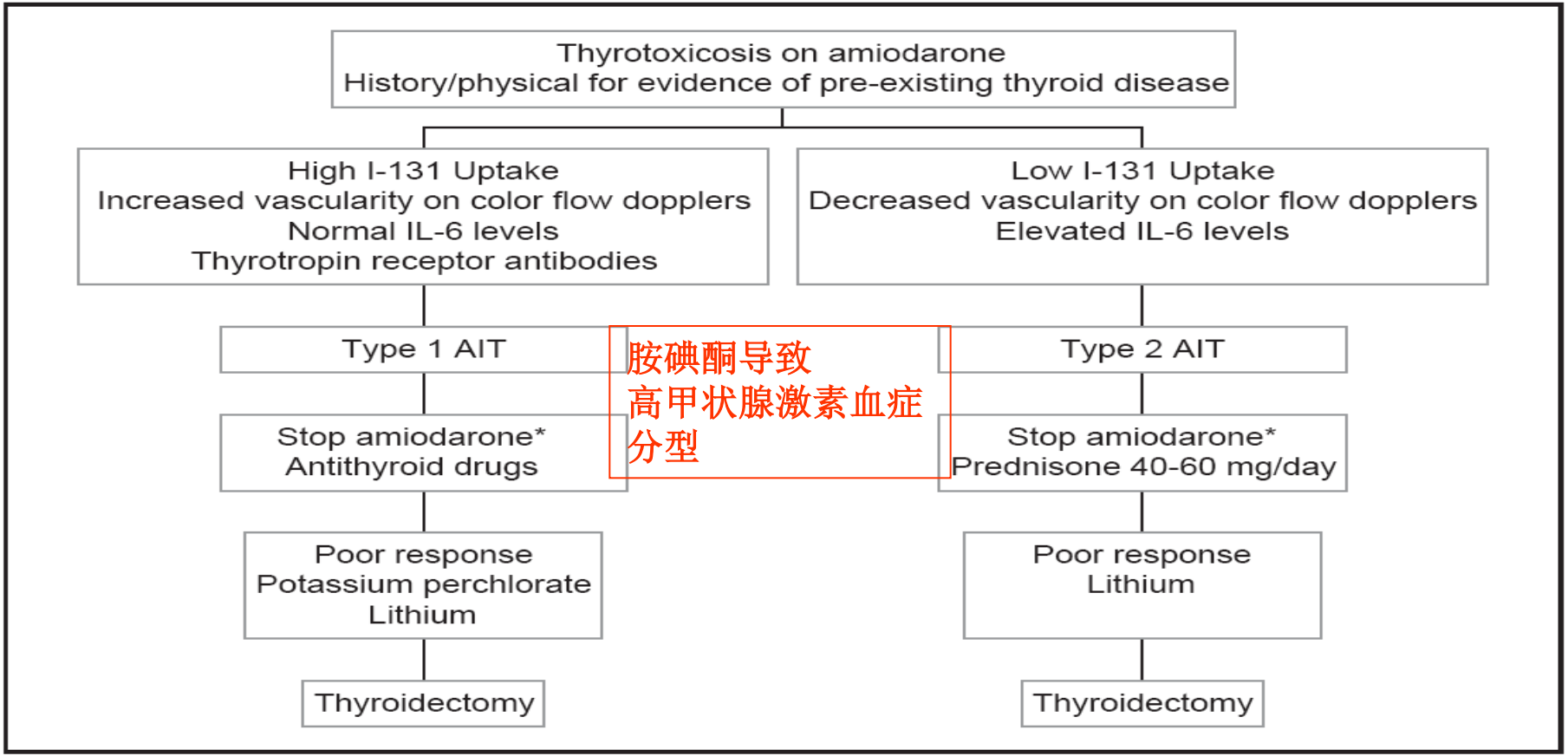


Figure 1) *Management of amiodarone-induced thyrotoxicosis (AIT). *If possible. I-131 Iodine-131; IL-6 Interleukin 6*

亚急性甲状腺炎的治疗

- 对轻型病例采用阿司匹林或其他止痛药。
- 非类固醇抗炎药，或应用糖皮质类固醇激素，如泼尼松，如病人服用泼尼松24-48h无反应，亚急性甲状腺炎的诊断应再评定。
- 皮质激素并不会影响本病的自然过程，如果皮质激素用后撤减药量过多、过快，反而会使病情加重。
- 伴有甲状腺功能亢进时一般不采用抗甲状腺药物治疗，通常采用非特异的药物，如口服β-受体阻滞剂。因本病伴高甲状腺激素血症是暂时的，且甲状腺摄碘率低不是放射碘治疗的指征。本病的甲减期也常是暂时的，所以不需甲状腺激素替代治疗，此时TSH分泌增加有助于甲状腺功能的恢复。除非病人甲减症状非常明显，甲状腺激素替代治疗应当禁忌。
- 约有10%的患者可发生永久性甲状腺功能减低。

亚急性甲状腺炎与室性心律失常

- 亚甲炎早期由于甲状腺滤泡被破坏，储存在内的T_4和T_3大量短时释放入血，导致高甲状腺激素血症。
- 血中过高的甲状腺激素水平，引起甲状腺毒症表现，可能出现心律失常（如频发室性早搏）。
- 治疗原则在治疗亚甲炎的同时，对症处理心脏情况。
- 随着亚甲炎好转后，心律失常可以消失。

典型病例

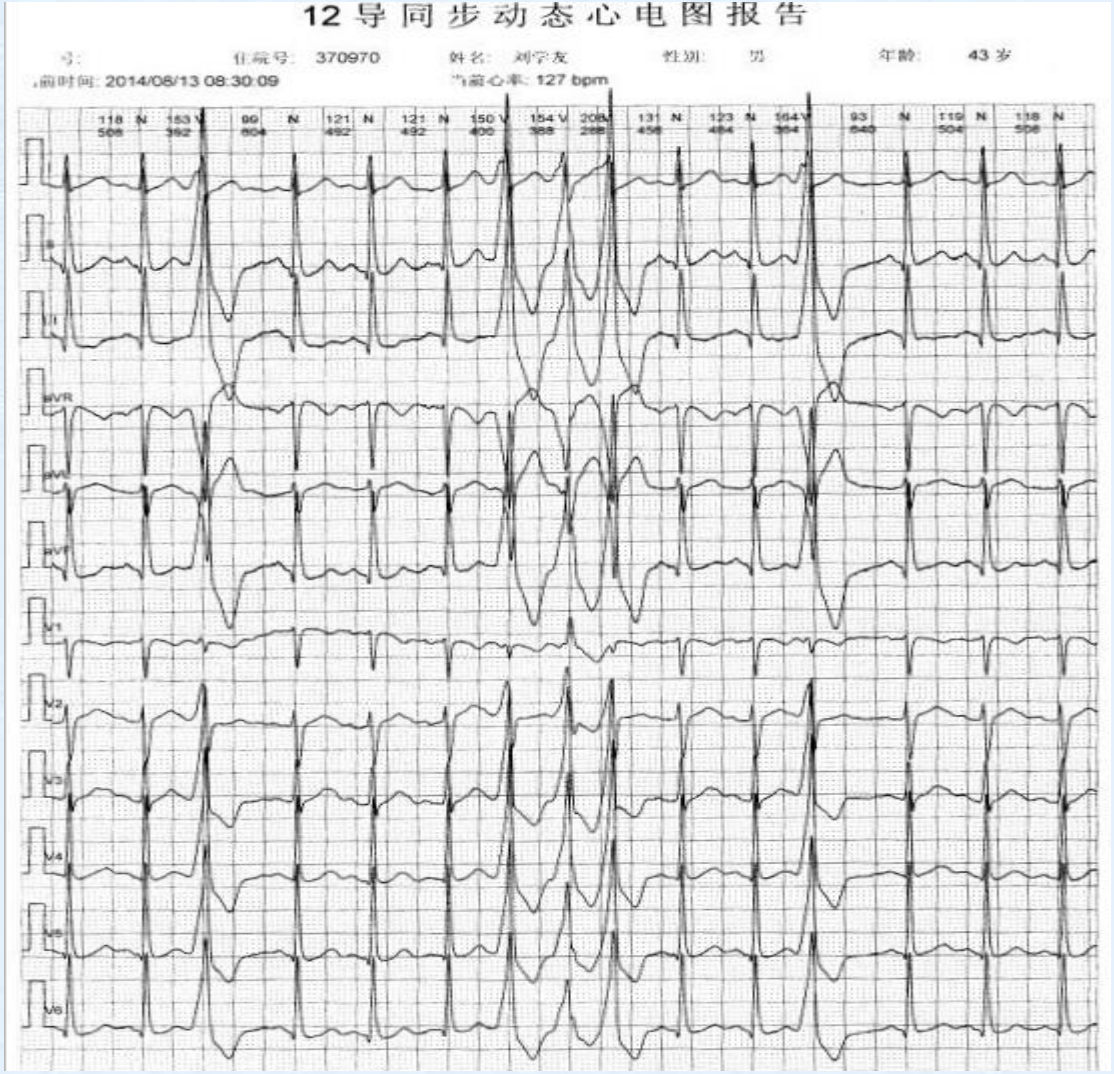
- 男性，43岁
- 主诉：突发心悸1天
- 患者无明显诱因下出现心悸，无胸闷胸痛，无呼吸困难，偶有头晕
- 心电图：窦性心律，频发室早
- 高血压病史3年，最高血压为170/100mmHg，未规律服用降压药
- 查体：心率100/分，律不齐，可闻及早搏；血压120/80mmHg，T 36.7 ℃，甲状腺I°大，无压痛；双手无震颤

动态心电图

- 窦性心律 ，心率100/分，心律不齐
- 房性期前收缩20次/24小时
- 持续性室性期前收缩22005次/24小时
- 部分成对出现，部分二联律，三联律
- 短阵室速7次/24小时
- ST-T改变

超声心动图

- LVS 7.75mm，左室舒张末内径 57.9mm，左房内径36.6mm，
- LVEF 70%
- 提示：左心增大，左室舒张功能下降



实验室检查

甲功：

T3 >12.32 nmol/l ↑ (0.92-2.79)

T4 297.40 nmol/l ↑ (58.1-140.6)

TSH 0.03 uIU/ml ↓ (0.35-5.50)

TPOAb 47.6 (0-60)

TRAb <0.300 (<1.75)

实验室检查

- 肌酸激酶 20 U/L，　肌酸激酶同功酶 2.7 U/L
- 肌钙蛋白I 0.001 ng/ml
- 钾3.8mmol/L，钠140.3 mmol/L，
- B型纳尿肽 5.2 pg/ml

甲状腺显像

甲状腺摄取功能明显减低

初步诊断

1. 亚急性甲状腺炎

   甲状腺毒症期

2. 心律失常

   频发室性期前收缩

   短阵室速

   窦性心动过速

3. 高血压病2级（很高危）

治疗

- 纠正心律失常、控制心室率
- 美托洛尔缓释片47.5mg，Qd
- 甲泼尼龙12mg，Qd口服

随访

- 1个月症状明显缓解，复查心电图无室性早搏
- 3个月甲状腺功能完全恢复正常，逐渐减量并停用美托洛尔，复查24小时动态心电图心律恢复正常

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