

Painful Subacute Thyroiditis is Commonly Misdiagnosed as Suspicious Thyroid Nodular Disease

Josephine H. Li, MD; Gilbert H. Daniels, MD; and Giuseppe Barbesino, MD

Abstract

Objective: To investigate and characterize the clinical and radiologic features of 10 patients with painful subacute thyroiditis with ultrasound findings considered suspicious for malignancy or for whom biopsy of a suspicious area was recommended by an attending radiologist.

Patients and Methods: Ten patients with painful subacute thyroiditis were seen from June 1, 2016, through January 1, 2019. All 10 patients presented to an endocrine or thyroid clinic with a neck ultrasound report stating findings suspicious for malignancy or nodular disease. Clinical, laboratory, radiographic, and pathologic data were (retrospectively collected and) reviewed.

Results: The mean \pm SD patient age was 49.0 ± 15.0 years at diagnosis; 8 patients were female. All the patients presented with a low or undetectable serum thyrotropin level. Six of 7 patients with available inflammatory markers had elevated levels. Thyrotropin receptor antibodies were absent in all 6 patients tested. On follow-up imaging, 8 patients had complete resolution or improvement of described findings, 1 was lost to follow-up, and 1 had an incidental nodule that was biopsied after the episode of thyroiditis and found to be papillary thyroid carcinoma.

Conclusion: Painful subacute thyroiditis demonstrates specific sonographic patterns that may be misdiagnosed as suspicious thyroid nodular disease. Recognition of the innocent and transient nature of these findings is important for the proper management and monitoring of these patients.

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From the Thyroid Unit, Department of Medicine, Massachusetts General Hospital and Harvard Medical School, Boston, MA.

First described by de Quervain in 1904, subacute granulomatous thyroiditis is a benign, self-limited inflammation of the thyroid gland.¹ It is also known as de Quervain thyroiditis, painful subacute thyroiditis (SAT), and postviral SAT. Painful SAT commonly occurs in epidemic fashion after a viral infection, and in most patients it is thought to be a postviral inflammatory process rather than an actual viral infection in the thyroid gland. Early reports have found a genetic predisposition to painful SAT in those who carry the *HLA-B*35* allele.^{2,3} More recent investigations have uncovered an association with the presence of additional antigens, namely, *HLA-B*18:01*, *DRB1*01*, and *HLA-C*04:01*.⁴

The annual reported age- and sex-adjusted incidence is 4.9 patients per 100,000 population.^{5,6} The distinctive clinical picture includes typical viral symptoms, such as malaise, fever,

and myalgias. Thyroid pain may be moderate to severe and commonly radiates to the jaw or the ear; after abating on one side, it often reappears on the other.⁷ Although severe thyroid tenderness is characteristic of the disease, over the years the pain seems less pronounced, and in some patients, otherwise typical granulomatous SAT may occur with little or no pain.^{8,9} In a recent case series, 4 of 64 patients who met the diagnostic criteria for postviral SAT had no neck or ear pain.¹⁰

Initially, serum thyrotropin concentrations are usually low or suppressed, with elevations in free thyroxine (FT₄) and total triiodothyronine (T₃) concentrations; symptoms of thyrotoxicosis may or may not be present. The erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) level are almost invariably elevated as a hallmark of painful SAT. Traditionally, evidence of autoimmunity is lacking, with anti-thyroid peroxidase

antibodies and thyrotropin receptor antibodies (TRAbs) either absent or transiently present in low titers.^{5,11} However, there have been rare cases of coexisting painful SAT and Graves disease, in which elevated TRAb levels were measured.¹² Interestingly, recent case series have observed an increasing detection of anti-thyroid antibodies.¹⁰ In painful SAT, the 24-hour radioiodine uptake is markedly reduced or nil when performed in the thyrotoxic phase.

The term *subacute* refers to the time course of evolution of the disorder rather than to the severity of the symptoms. As the preformed thyroid hormone is depleted, hyperthyroidism spontaneously resolves over several weeks. Although hypothyroidism may occur, long-term hypothyroidism is the exception rather than the rule.

Subacute granulomatous thyroiditis is a common cause of thyroid pain. Although hemorrhage into a thyroid nodule is a common cause of localized thyroid pain, that diagnosis is almost always evident from the typical thyroid ultrasound appearance. Graves disease, chronic autoimmune thyroiditis (Hashimoto thyroiditis), thyroid malignancy (malignant pseudothyroiditis), or acute infectious thyroiditis are rare causes of pain and thyrotoxicosis.¹³⁻¹⁶

A thyroid ultrasound may contribute additional information when considering the differential diagnosis of thyrotoxicosis. For example, Graves hyperthyroidism is associated with increased blood flow, whereas painless SAT (usually autoimmune) and painful SAT generally have decreased blood flow.^{17,18} Hyperthyroidism due to an autonomous adenoma (“hot nodule”) may be suspected when a nodule is demonstrated on ultrasound; nevertheless, confirmation requires a radionuclide scan.

In our clinical practice, we have observed that many primary care and other physicians reflexively order a thyroid ultrasound to evaluate patients with thyroid enlargement or thyroid pain. Ultrasound descriptions of painful SAT are variable but include diffuse heterogeneity, focal hypoechogenicity, and decreased vascularity,^{19,20} as well as nodular lesions.^{19,21,22} When used by an experienced radiologist or endocrinologist, ultrasound can be useful for diagnostic purposes. However,

many radiologists are unfamiliar with the spectrum of ultrasound findings in painful SAT, and in the hands of a less experienced clinician, ultrasound findings may be described in a way that results in unnecessary procedures or increased anxiety for the patient.

From June 1, 2016, through January 1, 2019, we recorded 10 patients with typical painful SAT with reported neck ultrasound findings “suspicious for malignancy or nodular disease” that were referred for a thyroid biopsy. Although the typical fine-needle aspiration (FNA) finding of a granulomatous infiltrate with multinuclear giant cells assists in confirming the diagnosis of subacute granulomatous thyroiditis,^{6,23} FNA is rarely necessary.⁵ The purpose of this study was to summarize the clinical presentation of these patients with painful SAT and describe the evolution of ultrasound findings that were initially considered suspicious by the attending radiologist.

MATERIALS AND METHODS

The research was conducted ethically in accordance with the World Medical Association Declaration of Helsinki. The study protocol was approved by the Mass General Brigham Human Research Committee before data collection. The study qualified for a waiver of consent due to collection of deidentified data only and because many of the patients no longer actively follow up with an endocrinologist given the self-limited nature of the disease being described. The protocol was approved under these circumstances, and, thus, the individuals included did not need to provide written informed consent. In our clinical practice, we identified 10 patients diagnosed as having painful SAT from June 1, 2016, through January 1, 2019, who were referred to a Mass General Brigham endocrinologist after an attending radiologist recommended an FNA for findings suspicious for malignancy or concerning nodular disease. The diagnosis of painful SAT was based on the presence of a typical presentation with severe and migrating thyroid tenderness as described previously herein, spontaneous resolution of hyperthyroidism, and spontaneous resolution of symptoms over a few weeks or months. We did not review the cases of all patients with painful SAT seen during this period

TABLE 1. Clinical Presentation of 10 Patients With Painful Subacute Thyroiditis^a

Patient No./Sex/Age (y)	Laterality of pain	Thyrotropin level ^b	FT ₄ or T ₃	CRP/ESR	TSI/TBII	TPO Ab	RAI uptake	Symptom duration (mo)
1/F/58	Bilateral	Low	Elevated	Elevated	Negative	Negative	ND	2.73
2/F/60	Right	Low	Normal	ND	ND	Negative	ND	3.63
3/M/54	UNK	Low	Normal	Normal	Negative	Negative	ND	UNK
4/F/43	Right	Low	Elevated	Elevated	Negative	ND	ND	1.61
5/F/57	Right then left	Low	Normal	Elevated	Negative	ND	ND	1.58
6/F/30	Right	Suppressed	Elevated	ND	Negative	Negative	ND	UNK
7/F/76	Bilateral	Low	Elevated	Elevated	Negative	Negative	ND	4.67
8/M/48	Bilateral	Suppressed	Elevated	Elevated	ND	Negative	0.5%	1.84
9/F/35	Right then left	Low	Normal	Elevated	ND	ND	ND	7.42
10/F/29	Unknown	Low	Normal	ND	ND	Negative	ND	UNK

^aCRP, C-reactive protein; ESR, erythrocyte sedimentation rate; FT₄, free thyroxine; ND, not done; RAI, radioiodine; TBII, thyrotropin-binding inhibitory immunoglobulin; TPO Ab, thyroid peroxidase antibody; TSI, thyrotropin-stimulating immunoglobulin; T₃, total triiodothyronine; UNK, unknown.

^bSuppressed thyrotropin level is defined as less than 0.01 μ IU/mL.

in the clinic but confined the review to cases known to us with “suspicious” ultrasound findings.

The medical records of these 10 patients were retrospectively reviewed for the presence and location of neck pain or thyroid tenderness. The length of time between onset of pain and its resolution was calculated based on medical record review of follow-up thyroid or endocrine clinic visits. Thyroid function tests (thyrotropin, FT₄, and T₃), inflammatory markers (ESR/CRP), and antithyroid antibodies (anti-thyroid peroxidase antibody, TRAb) were reviewed when available. We reviewed the results of thyroid scintigraphy when performed. Thyroid ultrasound images and accompanying radiology reports were reviewed. The description of vascular flow was assessed. We recorded whether a biopsy was performed after the initial ultrasound, and we collected the corresponding cytology reports when available. Follow-up ultrasound imaging, performed formally by radiology or at the bedside in the thyroid or endocrine clinic, was reviewed for resolution of suspicious or abnormal findings.

RESULTS

Table 1 summarizes the clinical presentation of the 10 patients. There were 8 women and 2 men, ranging in age from 29 to 76 years (mean \pm SD age, 49.0 \pm 15.0 years). At the time of diagnosis, all 10 patients had a low or suppressed thyrotropin level; 5 had an

elevated FT₄ or T₃ concentration and 5 had a normal FT₄ or T₃ concentration. Either ESR or CRP level was checked in 7 patients and was found to be elevated in 6. For the patient who had a normal ESR, this was checked by the endocrinologist 12 days after the initial set of laboratory tests performed by his primary care physician, which may explain why it was normal. The patient did not receive glucocorticoid therapy, which would have altered the measurement of the ESR. The TRAbs were absent in all 6 patients tested. Whereas a nil 24-hour radioiodine uptake helps establish the diagnosis of painful SAT during the thyrotoxic phase, only 1 patient had thyroid scintigraphy as part of the diagnostic evaluation before endocrine consultation, with a minimal uptake of 0.5%. The mean \pm SD time between onset of pain and its resolution was calculated based on patient-reported symptoms at follow-up clinic visits and was estimated to be 3.4 \pm 2.1 months.

The predominant sonographic findings identified by the radiologist are best described as ill-defined hypoechoic areas that were primarily localized to the anterior portion of the midthyroid lobe and generally corresponding to the region of tenderness indicated by the patient. Vascular flow, assessed in 9 patients, was reported as diffusely hypervascular in 2 (patients 3 and 7) and as mildly increased in a suspected nodule in patient 9. The remaining patients were described as having

TABLE 2. Sonographic and Cytologic Findings of 10 Patients With Painful Subacute Thyroiditis

Patient No.	Increased vascularity	Biopsy	Cytologic findings (if biopsy performed)	Outcome of suspicious/abnormal findings on follow-up
1	No	No		Resolved nodular findings
2	No	No		Resolved nodular findings
3	Yes	No		Resolved nodular findings
4	No	Yes	Nonnecrotizing granulomatous inflammation, benign-appearing follicular cells	Resolved nodular findings
5	Unknown	Yes	Papillary carcinoma	One nodule resolved, another incidental
6	No	No		Unknown, patient lost to follow-up
7	Yes	No		Resolving nodular changes
8	No	No		Resolved nodular findings
9	Yes	Yes	Nondiagnostic, paucicellular with multinucleated giant cells, scattered chronic inflammatory cells, rare benign follicular groups, and colloid	Resolved nodular findings
10	No	No		Resolved nodular findings

vascularity that was either normal or not increased. Of the 2 patients with diffuse hypervascularity, both had decreased thyrotropin concentrations at the time of ultrasound. The patient with mildly increased vascularity in a suspected nodule had a normal thyrotropin concentration of 1.61 μ IU/mL at the time the ultrasound was performed.

Three patients underwent a biopsy based on findings seen on initial ultrasound (Table 2). Patient 4 was found to have granulomatous inflammation, whereas patient 9 was a nondiagnostic specimen but was noted to have multinucleated giant cells and chronic inflammatory cells. Patient 5 had 2 right lower lobe nodules on the initial ultrasound. One was described as a solid, hypoechoic, 2.6-cm nodule for which a biopsy was recommended by the radiologist; the other was an incidental, smaller, solid, isoechoic, 1.2-cm nodule. After resolution of the patient's thyroiditis, the larger, 2.6-cm nodule was no longer present on follow-up imaging. However, the smaller nodule had grown slightly to 1.4 cm and was noted to be hyperechoic in appearance. The patient subsequently underwent FNA and was diagnosed as having papillary thyroid carcinoma.

On follow-up ultrasound, performed either formally or at the bedside during a subsequent thyroid or endocrine clinic visit, 8 patients had either complete resolution or improvement of the nodular and inflammatory

findings (Table 2). As noted, the incidental nodule found in patient 5 was subsequently diagnosed as papillary thyroid carcinoma. Follow-up was not available in the remaining patient. The mean \pm SD time between documentation of abnormal ultrasound findings and subsequent resolution was 4.6 ± 4.9 months. Figure 1 illustrates the abnormal findings noted on initial ultrasound for each patient and the formal follow-up ultrasound images when available. We note that 3 of the 9 patients who underwent a follow-up ultrasound did not have formal images available for presentation but were assessed in the clinic.

DISCUSSION

We report a case series of 10 patients with typical painful SAT whose thyroid ultrasound was considered to show suspicious findings. Biopsy was recommended in all patients before endocrine consultation. The patients seen in this series are typical for painful SAT, occurring more frequently in middle-aged individuals and in females.⁶ The present patients also had typical clinical and laboratory findings of painful SAT. We did not attempt a systematic review of the medical records or ultrasounds of patients with painful SAT seen in the thyroid or endocrine clinic who were not specifically referred for "suspicious" ultrasound findings.

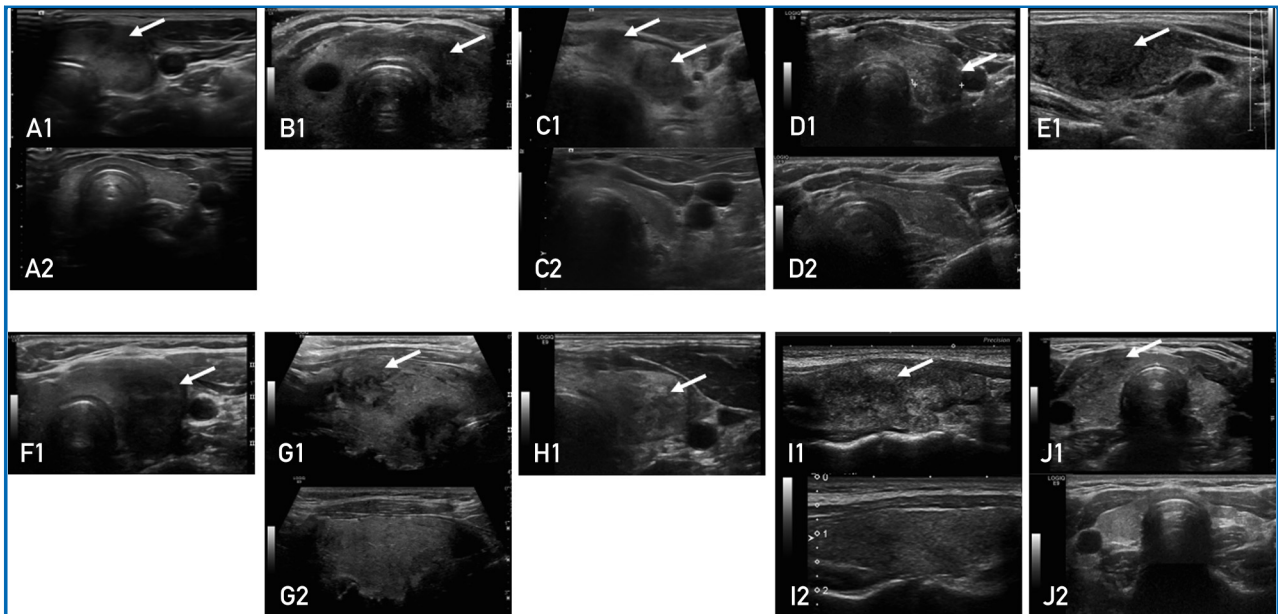


FIGURE. A1-J1, Abnormal findings seen on initial ultrasound for patients 1 through 10, respectively. A2-J2, The resolution of abnormal findings on follow-up ultrasound, when formal images are available. Arrows indicate abnormal areas as described in the formal radiology report. Patient 6 was lost to follow-up and patients 2, 5, and 8 had a follow-up visit and neck ultrasound was performed in the office by the endocrinologist, but the associated images from the visit either could not be retrieved or were of too low quality to include and so are missing from this figure (blacked out boxes).

The diagnosis of painful SAT is usually evident based on typical clinical and laboratory findings. A nil 24-hour radioiodine uptake in the presence of thyrotoxicosis is important evidence for painful SAT when the diagnosis is uncertain. In addition, when the clinical presentation is less obvious (ie, absence of pain), ultrasonography and other image modalities have shown utility.^{24,25} Published studies of thyroid ultrasound characteristics of painful SAT include descriptions such as a diffusely heterogeneous thyroid or the presence of focally marked hypoechoic areas.^{19,20} One ultrasound study reported a 41% prevalence of nodular lesions with marked hypoechoogenicity in patients with suspected SAT, although not all cases were characterized by pain.²⁶ Another recent study examined the correlation between HLA haplotype and sonographic pattern and found that deviations from the typical findings of painful SAT were associated with the presence of only *HLA-B*18:01*.²⁷ In the cases with only *HLA-B*18:01*, the ultrasound pattern tended to be more homogeneous, hypoechoic, and

unilateral, filling the entire affected thyroid lobe. Because ultrasound findings cannot specifically confirm the diagnosis of painful or painless SAT, a definitive diagnosis may require demonstration of a nil 24-hour radioiodine uptake in the setting of thyrotoxicosis.

In the present case series, we primarily observed ill-defined hypoechoic areas localized to the anterior portion of the midthyroid lobe. We note the difference between ill-defined and irregular margins, which is well-described in the American College of Radiology's Thyroid Imaging, Reporting and Data System.²⁸ According to this system, irregular margins are characterized as jagged, spiculated, or having sharp angles, and would raise suspicion for malignancy. We saw that 3 of the reports used the term *ill-defined* to describe the observed ultrasound findings, but the pattern was still considered to be suspicious and a biopsy was recommended in each case. This pattern is also distinct from what is most often observed in typical Hashimoto thyroiditis and Graves disease: either a diffuse, homogeneous, hypoechoic texture or an almost granular

alternation of small hypoechoic regions diffusely dispersed throughout the gland.²⁹

Traditionally, the thyrotoxic phase of painful or painless SAT is characterized by decreased vascularity on color Doppler, likely due to the lack of thyrotropin stimulation.^{17,18} This contrasts with the increased flow found in Graves disease, when the thyrotropin receptor is stimulated by TRAb, or in typical Hashimoto thyroiditis, when a normal or increased thyrotropin concentration is present. An increase in vascularity is observed during the recovery phase of painful SAT, likely as a consequence of thyrotropin stimulation.¹⁷ Interestingly, hypervascularity was reported in 3 of the present patients. In 1 patient, the thyrotropin level had normalized. The remaining 2 patients demonstrated hypervascularity despite a decreased serum thyrotropin level, raising the possibility that increased flow may result from inflammation rather than from thyroid stimulation. We caution that vascular flow must be carefully interpreted in relation to thyroid function test results and the disease course to avoid misdiagnosing painful SAT as Graves disease during its recovery phase.³⁰

Based on the present observations, we are concerned about the misdiagnosis of painful SAT as suspicious nodular thyroid disease requiring immediate biopsy. Several patients were convinced that they had aggressive thyroid cancer based on physician comments and the ultrasound report. In a previously reported case, a patient with a nodule considered suspicious on FNA underwent a total thyroidectomy for what proved to be subacute granulomatous thyroiditis without evidence of thyroid malignancy on final histopathologic analysis.²² Fortunately, none of the present patients had surgery for SAT, but 3 had biopsies performed based on the ultrasound readings. Most patients had relatively rapid resolution of their ultrasound findings, which supports observation as the most reasonable approach.

As a general rule, we recommend a radio-nuclide scan or endocrinology referral for patients with diffuse thyroid pain and thyrotoxicosis rather than an ultrasound examination. Although we do not recommend neck ultrasound for a diffusely tender thyroid, we acknowledge that ultrasound use for many

thyroid disorders is widespread and may be helpful. It is, therefore, important for clinicians to be familiar with the ultrasound findings in painful SAT. When suspicious ultrasound findings are noted for otherwise typical painful SAT, we favor close serial monitoring beyond the acute inflammatory phase, particularly if the diagnosis is confirmed by a nil radioactive uptake. Inflammatory findings on ultrasound are generally transient and disappear over the course of several months. Thus, FNA should be avoided but continued observation is crucial because the resolution of ultrasound findings that parallels the resolution of symptoms is definitive proof of benignity. As noted in patient 5, the presence of inflammatory nodules does not preclude the presence of suspicious neoplastic nodules or incidental carcinomas; when apparent nodules are present, monitoring beyond the resolution of thyroiditis symptoms is necessary.

Notably, in their case series, Stasiak et al³¹ presented 5 patients who were initially diagnosed as having painful SAT but were subsequently found to have a malignant tumor with poor prognosis. Thus, we concur that in the presence of escalating symptoms or atypical features, such as a rapidly enlarging thyroid gland, more urgent evaluation may be required to identify an aggressive malignancy in a timely manner. However, we disagree that ultrasound evaluation and FNA biopsy should be part of the diagnostic criteria for painful SAT. When one considers the comparative incidences of painful SAT and the rare aggressive malignancies causing malignant pseudothyroiditis, these interventions seem unnecessary in patients with otherwise typical painful SAT. Furthermore, patients receiving a nondiagnostic biopsy may experience undue anxiety and undergo additional follow-up procedures, which could pose their own risks and should not be minimized.

Limitations of this case series include its retrospective nature and small sample size. Consequently, some clinical parameters were not measured during routine care. The follow-up interval was variable and dependent on the timing of subsequent thyroid or endocrine clinic follow-up visits. We acknowledge that we did not perform a comprehensive review of ultrasound findings in all patients

with SAT seen in the thyroid or endocrine clinics during this period. We also did not systematically assess how many patients who were referred for painful SAT had suspicious ultrasound findings or underwent biopsy. In addition, we were unable to evaluate the role of nonsteroidal anti-inflammatory drugs or prednisone in modifying the evolution of abnormal ultrasound findings, which was not the purpose of this study. Given the referral nature of our practice, the prevalence of suspicious ultrasound findings in painful SAT may be exaggerated. A large-scale prospective study of ultrasound findings in painful SAT would be important but is beyond the scope of this case series.

CONCLUSION

In summary, we found that the ultrasound findings in painful SAT may be misdiagnosed as suspicious nodules. We identified 10 such cases in a 2.5-year period. We observed the resolution of these ultrasound patterns of inflammation in most patients within a few months. By reporting these findings, we hope to increase recognition of these ultrasound features, minimize unnecessary worry for patients and providers, and improve the care of individuals with painful SAT.

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Drs Daniels and Barbesino contributed equally to this work.

Abbreviations and Acronyms: CRP = C-reactive protein; ESR = erythrocyte sedimentation rate; FNA = fine-needle aspiration; FT₄ = free thyroxine; RAI = radioiodine; SAT = subacute thyroiditis; TBII = thyrotropin-binding inhibitory immunoglobulin; TPO Ab = thyroid peroxidase antibody; TRAb = thyrotropin receptor antibody; TSI = thyrotropin-stimulating immunoglobulin; T₃ = total triiodothyronine

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Correspondence: Address to Giuseppe Barbesino, MD, Thyroid Unit ACC 730, Massachusetts General Hospital, 15 Parkman St, Boston, MA 02114 (gbarbesino@mgh.harvard.edu).

ORCID

Josephine H. Li:  <https://orcid.org/0000-0003-2314-0914>

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