

SUBACUTE THYROIDITIS: DIAGNOSTIC DIFFICULTIES AND SIMPLE TREATMENT

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Subacute thyroiditis (SAT) constitutes 0.8% of referrals to Northland Thyroid Laboratory and is one tenth as common as hyperthyroidism. The peak age is 30–50 years, and women predominated by a factor of 4.5. Two thirds of the patients presented in typical fashion with a painful tender goiter, but for one third the presentation was atypical in that there was no pain, the principal complaint in most cases being painless goiter, thyroid nodule, or features of hyperthyroidism. Elevated erythrocyte sedimentation rates were found in all typical patients and in 11 of 14 atypical patients. Serum thyroxine iodide values were elevated in two thirds of the patients, typical and atypical. The radioactive iodine uptake was subnormal for all and responded subnormally to thyroid-stimulating hormone for 24 of 27 patients. Diagnosis of SAT is further supported by spontaneous resolution of complaints, goiter (nodule), and abnormal laboratory values. Failure to consider the possibility of SAT in the absence of neck pain may lead to erroneous diagnosis and improper therapy. Treatment with simple analgesics is satisfactory for most SAT patients. Evidence for permanent functional impairment was demonstrated for four patients.

Subacute thyroiditis (SAT) presumably results from viral infection of the thyroid gland, causing an acute exudative process which gradually regresses, eventuating generally in complete recovery. The hallmark of the acute phase of SAT is a painful, tender goiter, the characteristic local manifestations of the inflammatory process (1–3). Accompanying systemic manifestations of inflammation include low-grade fever, sweats, and malaise. Discharge of stored thyroid hormone by this inflammatory process may

produce the systemic manifestations of hyperthyroidism, usually mild.

The laboratory abnormalities are readily understandable in terms of the underlying pathophysiology. As a consequence of the diffuse inflammation, the thyroidal uptake of radioactive iodine (RAI) is impaired (1–9). The discharge of thyroid hormone elevates serum thyroxine values (ST₄) (1–9). The erythrocyte sedimentation rate (ESR) is elevated as a nonspecific response to inflammation (1–9), and occasionally there is an increase in the white blood cell count (WBC) as well (3,6–9).

As the disease progresses the local and systemic features decline until full recovery is realized, usually within 2–4 months. In some instances there is a temporary hypothyroid phase (2,3) which rarely may prove permanent (10). Lesser degrees of functional impairment may be less rare.

The possibilities for different presentations of SAT can be anticipated if one considers the implications of the protracted course of the illness, as well as variations in severity and extent of involvement, patient perceptivity, and the time lapse between onset of illness and consultation with the physician. When SAT presents without many of its cardinal features there may be diagnostic difficulties. Although failure to diagnose an acute, self-limited disorder may not be serious per se, the potential for incorrect alternative diagnoses leading to unnecessary or improper therapy, is an important concern. Therefore, a principal objective of this paper is an analysis of experience with SAT, emphasizing the recognition of the atypical presentation. Three particularly instructive patients will be reported in brief.

A secondary objective is to offer support for the

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recommendation of simple analgesics and reassurance as the treatment of choice for most patients with SAT (1-3,11-13).

METHODS

A review of the records of 62 SAT patients referred to the Northland Thyroid Laboratory constitutes the basis for this report. Historical data and physical findings were recorded on all patients. White blood counts (WBC) and erythrocyte sedimentation rates (ESR) were frequently performed. The serum thyroxine iodide concentration (ST_1) was determined by the competitive protein-binding method (Abbott Laboratory kit), normal range (Northland Thyroid Laboratory) 3.0-6.5 $\mu\text{g}\%$, expressed as T_4 iodide. For patients receiving estrogens or other medications which might alter binding globulin concentrations, a free thyroxine index was calculated using values for the ST_1 determination and the triiodothyronine resin uptake (Abbott Laboratory kit) as previously described (14).

A 24-hr radioactive iodine uptake value (RAI) was obtained for all patients. Standard methodology was used with the technical controls outlined in detail elsewhere (14). TSH testing was done after a preliminary RAI by administering 5 units intramuscularly (Thytropar, Armour Laboratories), a tracer capsule 8-10 hr later, and counting 12-15 hr afterwards. A normal response in our laboratory is an increment of at least 10% over the baseline value (confirmed by a study of 91 patients either taking thyroid hormone without evidence of thyroid disease or euthyroid with marginally low baseline RAI values).

In the case reports ST_1 values in some instances were performed elsewhere. If the normal ranges vary from those cited above, they will be given.

RESULTS

SAT is an uncommon disease. During a 2-year period between 1970 and 1972, 5,254 patients with thyroid disease were seen at the Northland Thyroid Laboratory. Forty-two had SAT, for an incidence of 0.8%. During the same period there were 428 patients with hyperthyroidism for a ratio of 10 to 1. Woolner reported 8 patients with Graves' disease for every one with SAT (15).

There is general agreement that most patients with SAT are between 30 and 50 years of age (5,6,11), with women outnumbering men by 5 to 1 (5,16). Table 1 indicates that our data are consistent with these earlier observations.

Table 2 reveals the relative frequency of the more prominent complaints elicited by history from SAT patients. Nervousness, fatigue, and certain other

TABLE 1. AGE AND SEX OF 62 PATIENTS WITH SUBACUTE THYROIDITIS

Age (years)	Men	Women	Total
20	0	4	4
20-29	0	8	8
30-39	4	18	22
40-49	3	9	12
50-59	4	7	11
60-69	0	5	5
Total	11	51	62

TABLE 2. COMPLAINTS REPORTED BY 62 SAT PATIENTS

Neck pain	40
Ear or jaw radiation	13
Migrating from one side to the other	8
Goiter or neck swelling	30
Weight loss	22
Palpitation	20
Heat intolerance	19
Malaise	18
Fever or night sweats	17
Tremor	11
Scanty menses	7
Loose bowels	7
Insomnia	3

symptoms which were commonly reported were not included for they are just as common with other thyroid disorders, and indeed in patients without thyroid disease. Symptoms other than local pain and swelling result from the systemic responses to inflammation or the discharge of thyroid hormone. Distinction between these two processes was often difficult. For example, tachycardia, palpitation, sensation of warmth, sweating, and insomnia could result from either or both processes. For most patients the systemic manifestations of SAT were mild, and were overshadowed by the local findings. More than one third of the patients were unaware of any thyroid abnormality until it was called to their attention, including eight patients who acknowledged tenderness for the first time when the thyroid was palpated by the author.

Table 3 lists the predominant considerations which led to thyroid consultation. The majority of patients were referred for either neck pain, goiter, or both. The firm-to-hard nature of thyroid tissue involved with SAT raised the index of suspicion for cancer when apparently solitary nodules were found. Laboratory data frequently proved confusing to the family physician, particularly the apparent discrepancy of an elevated ST_1 with a subnormal RAI, even though local findings were the dominant consideration in the referral. The patient with hair loss had an

TABLE 3. PRINCIPAL FINDING PRECIPITATING REQUEST FOR THYROID CONSULTATION

Neck pain	40
Painless goiter	12
Thyroid nodule	4
Suspected hyperthyroidism	3
Hair loss, unexpected high ST _i value	1
Confusion with laboratory data	1
Atrial fibrillation with goiter	1

TABLE 4. PHYSICAL FINDINGS ON EXAMINATION OF THE THYROID

Firm-to-hard consistency	56
Tenderness	48
Diffuse enlargement	34
Unilateral enlargement	15
Solitary nodule	5
Multinodular	4
No thyroid enlargement appreciated	4

ST_i, ordered by her physician to rule out hypothyroidism. When a high value was reported, consultation was requested.

Table 4 summarizes the findings on palpation of the thyroid. A firm-to-hard diffuse goiter was highly characteristic although at times involvement was limited to one lobe or even a solitary nodule.

Forty of the 62 SAT patients were considered typical in that the presenting complaint was neck pain and an enlarged, tender thyroid gland. The remaining 22 patients were considered atypical since pain was not spontaneously appreciated, although for eight patients some tenderness was acknowledged when the thyroid was subjected to palpation. For some of these atypical patients the diagnosis was made only retrospectively after further events clarified a confusing picture.

Table 5 summarizes the clinical and laboratory findings for typical and atypical patients. A history of systemic complaints (e.g., malaise, fever, sweats, palpitation, weight loss, tremor, loose bowels) was

obtained in more than three fourths of the typical patients but less than half of the atypical group. For both groups evidence of systemic involvement on physical examination (e.g., tachycardia, tremor, elevated temperature, hyperreflexia, excessive perspiration) was less common than by history, suggesting that by the time the patients were seen in consultation the disease was already subsiding in some instances.

An elevated white blood count was found in only 13 of 32 patients studied. However, the ESR was over 30 mm/hr in 38 of 41 patients and was 23, 26, and 28 mm/hr in the remaining 3. An elevated ESR was an important finding for atypical patients. Elevated ST_i values were found in 37 of 58 patients. For many, the magnitude of the elevation was rather modest; hence the discrepancy between the frequency of elevated ST_i values and the relative infrequency of physical findings of hyperthyroidism was more apparent than real.

RAI values were subnormal for all patients whether typical or not. Only two patients had values over 5%, one with 6% and one with 8%. A subnormal response to TSH (an increment of less than 10%) provided further confirmation of impaired thyroid function. TSH tests were done on 27 patients. The observed increments ranged from 0 to 16%, with an average of 5.2%. For only three patients were increments greater than 10% found (11, 13, and 16%). When the RAI was adequate for scanning, even though the response to TSH was subnormal, the pattern in most cases was one of patchy and irregular bilateral function, demonstrating foci of relatively less involved tissue in the diffusely inflamed gland. For six patients with significant responses to TSH, scanning also provided evidence of impaired thyroid function since uptake was asymmetrical and confined to uninvolved tissue. Figure 1 shows the scans for two such patients.

An attempt was made to correlate the duration of the disease before evaluation, as estimated by his-

TABLE 5. CORRELATION HISTORY, PHYSICAL FINDINGS, LABORATORY DATA ON TYPICAL AND ATYPICAL SAT PATIENTS

Type/ number	History of systemic complaints	Physical findings of systemic involvement	Type of thyroid enlargement				Laboratory findings			
			Dif.	Unilat.	Nod.	None	WBC > 10,000	ESR* > 30 mm/hr	ST _i * > 6.5 µg%	RAI < 9%
Typ/40	34	15	22	14	4	0	9/23	28/28	21/37	40
Atyp/22	9	4	12	1	5	0	3/9	11/14	16/21	22
Total/62	43	19	34	15	9	4	12/32	39/42	37/58	62

* For fractional expressions the numerator indicates the number of abnormal values, the denominator the number of patients tested.

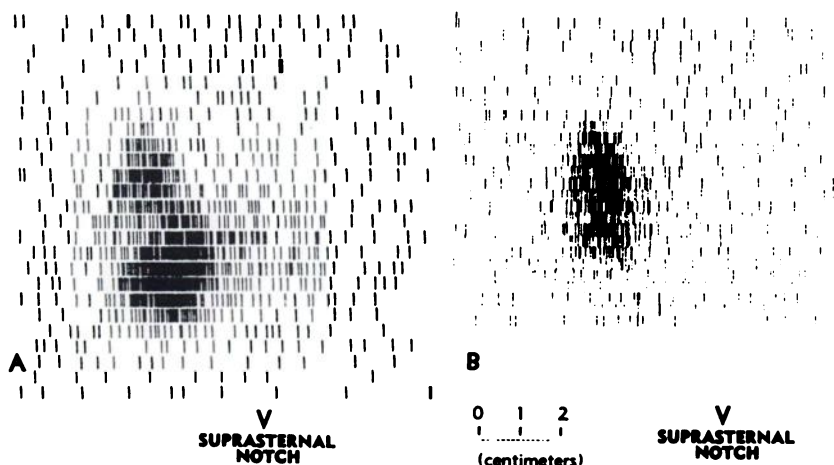


FIG. 1. Examples of grossly asymmetrical response in thyroidal ¹³¹I uptake following TSH in subacute thyroiditis.

tory and the observed clinical and laboratory features. The subacute nature of the disease and the retrospective nature of the study complicated this analysis. Nevertheless, for 24 typical patients the data seemed adequate for analysis. Since the severity and extent of involvement, as well as patient perceptivity are at least equally important determinants of the history and physical findings elicited, it is not surprising that Table 6 suggests that duration before consultation did not seem to have much influence upon the findings.

For the atypical patients it was much more difficult to obtain data on the duration of the disease since the presenting thyroid features were often unappreciated by the patient until examination was performed for some unrelated purpose.

Three instructive atypical patients will be presented briefly.

Case 1. A 37-year-old man complained of sweating, hand tremor, and a 5-lb weight loss. No thyroid enlargement or tenderness was noted. The RAI was 1.5%, and the ST₄ 11.2 μg% (NR 4.5–8.5 μg%). A diagnosis of hyperthyroidism was made. An “unknown chemical block” was invoked to explain the low RAI value. Treatment with surgery or antithyroid drugs was advised because “obviously ¹³¹I is out of the question in view of the poor uptake”. At this point thyroid consultation was requested.

There was no clinical evidence of hyperthyroidism. The thyroid gland was neither enlarged nor tender. The ST₄ was 3.1 μg% (1 month after the above elevated ST₄ value). The RAI was 5%, and increased only to 9% after TSH. The ESR was 54. A diagnosis of subsiding SAT was made. A repeat evaluation 2 months later revealed normal ST₄, RAI, and ESR values.

Case 2. A 62-year-old woman noted over a 3–4 day period the development of a large mass in the left side of the neck. There was neither pain nor tenderness, but an intermittent low-grade fever up to 100.5°F had been detected. There was also mild malaise. The RAI was 1%. A diagnosis of thyroid cancer was suspected but thyroid consultation was sought before surgery.

There was a 4-cm, firm, nontender mass in the left lobe of the thyroid gland. The ST₄ was 8.9 μg%, the ESR 51. An RAI after TSH was 8%. The scan (A of Fig. 2) revealed all of the tracer within the right lobe. A diagnosis of SAT was made and treatment with prednisone, 60 mg daily, was instituted. Within 1 week the mass completely disappeared. Prednisone was withdrawn over the next 6 weeks. One month later the ST₄ was 4.5 μg%, the ESR 15, and the RAI 14%. The scan (B of Fig. 2) revealed recovery of function in the left lobe.

Case 3. A 38-year-old physician was hospitalized

TABLE 6. CORRELATION SYMPTOMS, PHYSICAL FINDINGS, LABORATORY DATA WITH DURATION OF ILLNESS, 24 TYPICAL SAT PATIENTS

No. of patients	Duration illness	Systemic symptoms	Physical findings of systemic involvement	ESR > 30 mm/hr	ST ₄ > 6.5 μg%
5	2 weeks	4	3	5	4
14	2–4 weeks	11	9	14	6
5	4 weeks	3	3	5	3
Total 24		18	15	24	13

with an acute episode of atrial fibrillation. The working diagnosis was myocardial infarction. In the past year he had undertaken a vigorous exercise program including bicycling and running. He drank 10–20 cups of coffee daily. The thyroid gland was minimally enlarged and very slightly tender. He had been unaware of neck pain. Conversion to sinus rhythm was achieved with bed rest, digitalis, and quinidine. Studies for myocardial infarction were negative. Thyroid consultation was requested.

The ST_4 was $10.8 \mu\text{g}\%$, and the ESR 44. The RAI was 1%, unresponsive to TSH. A diagnosis of SAT was made. The patient was advised to avoid exercises and coffee but otherwise to resume normal activity.

During 3 months observation there were no recurrences of atrial fibrillation but the thyroid doubled in size. The patient noticed the onset of muscle cramps, paresthesia, and cold intolerance. The RAI was 10%, the ST_4 $0.6 \mu\text{g}\%$, and the serum TSH by radioimmunoassay was $172 \mu\text{U}/\text{ml}$ (greatly elevated). The antithyroglobulin titer was less than 1–16 (not elevated). He was given levothyroxine 0.2 mg daily for 4 months. Later, after levothyroxine had been withdrawn for 6 weeks, the thyroid gland was normal in size and thyroid function tests were also normal.

These case reports exemplify the errors which may result from failure to consider the possibility of SAT in the absence of neck pain. Therefore, the clinical features which should suggest atypical SAT and the procedures useful in confirming a presumptive diagnosis will be summarized.

Painless, diffuse, or local thyroid enlargement is the most common presenting complaint. Clinical features of hyperthyroidism with an unexplained subnormal RAI value, or the query by a confused physician who cannot explain a high ST_4 value in conjunction with a low RAI, are additional situations which should suggest the possibility of atypical SAT (assuming, of course, that a searching inquiry for interfering medications was unrewarding).

Further laboratory support for the diagnosis is provided by an elevated ESR and a subnormal response to TSH. Scan evidence of patchy and irregular or asymmetrical function following TSH stimulation is additional valuable data.

An important diagnostic feature is the complete spontaneous resolution of symptoms, thyroid enlargement, and abnormal laboratory data within 2–4 months.

For suspicious nodules a prompt and complete resolution induced by a short course of prednisone, and sustained afterwards, is of considerable diagnostic value.

Table 7 records the principal clinical and laboratory features of 22 atypical patients.

Some of these patients require special comment. For Patients 1, 2, 9, and 10 the laboratory data are minimal. These patients were seen before we were more alert to the problem of atypical SAT. The spontaneous disappearance of goiter or nodules and return of thyroid function to normal provided strong support for a retrospective diagnosis.

The findings for Patient 4, including a small goiter, high ESR, normal ST_4 , and suppressed RAI which was not responsive to TSH are compatible with Hashimoto's disease (which might also have an elevated ESR). However, recheck 5 months later revealed further deterioration of thyroid function while the ESR had fallen to normal. This course is more consistent with the progression of SAT. We hope to have further followup indicating complete recovery.

Patient 18 had neither an ESR determination nor followup data, but the high ST_4 in conjunction with a suppressed RAI which was unresponsive to TSH make at least a presumptive diagnosis of SAT.

Treatment for the entire series of patients included principally reassurance and simple analgesics, usually aspirin, occasionally supplemented with codeine for a few days. None of the patients received adrenal corticosteroid therapy other than for diagnosis (three patients presenting with discrete nodules). The value of reassurance deserves emphasis. Most patients with pain had already tried aspirin-containing medications, but without relief. However, after having been assured that their disease was not serious, these patients reported that the pain was either relieved by simple analgesics or not so severe as to cause either disability or concern. Analgesics were

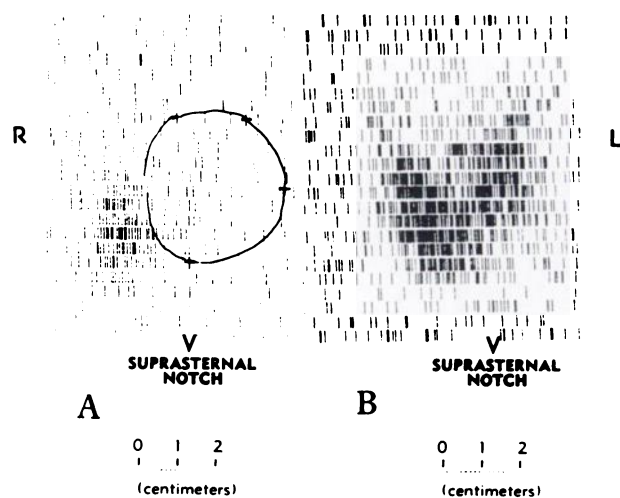


FIG. 2. A. Initial scan after TSH reveals uptake of ^{131}I only in right lobe, while left lobe mass is functionless. B. Recovery of bilateral function.

continued until the pain subsided and then discontinued on the patients' initiative. Relapses were not observed in this series although mild relapses may not have been reported.

Thirty-four patients were re-evaluated 2-3 months or longer after the acute episode. Twenty six had completely recovered, although two had transient hypothyroidism. One patient considered to have recovered had persistent goiter at 3 months. However, it was known that she had goiter before the development of SAT.

Of the eight patients who failed to recover, two had goiter and hypothyroidism, respectively, at 3 and 5 months after the acute episode. These patients may yet experience a complete resolution of the disease. Two additional patients had persistent nodules, one of which proved to have autonomous function and was probably pre-existing.

Four patients had evidence of impairment of function more than 1 year after the acute episode. One of these patients was hypothyroid at 3 months and at 15 months still had an ST_4 value of $1.2 \mu\text{g}\%$ and an RAI of 1% unresponsive to TSH. The second patient was normal at 3 months but at 12 months the ST_4 values were 3.0 and $3.5 \mu\text{g}\%$ on two determinations, and the serum TSH concentration by radioimmunoassay on three determinations was 21, 36, and $42 \mu\text{U}/\text{ml}$ (pooled euthyroid serum control $10.5 \mu\text{U}/\text{ml}$). A third patient on birth control medication had free thyroxine indices of 1.2 and 1.0 (NR 0.8-2.1) 13 months after the acute episode. The RAI was 18% and unresponsive to TSH. The scan revealed a normal appearing left lobe but a very small right lobe remnant measuring only $1 \times 1.5 \text{ cm}$. The serum TSH concentration by radioimmunoassay was 21 and $27 \mu\text{U}/\text{ml}$ on two determinations

TABLE 7. CLINICAL AND LABORATORY FEATURES, 22 PATIENTS WITH ATYPICAL SUBACUTE THYROIDITIS

No.	Age, Sex	Presenting complaint	Thyroid gland size*	ESR (mm/hr)	ST_4 ($\mu\text{g}\%$)	RAI % \bar{a} & \bar{p} TSH†	Thyroid scan	Followup
1	14F	Goiter	30 gm T		7.3	1		Recovery
2	31F	Goiter	30 gm T		12.7	1		Recovery
3	21F	Goiter	60 gm T	35	13.0	1		Goiter & hypo, 16 mo.
4	54F	Goiter	40 gm T	37	4.6	1 2		Hypo at 5 mo. ESR 17. No further study.
5	38F	Goiter	30 gm T	46	6.8	1		Recovery
6	38M	Atr. Fib.	30 gm T	44	9.4	1 1		Transient hypo then recovery
7	36F	Unexplained High ST_4	30 gm T	28	4.7	2 9	Patchy, bilat. function	Recovery
8	21F	Hair Loss High ST_4	20 gm T		8.3	1 3		Recovery
9	38M	Nodule	2.5 cm			3		Recovery
10	17F	Nodule	3.0 cm		7.0	1		Recovery
11	29F	Nodule	1.5 cm		6.1	2 9	Nod. cold, patchy funct. elsewhere	Recovery
12	62F	Nodule	4.0 cm		9.8	1 5	Rt. lobe funct. only	Recovery
13	20F	Goiter	30 gm	23	6.7	2 10	Patchy, bilat. function	Recovery
14	16M	Goiter	40 gm	26	7.2	1 4		Recovery
15	23F	Goiter	40 gm	40	6.4	1 2		None
16	35F	Goiter	40 gm	48	8.1	1 5	Midline patchy function	Recovery
17	23F	Goiter	30 gm	36	6.1	2 3		None
18	45F	Goiter	30 gm		8.6	1 1		None
19	37F	Goiter High ST_4	40 gm		7.4	1 1		High serum TSH, poor funct. Rt. lobe, 13 mo.
20	37M	Possible Hyper.	20 gm	54	11.2	2 9	Patchy, bilat. function	Recovery
21	49M	Possible Hyper.	20 gm	45	8.2	2 5		Recovery
22	21M	Possible Hyper.	20 gm	32	7.2	1 8	Patchy, bilat. function	None

* Size is estimated in grams with 20 gm considered normal. "T", indicates tenderness. For discrete nodules the average diameter is given.

† \bar{a} = before, \bar{p} = after.

(pooled euthyroid serum control 10.5 $\mu\text{U}/\text{ml}$). The fourth patient returned for the first time 11 years after a typical episode of SAT. She was euthyroid but had a diffuse goiter twice normal size with the right lobe larger than the left.

DISCUSSION

There are numerous synonyms for subacute thyroiditis (SAT). The eponym deQuervain refers to the pathologist who described the characteristic histologic features, features which have given rise to names favored in the pathology literature, including tubercular, pseudotubercular, giant cell, and granulomatous thyroiditis. Viral or mumps thyroiditis are names reflecting the presumptive etiology. The mumps virus was first to be associated with SAT. More recently a number of other viruses have been implicated (17). A designation which relates to the clinical picture has been preferred by most physicians. Acute thyroiditis is favored by some but can lead to confusion with suppurative thyroiditis which is also known as acute. Acute nonsuppurative thyroiditis avoids this pitfall but seems needlessly cumbersome. Furthermore, although the more overt presentation of SAT may justify the adjective acute, there is growing appreciation that for most patients the designation subacute is appropriate.

There is little difficulty in recognizing SAT in the classical presentation. A history of neck pain radiating to the jaw or ear often migrating from one side to the other is characteristic. Also common are malaise, low-grade fever, sweats, and mild manifestations of hyperthyroidism. This history in conjunction with a tender somewhat swollen thyroid gland makes the diagnosis almost certain.

It should be noted that the thyroid gland usually is not enlarged beyond one and one half to twice normal size (5,11,18). This enlargement generally is diffuse but may be asymmetrical, unilateral, or even present as a solitary nodule as in our Case 2 (19-22).

The most consistent laboratory abnormality is an elevated ESR, even to 100 mm/hr (2,3,8,11). Of course, this is not a specific finding and may be seen with Hashimoto's disease as well (23). The elevated PBI results from an inflammatory discharge of both thyroxine and other iodinated proteins. Hence there is a greater elevation in PBI than BEI or ST_4 (1,3). The greater simplicity and reliability of the ST_4 determination has led to discontinuation of the PBI in many laboratories. However, when the ST_4 is used for diagnosis of SAT, a lesser value will be obtained than would have been the case with the PBI. An elevated white blood count (WBC) has been observed by some (3,6-9), and denied by others

(5,16). Since most SAT patients do not have an elevated WBC, the study is of little diagnostic value; however, awareness of this occasional finding is important, if only to avoid needless search for another explanation.

The RAI is usually suppressed. If the disease is localized to a small area, a normal value may be obtained (9,20,22). The suppressed RAI may simply reflect cellular injury if the involvement is diffuse (9,19,24). This is why there is a subnormal response to the relatively small doses of exogenous TSH which readily stimulate normal thyroid tissue (1,24). Prolonged stimulation with larger TSH doses may produce greater RAI increments but still considerably less than that observed in normals (9). Even though SAT has spared a substantial portion of the thyroid the RAI may still be suppressed because the inflammatory process not only impairs cellular function in the involved area but also discharges thyroid hormone, elevating serum concentrations and thus suppressing pituitary TSH release (19,25). For these patients exogenous TSH is more likely to produce a significant increment in the RAI, and scanning will show the location of the uninvolved or less involved tissue (Figs. 1 and 2).

SAT usually progresses from a more or less acute phase to recovery over a period of 2-4 months. Occasionally the duration may be prolonged, even to 1 year or more, and punctuated with relapses of acute manifestations. A transient period of hypothyroidism may occur in as many as one in four patients (2,3). This may be unappreciated if the patients are not seen frequently.

This classical picture of SAT may actually be the exception rather than the rule (18). The clinical and laboratory features which the physician observes will depend upon the rapidity of evolution of the disease, the severity and extent of involvement, patient perceptivity, and the time lapse before the patient seeks medical advice. Since consulting physicians generally see patients later in the course of the illness, often after prior studies have provided data confusing to the attending physician, the screening process may favor the consultant with an experience consisting of a greater proportion of atypical and less acute SAT patients. For example, Cassidy observed the absence of tenderness in 12% of his patients, and only 20% had the characteristic elevation in PBI with a depressed RAI (18). Similarly, for 22 of our 62 patients pain was not the presenting complaint. Although the RAI was subnormal in all patients, of 58 patients for whom ST_4 tests were performed, 21 had values which were not elevated.

Most helpful in the recognition of the atypical SAT patient is the unexplained finding of an ele-

vated ST_4 with a suppressed RAI in conjunction with an elevation in the ESR. The subnormal or asymmetrical response of the RAI to TSH provides helpful additional information. The spontaneous resolution of the clinical features, including the goiter (nodule) and abnormal laboratory values provides confirmation of the presumptive diagnosis of atypical SAT.

Lack of awareness of the changing clinical and laboratory features of SAT as it progresses through its course can cause the diagnosis of SAT to be overlooked. This is not serious since SAT is a self-limited disorder which almost always results in complete recovery. The potential for incorrect diagnosis is of greater concern. Acute pharyngitis is the most common diagnostic error (3). However, the incorrect diagnoses which may lead to serious therapeutic error include suppurative thyroiditis, hyperthyroidism, and thyroid neoplasm.

Suppurative thyroiditis is exceedingly rare, and usually involves a preexisting goiter. The patient is very ill. There is high fever, an exquisitely tender fluctuant mass with inflamed overlying skin, and enlarged tender adjacent lymph nodes. The WBC is very high, usually over 20,000.

In the absence of appreciable pain and tenderness hyperthyroidism may be considered early in the course of SAT on the basis of evidence of hypermetabolism and an elevated ST_4 value (Case 1). The low RAI should suggest SAT. Thyrotoxicosis factitia is also possible, but here the RAI responds to TSH, and the scan reveals a normal thyroid gland rather than the asymmetrical or patchy uptake seen in those SAT patients who respond to TSH. If a patient with thyrotoxicosis factitia happens also to have primary hypothyroidism (the reason for the initial access to thyroid hormone), there would be a subnormal response to TSH. However, the ESR would not be elevated. Patients with bona fide hyperthyroidism may have suppressed RAI values if they have received iodides (a fact of which neither patient nor physician may be aware). Again the ESR should be helpful. Also, assuming iodide ingestion to cease, the suppressed RAI will rebound in 1 or 2 weeks in hyperthyroidism.

Unnecessary surgery may be advised for patients with SAT (Case 2). In one surgical series approximately 10% of patients with thyroiditis had SAT (26). It has been said that surgery may be unavoidable for some SAT patients (8). Rapidly growing thyroid cancer may be tender (3); hence tenderness is not diagnostic of a benign process. However, if there is an elevated ESR a trial of adrenal corticosteroids may prove definitive (19,20). The goiter of Hashimoto's disease may regress promptly in re-

sponse to this treatment, but in contrast to SAT the condition relapses equally promptly when the medication is withdrawn (27). It is possible that a localized lymphoma might respond partially to corticosteroid treatment; however, these lesions are exceedingly rare and would be unlikely to exhibit the prompt and complete regression characteristic of SAT (Case 2).

The discharge of thyroid hormone which occurs with SAT may adversely affect a marginal cardiovascular system. Therefore, it seems likely that other SAT patients have had experiences similar to that of Case 3. However, the predominance of SAT in relatively young women may explain the lack of emphasis in the literature upon cardiovascular complications (6). In Case 3 it would appear that it was the fortuitous combination of mild hyperthyroidism, strenuous exercise, and excessive coffee which produced the unusual presenting manifestation of atrial fibrillation.

Although adrenal corticosteroids produce prompt relief of the pain and goiter of SAT (2,21), upon withdrawal of this medication relapse may be a problem (6,28). Indeed, the frequency of relapse may be increased by this treatment (23). Steroids may not be suitable for patients with diabetes, peptic ulcer, or a history of tuberculosis. Our experience supports the contention that many SAT patients can be treated successfully with simple analgesics and reassurance (1-3,11-13). We, too, were impressed with relapses in our early experience when we used steroids. In our aspirin-treated patients relapse was not a problem. Patients failing to respond to simple treatment may be given steroids, unless contraindicated, or may be considered for x-ray therapy (16).

The end result of SAT is almost always complete recovery. Persistent goiter may occur (4,6). Although transient hypothyroidism is rather common during recovery (2,3), permanent hypothyroidism is rare (3,5,8-10,17). Three of our patients developed this complication. For two the clinical manifestations were minimal as would have been expected in view of the relatively minor deviations in the laboratory data. In fact it might be more precise to say that these patients have impairment of thyroid function rather than frank hypothyroidism. The availability of TSH radioimmunoassay was of particular value in the confirmation of the functional deficiency. A fourth patient, although euthyroid, had impairment of function as evidenced by a diffuse goiter. It seems possible that patients with mild hypothyroidism or functional impairment might have been overlooked in earlier reports which emphasized complete recovery in all instances.

It has been suggested that the cellular injury pro-

duced by SAT may predispose to the remote onset of hypothyroidism (as has been the case with ^{131}I treated hyperthyroids) (18). One of our patients would seem to exemplify this possibility. It seems prudent to suggest annual evaluation of thyroid function for those who have had SAT.

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