to differences in immunological techniques should be excluded. This is of more than theoretical importance because there are sufficient clinical differences between the North European and Japanese forms of the disease to suggest that they may not be the same[12] and at the present stage of development of B-lymphocyte alloantigen typing, precise uniformity of reagents from different laboratories has yet to be established.

At least two further possible explanations require consideration. The first is that genetic susceptibility to M.S. is not directly related to the presence of a gene coding for a particular B-lymphocyte alloantigen, but to the presence of another closely linked gene; in different populations the susceptibility gene may associate with different alleles coding for B-lymphocyte and other HLA alloantigens. If B-lymphocyte alloantigens are merely indirect markers, the true susceptibility gene product has yet to be identified.

The second explanation incorporates the assumption that an environmental agent,[13] possibly a virus,[14] is involved in the pathogenesis of M.S. Evidence from mouse model systems indicates that recognition and handling of antigens, including viruses, involves association of the antigen with products of the major histocompatibility complex (M.H.C.).[15-20] Individual M.H.C. products may have differing affinities for viral and other antigens, and therefore govern susceptibility to disease. We suggest that the association of different B-lymphocyte alloantigens with M.S. in Arab and North European populations is due to the involvement of different viruses in its pathogenesis, but both operating by the same mechanism and resulting in a similar clinical disease. This hypothesis can be tested by investigation of B-lymphocyte alloantigens in migrant populations. For example, patients with M.S. who grew up in Northern Europe but whose parents were Arab should have a significantly raised frequency of BT 101 compared to the patients in the present study who grew up in Jordan and had a high frequency of BT 102.

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POST-PARTUM TRANSIENT THYROTOXICOSIS WITH PAINLESS THYROIDITIS*

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Summary 5 patients presented with transient thyrotoxicosis and painless thyroiditis in the post-partum period. Thyrotoxicosis developed within 1–6 months of delivery. All had small non-tender goitres. Initially, all patients had elevated values for thyroxine (T4), triiodothyronine (T3) uptake, and triiodothyronine by radioimmunoassay (T3[R.I.A.]). Radioactive-iodine uptake (R.A.I.U.) was suppressed in all. In all patients, thyrotoxicosis resolved within 4 months; subsequent transient hypothyroidism occurred in 4, 1 developing permanent myxœdema. 2 had persistently elevated thyroid-antibody titres and needle-biopsy findings compatible with chronic thyroiditis. 1 became permanently hypothyroid. The other 3 patients had serial clinical and laboratory findings suggestive of painless subacute thyroiditis. Serial thyrotrophin-releasing-hormone (T.R.H.) stimulation tests are also reported. The importance of the low R.A.I.U. in recognising painless thyroiditis is emphasised. Since the disorder is self-limited, conservative therapy should be given.

Introduction

TRANSIENT hypothyroidism has been reported 3–6 mo after delivery.[1] Subacute thyroiditis was excluded by the absence of pain, fever, and raised erythrocyte-sedimentation rate. However, subacute thyroiditis without these manifestations has been reported.[2-7] Gluck et al. have reported 4 cases of chronic lymphocytic thyroiditis with transient thyrotoxicosis and low radioactive-iodine uptake (R.A.I.U.).[8] We report serial observations on 5 patients who presented in the post-partum period with transient thyrotoxicosis secondary to painless thyroiditis.

Methods

The 5 patients were outpatients attending the endocrine clinic, Mount Sinai Hospital, University of Toronto.

The triiodothyronine (T3) resin uptake was performed using silica-talc resin (normal 84–116%). Serum-thyroxine (T4) was assayed by competitive protein binding[9] (normal 4·6–11·8 μg/dl). Serum-triiodothyronine (T3) was performed by the radioimmunoassay of Chopra et al.[10] (normal 90–220 ng/dl) and serum-thyrotrophin (T.S.H.) by the radioimmunoassay of Patel et al.[11] (normal 1–6 μU/ml). The National Institutes of Health (National Institute of Arthritis, Metabolic and Digestive Diseases) human-T.S.H. assay kit and T.S.H. international reference standard 6838 from the W.H.O. International Laboratories for Biological Standards, Holly Hill, London, were used.

Antithyroglobulin antibodies (A.T.G.A.) were measured by tanned-red-cell agglutination. Antimicrosomal antibodies (A.M.S.A.) were estimated by quantitative hæmagglutination

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SUMMARY OF SERIAL T.R.H. STIMULATION-TEST RESPONSES WITH BASELINE THYROID INDICES

Case no.	Interval since delivery	Baseline thyroid indices				T.R.H. stimulation response	
		Serum-T4 (μg/dl)	T3 uptake (%)	T3 (R.I.A.) (ng/dl)	T.S.H. (μU/ml)	T.S.H. peak (at 20 min) (μU/ml)	T3 (R.I.A.) peak (at 120 min) (ng/dl)
1	11 mo	4·7	101	101	4·3	52·2	181
	2 yr	6·4	97	104	2·8	32·8	134
2	1 mo	14·4	90	369	1·5	2·7	396
	3 mo	29·6	183	499	1·0	1·0	643
3	5 mo*	8·3	97	202	15·3	55·1	317
	10 mo*	8·7	76	177	6·5	34·2	378
4	2 yr	6·5	92	104	1·0	12·7	140
5	4 mo	1·0	51	23	64·0	64·0	10
	1½ yr*	8·3	76	180	8·5	64·0	238
	3 yr	5·8	97	162	28·4	64·0	169
	Normal range	4·6–11·8	84–116	90–220	1·0–6·0	<25·0	increment >30%

*Performed while patient was taking oral contraceptive hormones.

using the 'Sera-Tek' kit (Ames Laboratories) or by complement fixation. Thyrotrophin-releasing hormone (T.R.H.) was supplied by Cal-Biochem Laboratories, San Diego, California, and prepared for human use with the approval of the University of Toronto Human Experimentation Committee. For the T.R.H. stimulation test, an intravenous injection of 200 μg T.R.H. was used. T3 and T.S.H. responses were interpreted according to the criteria of Shenkman et al.[12] and Walfish and Ulbright.[13] Needle biopsy was done by a fine-needle technique as described by Walfish et al.[14] The 24-hour R.A.I.U. was also measured (normal 8–26%).

Case-reports

Case 1

A 31-year-old woman, 6 months post-partum, complained of fatiguability, palpitations, nervousness, and heat intolerance. Her weight was stable and menses normal. Surreptitious use of thyroxine or iodine was excluded. 1 month post-partum, she was treated for staphylococcal septicæmia. A hysterosalpingogram had been done 2 yr before. On examination, sinus tachycardia, brisk reflexes, and a diffuse (25 g), firm, non-tender thyroid were noted. Serum-T4 was 15·5 μg/dl and T3 uptake 130%. Thyroid antibodies were negative. R.A.I.U. was less than 1%. By 7 mo post-partum, she appeared clinically euthyroid. By contrast, serum-T4 and T3 uptake were then in the hypothyroid range, but they returned to normal within a month. R.A.I.U. had risen to 11·7%. 11 months post partum, while she was clinically and biochemically euthyroid, a T.R.H. stimulation test revealed borderline primary hypothyroidism (see table). Over the following 8 months she remained well with normal T4, T3 uptake, and T.S.H. levels. A repeat T.R.H. test 1½ yr after the onset of thyrotoxicosis revealed borderline normal T.S.H. and T3 responses (see table). 2 yr post partum, serum-T4 was 6·4 μg/dl, T3 uptake 94%, T.S.H. 2·8 μU/ml, and thyroid antibodies negative.

Case 2

A 27-year-old woman was seen for possible unilateral exophthalmos 3 days post partum. There were no symptoms of thyroid dysfunction. Several years previously a hysterosalpingogram had been performed. Clinical examination revealed no goitre, neck pain, or true exophthalmos. R.A.I.U. was 25%. T4 was 17·9 μg/dl and T3 uptake 60%, reflecting the increased thyroxine-binding globulin. 1 month post partum, although she had no symptoms, a diffuse non-tender goitre was noted. Serum-T4 was 14·4 μg/dl and T3 uptake 90%. A T.R.H. stimulation test was compatible with hyperthyroidism (see table). 3 months post partum, serum-T4 and T3 uptake were unchanged. A.T.G.A. were negative. A.M.S.A. by complement fixation were 3+. A repeat T.R.H. stimulation test was unchanged. Within 2 wk, increasing nervousness and mild tremor were noted. There was no pain in neck, ear, or jaw. The patient denied thyroid or iodine ingestion. On examination, sinus tachycardia, lid lag, brisk reflexes, and a diffuse (35 g) non-tender goitre were noted. R.A.I.U. was only 2%. An iodine-131 scan of the chest and abdomen was negative. Needle-biopsy findings were consistent with chronic thyroiditis. She was given amylobarbitone sodium and propranolol as symptomatic therapy. By 5 months post partum, the symptoms of thyrotoxicosis had resolved and the goitre regressed. R.A.I.U. had risen to 6%. Serum-T4 was 9·4 μg/dl, T3 uptake 102%, T3(R.I.A.) 140 ng/dl, and serum-T.S.H. <1 μU/ml. She was lost to follow-up.

Case 3

A 21-year-old woman, was referred 6 wk post partum because of increasing nervousness. She had no localised pain. Ingestion of thyroxine or iodine was denied. Serum-T4 was >25 μg/dl and T3 (R.I.A.) 700 ng/dl. On examination, sinus tachycardia, lid lag, tremor, brisk reflexes, and a diffuse (35 g) non-tender goitre were noted. R.A.I.U. was <1%. A.M.S.A. were 1/1600. Amylobarbitone sodium and propranolol were given. 10 wk post-partum, sinus tachycardia and brisk reflexes were still present. Serum-T4 was 4·5 μg/dl, T3 uptake 89%, T3(R.I.A.) 122 ng/dl, and serum-T.S.H. 1·7 μU/ml. A.M.S.A. titre had increased to 1/6400. R.A.I.U. had risen to 38%. 4 months after presentation there was no clinical evidence of thyroid dysfunction. Serum-T4 was 8·3 μg/dl, T3 uptake 97%, and T.S.H. 15 μU/ml. Subsequently, oral contraceptives were started. A T.R.H. stimulation test revealed a hyperreactive serum-T.S.H. but a normal T3 response (see table). 8 months post partum serum-T4 was 9·0 μg/dl, T3 uptake 76%, T3(R.I.A.) 180 ng/dl, and serum-T.S.H. 7·4 μU/ml. 10 months post partum, serum-T4 was 8·7 μg/dl, T3 uptake 76%, and serum-T.S.H. 6·5 μU/ml. A.M.S.A. titre had fallen to 1/400. A T.R.H. stimulation test was borderline normal (see table).

Case 4

A 35-year-old woman presented with increasing anxiety 3 months after delivery. A hysterosalpingogram had been performed 3 yr before. Examination revealed diffuse (30 g) non-tender goitre, sinus tachycardia, lid lag, and brisk reflexes. Serum-T4 was 15·2 μg/dl, T3 uptake 155%, and T3(R.I.A.) 392 ng/dl. R.A.I.U. was <1%. Thyroid antibodies were negative. 4 months post partum, serum-T4 was 4·2 μg/dl and T3 uptake 88%. The patient was clinically euthyroid. At 7 months there were no signs of thyroid dysfunction. The thyroid was only mildly enlarged. Serum-T4 was 7·7 μg/dl, T3(R.I.A.) 140 ng/dl, and serum-T.S.H. 2·2 μU/ml. She has remained clinically and biochemically euthyroid for 38 months (see table). R.A.I.U. is 10·2%.

Case 5

A 21-year-old woman delivered in December, 1973. Because

of a low serum-T4 value in the cord blood, thyroid indices were obtained from both infant and mother 4 wk post partum. These were normal in the child. However, the mother's serum-T4 was 12·9 μg/dl, T3 uptake 166%, T3(R.I.A.) 418 ng/dl, and serum-T.S.H. <1 μU/ml. She had no symptoms of thyrotoxicosis. A diffuse (35 g) firm goitre and brisk reflexes were noted on examination. R.A.I.U. was 6%.

At 4 months the patient had fatigue, dry skin, and muscle cramps. Periorbital œdema and delayed reflexes were noted. The goitre was unchanged. While she was on oral contraceptives serum-T4 was 1 μg/dl and T3 uptake 51%. T.R.H. stimulation was compatible with hypothyroidism (see table). L-Thyroxine was prescribed. By 6 months post partum she was clinically and biochemically euthyroid, and the goitre had regressed. She remained well on L-thyroxine for 8 months. 6 months after withdrawal of L-thyroxine she was clinically euthyroid. Serum-T4 was 8·3 μg/dl, T3 uptake 76%, serum-T.S.H. 8·5 μU/ml. A repeat T.R.H. stimulation test indicated a hypothyroid response (see table). She remained well for 18 months without treatment. Then increasing fatigue, sinus bradycardia, and delayed reflexes appeared. The thyroid was diffusely enlarged to 1½ times normal size. 3 months after discontinuation of oral contraceptives serum-T4 was 5·8 μg/dl and T3 uptake 97%, but the basal serum-T.S.H. was elevated at 15 μUm/l. A repeat T.R.H. stimulation confirmed hypothyroidism (see table). A.M.S.A. were 1/25 600. Thyroid-biopsy findings were consistent with chronic thyroiditis. A month later, serum-T4 was 3·4 μg/dl, T3 uptake 102%, and serum-T.S.H. 28·4 μU/ml. L-Thyroxine was started again.

Discussion

Subacute thyroiditis is typically characterised by an enlarged acutely tender thyroid gland. Initially, elevated thyroid indices with a suppressed R.A.I.U. are seen.[2–4] Pain occurs in over 90% of patients.[3 4] However, recent reports[6 7] suggest that painless subacute thyroiditis may be more common than previously believed.

Our patients, who presented post partum, all had characteristic laboratory features of the toxic phase of subacute thyroiditis, with an increased serum-T4 and T3 uptake and a depressed R.A.I.U. None had pain in neck, jaw, or ear. Accordingly, we propose that the post-partum syndrome reported by Amino et al.[1] (transient hypothyroidism occurring 3–6 months after delivery) might have been the consequences of a similar painless-thyroiditis syndrome.

Only 1 patient (case 1) had obvious symptoms of thyrotoxicosis. 1 patient (case 5) was symptom-free, and the other 3 had noticed only mild nervousness. All had clinical signs of thyrotoxicosis, with sinus tachycardia and brisk reflexes. All had diffuse goitres, but only 1 patient (case 5) had noticed this previously. No patient received corticosteroids. 4 received only amylobarbitone sodium and propranolol during the thyrotoxic phase. Thyrotoxicosis had resolved in all patients by 4 months. 1 patient (case 2) had two T.R.H. stimulation tests during the acute phase which showed hyperthyroid responses. Poor T.S.H. responsiveness to T.R.H. has been reported during the acute phase of subacute thyroiditis.[15]

3 patients (cases 1, 2, 4) had hysterosalpingograms performed several years before presentation. There have been reports of hyperthyroidism following the use of iodinated contrast media.[16] However, because of the long interval (2–3 yr) between radiography and the onset of hyperthyroidism in our patients, we do not think that contrast media contributed to their symptoms. Iodinated radiography material may suppress the R.A.I.U.[17] Suppressed R.A.I.U. levels subsequently recovered in the 3 patients who had had hysterosalpingograms, indicating that this was not a contributing factor.

In cases 1–4, indices of thyroid function returned to normal within 6 months. Transient biochemical hypothyroidism was seen in all but case 2. All except case 5 5 have remained clinically euthyroid for 4 months–2½ yr. In case 1 responses to T.R.H. stimulation 6 months and 1½ yr after onset were consistent with possible latent hypothyroidism. However, serum-T4, T3 uptake, and serum-T.S.H. have remained normal. Case 3 showed a similar T.R.H. test at 4 months, but a further test at 8 months was normal. In a recent study,[15] 5 patients had T.R.H. stimulation tests 2–6 months after subacute thyroiditis. 4 of these patients had high baseline-T.S.H. values at 2–4 months, and an exaggerated response to T.R.H. was seen in 3, 1 of whom showed this type of response a year after the acute phase. As in the previously cited report,[15] T.R.H. stimulation tests in our cases 2 and 4 may indicate that normalisation of the hypothalamo/pituitary/thyroid axis may be a slow process.

Permanent myxœdema in subacute thyroiditis is very rare. Volpe et al.[18] described a patient with biopsy-proven subacute thyroiditis who initially had high antibody titres, which rose even higher coincident with the development of hypothyroidism. Ivy[19] reported a case of subacute thyroiditis which terminated in permanent myxœdema. Gluck et al.[8] reported 4 cases of chronic thyroiditis confirmed by needle biopsy, with transient thyrotoxicosis and low R.A.I.U. Unfortunately, the follow-up was short, and permanent hypothyroidism was not documented. In our cases 2 and 5, elevated antibody titres and needle-biopsy findings were compatible with a diagnosis of chronic lymphocytic thyroiditis. In case 5, after a recovery phase of euthyroidism, hypothyroidism recurred in association with goitre, persistently elevated A.M.S.A. titres, and needle-biopsy evidence compatible with chronic thyroiditis. Baseline T.S.H. and T.R.H. responses also indicated hypothyroidism. Although painless subacute thyroiditis is probably the underlying condition in most of our patients, chronic thyroiditis may also occur.

Painless thyroiditis may exist in the post-partum period, and only the presence of a low R.A.I.U. can differentiate it from Graves' disease. Awareness of this syndrome will assist in the exclusion of post partum functional disorders. Conservative management should be used in all phases. Antimicrosomal antibodies should be monitored throughout the course of the disorder. Persistent rather than transiently elevated titres, as well as needle biopsy findings, may indicate underlying chronic thyroiditis and the associated risk of developing permanent hypothyroidism.

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References overleaf

SUBCUTICULAR SUTURING AFTER APPENDICECTOMY

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Summary The results of using interrupted nylon skin sutures or subcuticular polyglycolic acid (P.G.A.) sutures after appendicectomy were compared in a prospective controlled trial in 127 patients. Wound infections were significantly more common when subcuticular skin closure was used.

Introduction

SUBCUTICULAR suturing is becoming increasingly popular as a method of skin closure.[1] However, it has been suggested that it may increase the risk of wound infection by "sealing in" infection in some wounds.[2 3] This effect has never been quantitatively demonstrated, however, and therefore we compared the results of subcuticular polyglycolic acid (P.G.A.) suturing with interrupted 00 nylon sutures after appendicectomy in a prospective clinical trial.

Methods

All patients undergoing appendicectomy through a right iliac fossa incision over a 16-month period were included in the trial. Patients in whom a midline or paramedian incision was made were not included.

Patients were randomly allocated into two groups at the start of the operation. Skin in one group was sutured with subcuticular P.G.A. and in the other with interrupted 00 nylon mattress sutures spaced 1 cm apart. In all cases the deeper layers of the incision were approximated with 00 P.G.A. Wounds were kept as small as was compatible with good access, but were extended without hesitation if difficulties were encountered. The appendix stump was invaginated. When free pus without any evidence of localisation was present the peritoneal cavity was drained. In these cases the drain was brought through a stab wound below the main incision, the wound being irrigated with noxythiolin during closure. The appendix at operation was described as normal, inflamed, gangrenous, or perforated, but the final classification depended on the histological features of the specimen.

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All wounds were inspected daily while the patient was in hospital. Patients usually returned home after 5 days. Sutures, if present, were removed by a visiting district nurse. Wounds were inspected again a month after leaving hospital, when patients were questioned about the type and duration of any wound discharge noted after their return home. If this occurred they had been asked to return to the ward for swabs to be taken for cultures and antibiotic sensitivity tests. An infection was defined as an obvious discharge of pus either spontaneous or after incision.[4]

Results

127 patients were included in the trial; 62 skin incisions were sutured with subcuticular P.G.A. (group A) and 65 with interrupted 00 nylon (group B). There were 30 wound infections overall (table I). In the 62 patients sutured with subcuticular P.G.A. there were 21 wound infections (33%). 9 (14%) incisions sutured with interrupted nylon sutures became infected. This difference is significant ($\chi^2=5{\cdot}98$; $P<0{\cdot}02$).

The frequency of a subsequent wound infection was examined in relation to the degree of appendicular inflammation (table II). In the gangrenous and perforated cases 9 of 11 of the wounds closed by subcuticular suturing became infected, compared with 4 out of 10 wounds closed with interrupted nylon sutures. However, because of the small numbers involved these differences are not statistically significant. When the appendix was classified as inflamed, 7 wound infections followed in the subcuticular group while all the wounds closed with interrupted nylon healed without infection developing ($\chi^2=4{\cdot}63$; $P<0{\cdot}05$). 4 of the 7 infections were not apparent when the patient returned home between the 4th and 6th postoperative days; the wounds started to discharge 9 to 12 days postoperatively. On the other hand, in the perforated or gangrenous group all infections became apparent earlier, while the patient was in hospital—i.e., between the 4th and 7th postoperative days. In all infected cases the wound culture revealed coliform and bacteroides organisms together, in some cases, with non-hæmolytic streptococci.

TABLE I—WOUND INFECTIONS IN RELATION TO SUTURE METHOD

—	Total	Infected wounds
Group A (subcuticular P.G.A.)	62	21
Group B (interrupted nylon)	65	9
Total	127	30

$\chi^2=5{\cdot}98$ $P<0{\cdot}02$

TABLE II—STATE OF APPENDIX IN RELATION TO POSTOPERATIVE WOUND INFECTION

			A		B	
	Total	Infected	Uninfected	Infected	Uninfected	Infected
Normal	24	1	9	–	14	1
Inflamed	66	7	29	7*	30*	0
Gangrenous	16	9	1	5	6	4
Perforated	21	13	2	9	6	4
Totals	127	30	41	21	56	9

* $\chi^2=4{\cdot}63$; $P<0{\cdot}05$.