Edematous wall thickening of the gallbladder induced by hyperthyroidism

A case report

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Abstract

Rationale: Hyperthyroidism, such as Basedow disease, causes fluid retention, although the common cause is volume overload due to congestive heart failure. In addition, hyperthyroidism and Basedow disease are known to cause pulmonary hypertension. Edematous thickening of the gallbladder wall is caused by venous blood congestion. The feature of edematous wall thickening of the gallbladder on abdominal computed tomography (CT) is subserosal edema and is often accompanied by a periporal collar sign.

Patient concerns: A 30-year-old woman was referred to our hospital because of liver dysfunction, edematous gallbladder wall thickening, and fluid retention. In addition, the patient developed hyperthyroidism and heart failure. Enhanced abdominal CT revealed edematous wall thickening of the gallbladder and a periporal collar sign.

Diagnosis: We suspected that fluid retention and congestion were caused by hyperthyroidism and Basedow disease.

Interventions: On admission, we started thiamazole therapy for Basedow disease, and her thyroid hormone levels normalized.

Outcomes: Abdominal CT revealed disappearance of edematous wall thickening of the gallbladder, which was likely associated with an improvement in thyroid function. The patient was discharged 10 days after admission.

Lessons: We encountered a case of hyperthyroidism and Basedow disease accompanied by edematous wall thickening of the gallbladder and various fluid retentions as the first symptoms. Such edematous wall thickening of the gallbladder and various fluid retentions were reduced, together with the improvement of hyperthyroidism.

Abbreviations: BNP = brain natriuretic peptide, CT = computed tomography.

Keywords: Basedow disease, edematous wall thickening of the gallbladder, fluid retention, hyperthyroidism

1. Introduction

Fluid retention is sometimes observed in patients with thyroid disease, especially with hypothyroidism.[1] Under hypothyroid conditions, edema is caused by a reduction in enzymatic activity, resulting in a buildup of mucopolysaccharides, which is called nonpitting edema. On the other hand, hyperthyroidism, such as Basedow disease, rarely causes fluid retention, although the common cause is volume overload due to congestive heart failure.[2] In addition, it is known that hyperthyroidism and Basedow disease sometimes cause pulmonary hypertension.[3] Moreover, hypoproteinemia and iron deficiency anemia can be additional factors for fluid retention under hypothyroid conditions.

Edematous wall thickening of the gallbladder is caused by venous blood congestion, which is associated with heart failure, hypoproteinemia, acute hepatitis, and liver cirrhosis.[4] The feature of edematous wall thickening of the gallbladder on abdominal computed tomography (CT) is subserosal edema because the connective tissue around the gallbladder is thin and dense. Edematous wall thickening of the gallbladder differs from infectious wall thickening of the gallbladder, which is sometimes caused by acute cholecystitis. In addition, edematous wall thickening of the gallbladder is often accompanied by a periporal collar sign, which likely represents periporal edema.[5]
In this report, we show a subject with edematous wall thickening of the gallbladder and various fluid retention induced by hyperthyroidism and such edematous wall thickening of the gallbladder was the first symptom for diagnosis of Basedow disease. Her edematous wall thickening of the gallbladder and various fluid retentions were improved, which were likely associated with decreased thyroid hormone levels.

2. Case presentation

A 30-year-old Japanese woman was referred to our hospital because of liver dysfunction, edematous wall thickening of the gallbladder, and fluid retention on an abdominal CT. She had no remarkable or family history. She was a nonsmoker and did not drink alcohol. The patient height and body weight were 164.2 cm and 48.8 kg, respectively. Her vital signs were as follows: temperature, 36.0°C; blood pressure, 130/60 mmHg; heart rate, 60 bpm; and oxygen saturation, 98%. Table 1 shows the laboratory data on admission. She had iron deficiency anemia (red blood cells, 397×10⁶/μL; Hemoglobin, 9.8 g/dL; iron, 17 μg/dL; ferritin, 7 ng/mL). Renal function was within the normal range, and liver dysfunction was observed as follows: aspartate aminotransferase, 109 U/L; alanine transaminase, 178 U/L; albumin, 4.6 g/dL; PT-INR, 1.09; and ferritin, 7 ng/mL. Renal function was within the normal range, and liver dysfunction was observed as follows: aspartate aminotransferase, 109 U/L; alanine transaminase, 178 U/L; albumin, 4.6 g/dL; PT-INR, 1.09; and ferritin, 7 ng/mL.

In addition, enhanced ultrasonography revealed edematous wall thickening of the gallbladder and subserosal edema (Fig. 1). In addition, enhanced chest and abdominal CT revealed edematous wall thickening of the gallbladder and a periportal collar sign, which likely represented gallbladder and subserosal edema (Fig. 1). In addition, enhanced ultrasonography revealed edematous wall thickening of the gallbladder and subserosal edema (Fig. 1).

In this report, we show a subject with edematous wall thickening of the gallbladder and various fluid retention induced by hyperthyroidism and such edematous wall thickening of the gallbladder was the first symptom for diagnosis of Basedow disease. Her edematous wall thickening of the gallbladder and various fluid retentions were improved, which were likely associated with decreased thyroid hormone levels.

Table 1

| Variable                  | Result       | Reference range |
|---------------------------|--------------|-----------------|
| Peripheral blood          |              |                 |
| White blood cells (μL)    | 4230         | 3300–8600       |
| Red blood cells (×10⁶/μL)| 397          | 386–492         |
| Hemoglobin (g/dL)         | 9.8          | 11.6–14.8       |
| Platelets (×10⁶/μL)       | 14.4         | 15.8–34.8       |
| Blood biochemistry        |              |                 |
| Total protein (g/dL)      | 5.0          | 6.6–8.1         |
| Albumin (g/dL)            | 2.9          | 4.1–5.1         |
| Globulin (g/dL)           | 2.1          | 2.2–3.4         |
| Total bilirubin (mg/dL)   | 0.6          | 0.4–1.5         |
| AST (U/L)                 | 109          | 7–23            |
| ALT (U/L)                 | 178          | 13–30           |
| LDH (U/L)                 | 166          | 124–222         |
| ALP (U/L)                 | 115          | 106–322         |
| γ-GTP (U/L)               | 33           | 9–32            |
| BUN (mg/dL)               | 7            | 8–20            |
| Creatinine (mg/dL)        | 0.46         | 0.46–0.79       |
| Cholinesterase (U/L)      | 201          | 201–421         |
| Uric acid (mg/dL)         | 3.9          | 2.6–5.5         |
| Creatine Kinase (U/L)     | 17           | 41–153          |
| Amylase (U/L)             | 42           | 44–132          |
| CRP (mg/dL)               | 0.13         | <0.14           |
| BNP (pg/mL)               | 565.9        | 0.0–18.4        |
| Plasma glucose (mg/dL)    | 92           |                 |
| Total cholesterol (mg/dL) | 100          | 142–248         |
| Iron (μg/dL)              | 17           | 40–188          |
| Ferritin (ng/mL)          | 7            | 5–160           |
| Thyroid marker            |              |                 |
| TSH (μU/mL)               | <0.010       | 0.400–6.000     |
| FT3 (pg/mL)               | 8.14         | 2.50–4.20       |
| FT4 (ng/mL)               | 2.02         | 0.80–1.60       |
| Electrolytes              |              |                 |
| Sodium (mmol/L)           | 142          | 138–145         |
| Potassium (mmol/L)        | 3.4          | 3.6–4.8         |
| Chloride (mEq/L)          | 108          | 101–108         |
| IP (mg/dL)                | 4.7          | 2.7–4.6         |
| Calcium (mg/dL)           | 8.1          | 8.8–10.1        |
| Magnesium (mg/dL)         | 1.9          | 1.9–2.6         |
| Coagulation test           |              |                 |
| PT-sec (s)                | 12.2         | 9.3–12.5        |
| PT-INR                    | 1.09         | 0.85–1.13       |
| PT-activity (%)           | 86.4         | 80.7–125.2      |
| APTT (s)                  | 31.0         | 26.9–38.1       |
| Fibrinogen (mg/dL)        | 189          | 160–380         |
| D-dimer (μg/mL)           | 5.2          | <1.0            |
| Virus antibody            |              |                 |
| HBs antigen               | (–)          | (–)             |
| HCV Ab                    | (–)          | (–)             |
| HA-IgM Ab                 | (–)          | (–)             |
| CMV IgG Ab                | 12.1 (+)     | <2.0            |
| CMV IgM Ab                | 0.31 (+)     | <0.80           |
| EBV anti-VCA IgG Ab       | 11.3 (+)     | <0.5            |
| EBV anti-VCA IgM Ab       | 0.0 (+)      | <0.5            |
| EBV anti-EBNA IgG Ab      | 3.8 (+)      | <0.5            |
| Chlamydia trachomatis IgA | 0.18 (+)     | <0.90           |
| Chlamydia trachomatis IgG | 0.12 (+)     | <0.90           |
| Thyroid antibody          |              |                 |
| TSB (μg/mL)               | 1078         | 1290            |
| TRAb (U/L)                | 8.7          | <1.0            |
| TPOAb (μU/mL)             | 18.4         | 18.4–28.0       |

γ-GTP = γ-glutamyltransferase, Ab = antibody, ALP = alkaline phosphatase, APTT = activated partial thromboplastin time, AST = aspartate aminotransferase, ALT = alanine aminotransferase, BUN = blood urea nitrogen, BNP = brain natriuretic peptide, CRP = C-reactive protein, EBNA = EBV nuclear antigen, EBV = Epstein-Barr virus, FT3 = free triiodothyronine, FT4 = free thyroxine, HBs = hepatitis B surface, HCV = hepatitis C virus, IgG = immunoglobulin G, IgM = immunoglobulin M, IP = inorganic phosphorus, LDH = lactate dehydrogenase, PT = prothrombin time, PT-INR = PT-international normalized ratio, TgAb = thyroid stimulating antibody, TgAb = thyroid peroxidase antibody, TRAb = thyroid-stimulating hormone receptor antibody, TgAb = thyroid-stimulating antibody, TSH = thyroid-stimulating hormone, VCA = viral capsid antigen.
performed chest and abdominal CT again (Fig. 2, right panel), and her symptoms improved. Chest and abdominal CT revealed the disappearance of edematous wall thickening of the gallbladder, which was likely associated with improvement in thyroid function. Moreover, the periportal collar sign, the increase in inferior vena cava diameter with heart failure, and splenomegaly were improved, and pleural effusion and ascites also disappeared, probably due to the improvement of thyroid hormones.

3. Discussion and conclusions

Herein, we report a case of edematous gallbladder wall thickening associated with fluid retention and congestion. In addition, this case is very rare and interesting because her edematous wall thickening of the gallbladder was induced by hyperthyroidism and Basedow disease. Edematous wall thickening of the gallbladder is different from infectious wall thickening of the gallbladder. In general, infectious wall thickening of the gallbladder is associated with infectious diseases, such as cholecystitis; therefore, in many cases, there is gallbladder distention and diffuse wall thickening. In contrast, edematous wall thickening of the gallbladder is caused by congestion of venous blood, which is associated with heart failure, hypoproteinemia, acute hepatitis, and liver cirrhosis. Reflecting congestion of venous blood, the features of edematous wall thickening of the gallbladder on abdominal CT are subserosal edema and periportal collar sign, which likely represent periportal edema. In our patient, edematous wall thickening of the gallbladder was observed on abdominal CT. In addition, since she was complicated by liver dysfunction, we checked for viral infection associated with the liver. In addition, we evaluated thyroid hormones because thyroid disease can result in fluid retention. Hepatitis virus antibodies showed a pattern of prior infection. However, we detected hyperthyroidism and diagnosed the patient with Basedow disease. Therefore, we concluded that the pathology of the edematous wall thickening of the gallbladder was caused by hyperthyroidism and its complications, such as fluid retention and heart failure.

In general, it is well known that nonpitting edema is caused by hypothyroidism. However, hyperthyroidism causes fluid retention with volume overload owing to congestive heart failure. In addition, hyperthyroidism and Basedow disease sometimes cause mild pulmonary hypertension, although severe pulmonary hypertension is rare in patients with Basedow disease.[6] Our patient had mild pulmonary hypertension on echocardiography, and it seemed that she had heart failure due to elevated BNP levels. Her pulmonary hypertension and heart failure were caused by fluid retention and congestion, which seemed to have caused edematous wall thickening of the gallbladder. Moreover, she had liver dysfunction, which was often complicated by Basedow disease. Therefore, we initially considered that the liver and gallbladder were associated with the causes of fluid retention and congestion with heart failure. Her subjective symptoms improved, with improvement in heart failure and pulmonary hypertension. However, her edematous wall thickening of the gallbladder persisted until the hyperthyroidism improved. Therefore, we believe that edematous wall thickening of the gallbladder was at least in part associated with hyperthyroidism and Basedow disease, although the main factors of edematous wall thickening of the gallbladder might have been heart failure and pulmonary hypertension.

Her heart failure may have been caused by other factors. Chronic heart failure is a chronic condition of heart failure or valvular heart disease that occurs mainly in elderly patients. On the other hand, acute heart failure is mainly caused by acute myocardial infarction or excessive stress. She was 30 years old, and her electrocardiogram and echocardiography did not show any possibility of acute myocardial infarction or valvular heart disease. Since she was the mother of 2 children, she might have been under excessive stress. However, since her fluid retention improved after normalization of thyroid function, it was likely that her various fluid retentions and heart failure were mainly caused by hyperthyroidism.

Taken together, we should bear in mind that hyperthyroidism and Basedow disease could be accompanied by edematous wall thickening of the gallbladder and various fluid retentions as the first symptoms associated with heart failure and pulmonary hypertension. In addition, we should know that such edematous wall thickening of the gallbladder and various fluid retentions could be reduced together with the improvement of hyperthyroidism and Basedow disease. Therefore, when examining subjects with edematous wall thickening of the gallbladder, the

Figure 1. Abdominal ultrasonography. Edematous wall thickening of the gallbladder is detected (12.0 mm) (white arrow), and the inner cavity is not enlarged, although the gallbladder is enlarged in diameter (44.0 mm). In addition, edematous wall thickening of the gallbladder was observed, accompanied by subserosal edema (white arrowhead).
possibility of hyperthyroidism and Basedow disease should be considered.

Author contributions

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