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Clinical characteristics and outcomes of an exogenous thyrotoxicosis epidemic in prison

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ABSTRACT

Introduction An outbreak of exogenous thyrotoxicosis is an uncommon cause of thyrotoxicosis. This study aimed to investigate the characteristics and outcomes of exogenous thyrotoxicosis and electrolyte imbalance in a prison during an outbreak of exogenous thyrotoxicosis in the Phitsanulok, Thailand prison.

Methods This study collected cross-sectional data during an outbreak of thyrotoxicosis among inmates at Phitsanulok prison between 29 December 2019 and 17 January 2020. In the first phase, a total of 2815 prisoners were screened for thyroid-stimulating hormone (TSH), potassium levels and pulse rate. In the second phase, samples from 490 male prisoners were collected for test on thyroid function, serum electrolytes and urine electrolytes. Thyroglobulin levels were also measured in patients with thyrotoxicosis. A questionnaire was used to obtain patient information about signs and symptoms of thyrotoxicosis.

Results The prevalence of subclinical thyrotoxicosis was 78.1%. The pulse rate was significantly higher in the subclinical thyrotoxicosis group. Weight loss, palpitation, muscle weakness and fatigue were found predominantly in the subclinical thyrotoxicosis group. The prevalence of hypokalaemia was 38.4%; however, there was no difference between subclinical thyrotoxicosis and normal TSH. The mean magnesium levels were significantly lower in the subclinical thyrotoxicosis group. Patients with hypokalaemia mainly showed potassium loss through the kidneys. Almost all patients with suppressed TSH levels had low to normal thyroglobulin levels. In addition, the mean of calculated total step-up deiodinase activity in patients with subclinical thyrotoxicosis was lower than 30 nmol/s, which was an additional clue to confirm exogenous thyrotoxicosis. The frozen meat during the outbreak had higher levels of thyroid hormone compared with the control group.

Conclusions With an outbreak of thyrotoxicosis, most likely due to exposure to exogenous thyroid hormone in frozen meat, our findings have raised awareness of nutritional problems in prison. The development of surveillance systems to prevent outbreaks is urgently needed.

WHAT IS ALREADY KNOWN ON THIS TOPIC

⇒ Hamburger thyrotoxicosis, a rare aetiology of thyrotoxicosis, arises from the inadvertent ingestion of thyroid hormones via contaminated meat sources.
⇒ Incidences of hamburger thyrotoxicosis have been documented across several nations, including Thailand.

WHAT THIS STUDY ADDS

⇒ Three-quarters of inmates at Phitsanulok prison exhibited subclinical thyrotoxicosis during the thyrotoxicosis epidemic.
⇒ The prevalence of electrolyte imbalances did not exhibit significant differences between the subclinical thyrotoxicosis group and the group with normal thyroid-stimulating hormone levels.

HOW THIS STUDY MIGHT AFFECT RESEARCH, PRACTICE OR POLICY

⇒ This study raises awareness regarding dietary concerns within the prison setting and underscores the imperative need for the establishment of surveillance systems to prevent future epidemic occurrences.

INTRODUCTION

Thyrotoxicosis is a manifestation of excessive thyroid hormones, which has a hypermetabolic effect. Common aetiologies of thyrotoxicosis are Graves' disease and toxic multinodular goitre. Uncommon aetiologies are thyroiditis, ectopic thyroid hormone production and exogenous thyrotoxicosis, both intentional and unintentional, such as ingestion of meat contaminated with thyroid tissue or hamburger thyrotoxicosis.

The first case of hamburger thyrotoxicosis was reported in 1984.[1] During the same period in 1985, the Minnesota case-control study found that gullet trimming of thyroid tissue, a procedure that removes muscle from the bovine larynx for beef, was tolerant to heat. From these studies, the US Department of Agriculture prohibited the procedure of

gullet trimmings in all meat-packing plants.[2] However, outbreaks of hamburger thyrotoxicosis have been reported in many countries.[3–7]

In Thailand, thyrotoxicosis outbreaks have been reported in 10 prisons with a total of 1047 cases from 2016 to 2019. A total of 30 patients were admitted to the hospital. In 2016, a case-control study of the first outbreak in Thailand showed that pork consumption was associated with the outbreak.[8] In 2019, the sixth outbreak in Thailand, 145 patients were diagnosed with thyrotoxicosis and 14 patients with hypokalaemia. The pork sample was examined, and a high level of thyroid hormone was found.[9] The assumption about the aetiologies of thyrotoxicosis and hypokalaemia was the contamination of meat due to the presence of thyroid tissue. However, previous studies reported only the data of hospitalised patients.

The latest outbreak of thyrotoxicosis among prisoners was reported on 29 December 2019 at Phitsanulok prison. Many patients presented with generalised weakness, tachycardia, myalgia and fatigue. Laboratory investigation found low thyroid-stimulating hormones (TSH), high levels of free triiodothyronine (free T3) and free thyroxine (free T4), and hypokalaemia.[10] This study aimed to investigate the characteristics and outcomes of exogenous thyrotoxicosis and electrolyte imbalance in the prison during an outbreak of exogenous thyrotoxicosis.

METHODS

Population

A retrospective study was carried out on inmates at Phitsanulok prison who were older than 18 years and were imprisoned during the outbreak of thyrotoxicosis.

Approach to participants

The first index case was reported on 29 December 2019. Nine days later, 2815 prisoners were screened with pulse rate measurements and blood tests for TSH and potassium levels. Two weeks after the first report of thyrotoxicosis, we obtained information from 490 prisoners about signs and symptoms through a questionnaire. We also performed a complete laboratory investigation, including thyroid function tests, serum sodium, potassium, chloride, carbon dioxide, calcium, phosphate, magnesium, blood urea nitrogen, creatinine, osmolality, and urine for sodium, potassium, chloride, creatinine and osmolarity. Patients with suppression of TSH were tested for serum thyroglobulin. Figure 1 depicts the timeline of the study.

Laboratory investigation

In the first phase, prisoners were screened for TSH and potassium levels. Blood samples were collected at Buddhachinaraj Hospital and Wang Tong Hospital in Phitsanulok province, Thailand. TSH was measured using a chemiluminescent immunoassay.

A complete laboratory investigation, for each person, including blood tests, was collected and kept at −80°C. The urine electrolyte was collected at 50 mL. The samples were performed at King Chulalongkorn Memorial Hospital.

Serum and urine urea nitrogen, creatinine, sodium, potassium, chloride and carbon dioxide were measured using Alinity (Abbott Diagnostics, Chicago, Illinois, USA). Serum calcium, phosphate and magnesium were measured using Alinity (Abbott Diagnostics). Serum and urine osmolarities were measured using an Advanced Instruments Model 3250 single-sample osmometer (Fisher Scientific, Hampton, New Hampshire, USA). Thyroid function tests, including free triiodothyronine (T3), free thyroxine (T4) and TSH, were completed using Alinity I, which was a chemiluminescent microparticle immunoassay (Abbott Diagnostics). Serum thyroglobulin was measured using an electrochemiluminescence immunoassay using the Elecsys Tg II assay and Cobas e 801 analysers (Roche Diagnostics, Mannheim, Germany).

Global deiodinase activity

The sum activity of peripheral deiodinases (SPINA-GD) offers an assessment of the peak global activity of peripheral step-up deiodinases per unit of time (in nmol/s), termed as deiodinase activity. This activity is determined using equilibrium levels of FT3, FT4, as well as constants related to plasma protein binding, distribution and elimination.[11 12]

Definition

Overt hyperthyroidism/thyrotoxicosis is an excess of thyroid hormone (free T3 or free T4) and suppressed

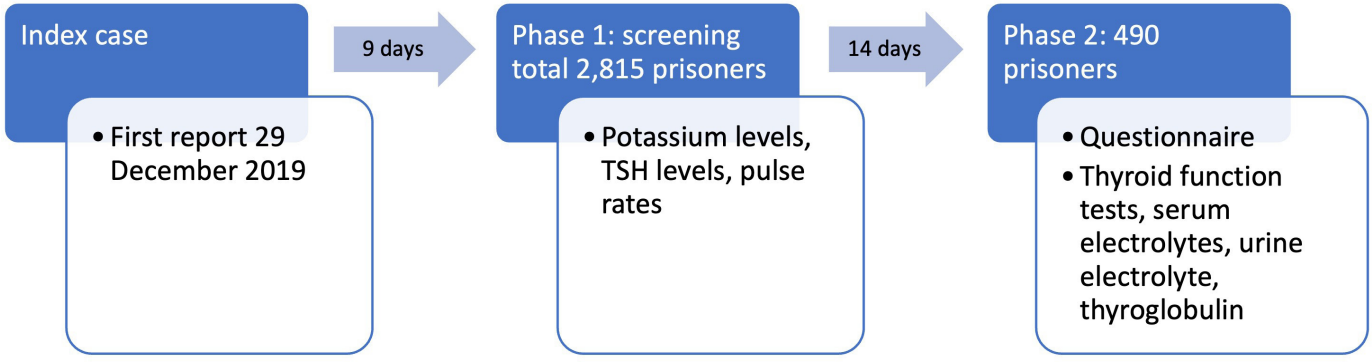

Figure 1 The study phase and protocol to investigate the outbreak of thyrotoxicosis 245×78 mm (300 × 300 DPI).

TSH. Subclinical hyperthyroidism/thyrotoxicosis is biochemically defined as a low level of TSH (equal to less than 0.35 uIU/ml) with normal levels of thyroid hormone. It is classified as either grade 1 subclinical hyperthyroidism/thyrotoxicosis (TSH 0.1–0.35 uIU/ml) and grade 2 subclinical hyperthyroidism/thyrotoxicosis (TSH<0.1 uIU/ml).[12 13]

A differentiation was established between hyperthyroidism and thyrotoxicosis. Hyperthyroidism results from an overactive thyroid gland and excessive endogenous thyroid hormone production, while thyrotoxicosis is a consequence of exogenous LT4 intake and drug overdose.

Renal potassium loss is defined as a transtubular potassium gradient (TTKG=(urine potassium × serum osmolarity)/(serum potassium × urine osmolarity)) greater than 4 or a urine potassium/urine creatinine ratio greater than 13 mEq/g creatinine.[14]

Statistical analysis

The clinical information and signs and symptoms of thyrotoxicosis were defined as categorical variables and described by counts and percentages. The proportions between the two groups were compared using the χ^2 test. The continuous variables were described by mean with SD, while comparing means between two groups using an independent t-test. Binary logistic regression was used to analyse the risk factors for thyrotoxicosis. The SPSS program V.22 was used for statistical analysis.

RESULTS

During the outbreak, 69 prisoners were admitted due to generalised weakness, tachycardia, myalgia and fatigue. Four deaths were reported.

Phase 1: screening for thyrotoxicosis in Phitsanulok

In the first phase, 2815 prisoners were screened. The prevalence of subclinical thyrotoxicosis was 78.1%. Most of the patients (71.6%) were classified as grade 2 subclinical thyrotoxicosis, and 184 patients (6.5%) were classified with grade 1 subclinical thyrotoxicosis. Pulse rates were significantly higher in the subclinical thyrotoxicosis group (91±16 beats/min vs 87±15 beats/min; p≤0.001). Serum potassium levels were significantly higher in the subclinical thyrotoxicosis group; however, the prevalence of hypokalaemia was not different between the subclinical thyrotoxicosis group and the normal TSH group (37.9% vs 40.1%, p=0.398) (table 1).

Phase 2: complete laboratory evaluation at King Chulalongkorn Memorial Hospital

Thyroid function status

Patients were divided into group 1: subclinical thyrotoxicosis (n=409, 83.4%) and group 2: normal TSH (n=81, 16.6%). Table 2 shows the patient characteristics, signs and symptoms of these two groups. We retrieved and evaluated clinical information on age, body mass index, underlying disease, social history, family history, symptoms of thyrotoxicosis and pulse rate measurements. In general, there were no differences in baseline clinical characteristics between the two groups except that the history of herbal use and numbness were significantly lower, and the mean pulse rate was significantly higher in the subclinical thyrotoxicosis group compared with the normal TSH group (mean pulse rate 81±15 beats/min vs 77±14 beats/min; p=0.026). Using logistic regression analysis, we found an association between an increase in pulse rate and an increase in the risk of subclinical thyrotoxicosis (OR 1.027; 95% CI 1.003 to 1.051; p=0.027).

Thyroid status and laboratory evaluation

Table 3 shows the laboratory evaluation of these two groups. Thyroid hormone levels were in the normal range in both groups; however, mean levels of free T3 and free T4 were significantly higher in the subclinical thyrotoxicosis group. The mean levels of sodium, potassium and calcium were not different between the groups, but the mean levels of phosphate were significantly higher and the mean levels of magnesium were significantly lower in the subclinical thyrotoxicosis group. Furthermore, there was no statistical significance in the prevalence difference of hyponatraemia, hypokalaemia, hypocalcaemia, hypophosphataemia, and hypomagnesaemia between the two groups (table 4). The prevalence of hypokalaemia in prisoners during the outbreak was 5.7% (28 of 490 patients). Only one patient was classified as having moderate hypokalaemia (potassium level 2.5–3.0 mmol/L), and the rest had mild hypokalaemia (potassium level 3.0–3.5 mmol/L).

Hypokalaemia

Among the 28 prisoners with hypokalaemia, 71.4% were also found to have hypomagnesaemia. In addition, 21 of 28 patients with hypokalaemia had hyponatraemia (75%) and 89.3% and 92.9% met the renal potassium loss criteria determined by urine

Table 1 Means (SD) of serum potassium levels, pulse rate and prevalence of hypokalaemia between the subclinical thyrotoxicosis group and the normal thyroid-stimulating hormones (TSH) group

	Subclinical thyrotoxicosis (n=2199)	Normal TSH (n=616)	P value
Potassium (mmol/L)	3.60 (0.32)	3.55 (0.30)	0.008
Pulse rate (beats/min)	91.19 (15.73)	86.52 (15.36)	<0.001
Hypokalaemia (K<3.5 mmol/L)	568 (37.9%)	167 (40.1%)	0.398

Table 2 Baseline characteristics and signs and symptoms of a prisoner between subclinical thyrotoxicosis and the normal TSH group

	Subclinical thyrotoxicosis (n=409) *	Normal TSH (n=81) *	P value
Age (years)	34.38 (11.04)	32.84 (9.75)	0.257
BMI (kg/m^2)	22.12 (3.24)	22.27 (3.04)	0.725
Underlying disease			
Hypertension	11 (2.7%)	3 (3.8%)	0.483
Diabetes mellitus	5 (1.2%)	N/A	1.000
Dyslipidaemia	3 (0.7%)	N/A	1.000
Ischaemic heart disease	1 (0.2%)	N/A	1.000
HIV infection	4 (1.0%)	N/A	1.000
Social history			
Smoking	403 (100%)	78 (100%)	Not computed
Alcohol consumption	98 (24.3%)	16 (20.5%)	0.469
Drug abuse	310 (76.9%)	63 (80.8%)	0.456
Dietary supplement	9 (2.2%)	3 (3.8%)	0.422
Herbal use	4 (1.0%)	4 (5.1%)	0.027
Monosodium glutamate	310 (76.9%)	63 (80.8%)	0.456
Family history			
Unknown cause of death	7 (1.7%)	N/A	0.605
Death before expected age (M<60 y, F<65 y)	74 (18.4%)	12 (15.4%)	0.53
Weakness	15 (3.7%)	3 (3.8%)	1.000
Palpitation	27 (6.7%)	6 (7.7%)	0.751
Faint	34 (8.4%)	12 (15.4%)	0.056
Chest pain	26 (6.5%)	6 (7.7%)	0.687
Symptoms			
Weight loss	41 (10.2%)	6 (7.7%)	0.499
Palpitations	53 (13.2%)	8 (10.3%)	0.482
Diarrhoea	3 (0.7%)	3 (3.8%)	0.057
Muscle weakness	20 (5.0%)	2 (2.6%)	0.554
Fatigue	12 (3.0%)	1 (1.3%)	0.703
Chest pain	10 (2.5%)	2 (2.6%)	1.000
Headache	7 (1.7%)	1 (1.3%)	1.000
Numbness	3 (0.7%)	4 (5.1%)	0.015
Myalgia	10 (2.5%)	2 (2.6%)	1.000
Physical examination			
Pulse rate (beats/min)	81.35 (14.64)	76.61 (14.35)	0.026

Data about age, BMI, and pulse rate were shown as means (SD). Other data were shown as counts (percentages).
*Not all data are complete.
BMI, body mass index; TSH, thyroid-stimulating hormones.

potassium/urine creatinine≥13 mEq K/g Cr and TTKG≥4, respectively.

Thyroglobulin level

To determine the aetiology of subclinical thyrotoxicosis, serum thyroglobulin levels were measured in 192 prisoners with a TSH level lower than 0.0083 uIU/ml. The median serum thyroglobulin level was 7.79 ng/mL (IQR: 8.97 ng/mL). More than 99% of the patients had low normal thyroglobulin levels (normal range 3.5–77 ng/mL).

Global deiodinase activity

The mean of total step-up deiodinase activity (SPINA-GD) in patients with subclinical thyrotoxicosis was 27.47±5.47 nmol/s (95% CI 26.95 to 28.01). Deiodinase activity less

Table 3 Mean (SD) of thyroid function test, serum electrolyte between subclinical thyrotoxicosis and normal TSH group

	Subclinical thyrotoxicosis (n=409)	Normal TSH (n=81)	P value	Normal range
Free T3 (pg/mL)	1.928 (0.482)	1.801 (0.297)	0.002	1.6–4.0
Free T4 (ng/dL)	0.785 (0.164)	0.703 (0.109)	<0.001	0.7–1.48
Sodium (mmol/L)	136.24 (9.51)	135.95 (10.00)	0.801	136–145
Potassium (mmol/L)	4.08 (0.37)	3.99 (0.39)	0.063	3.4–4.5
Calcium (mg/dL)	8.97 (1.06)	9.06 (1.17)	0.46	8.5–10.5
Phosphate (mg/dL)	3.44 (0.66)	3.26 (0.58)	0.028	2.3–4.7
Magnesium (mmol/L)	0.78 (0.10)	0.82 (0.11)	0.008	0.66–1.07

than 30 nmol/s in patients with suppressed TSH usually suggests exogenous thyrotoxicosis.[12]

Thyroid tissue in meat

The frozen meat from the prison during the outbreak of thyrotoxicosis was investigated and found to have higher levels of thyroxine (T4) compared with a control group (10.68±4.72 µg/g protein vs 5.82±2.79 µg/g protein; p=0.360).

DISCUSSION

During the first phase of screening with measurements of pulse rate, TSH level and potassium level in 2815 prisoners, we found that the prevalence of subclinical thyrotoxicosis was 78.1%. Most of the cases were classified as grade 2 subclinical thyrotoxicosis. The prevalence of hypokalaemia was 38.4% with no statistically significant difference between the two groups. The pulse rate was significantly higher in the subclinical thyrotoxicosis group.

In the second phase, we further investigated 490 prisoners with a questionnaire and a complete laboratory investigation 2 weeks after the first report. We found that the prevalence of subclinical thyrotoxicosis was 83.4%. All prisoners in the subclinical thyrotoxicosis group had a thyroid hormone level in the normal range. These results may be due to the sensitivity of the hypothalamic–pituitary–thyroid axis, which is more sensitive to TSH than to thyroid hormone.[15] Thus, the thyroid hormone level reached a normal level earlier than the TSH level.

Weight loss, palpitation, muscle weakness and fatigue were more frequent in the subclinical thyrotoxicosis group. Due to the limited time to approach the prisoners, only the pulse rate was measured. We found that the pulse rate was significantly higher in the subclinical thyrotoxicosis group.

Hypokalaemia is a common electrolyte imbalance found in patients with thyrotoxicosis caused by a thyroid hormone-induced intracellular shift that increases sodium/potassium ATPase activity in skeletal muscle, liver and kidney.[16] We found a 38.4% prevalence of hypokalaemia among 2815 prisoners in the first screening phase and only 5.7% of 490 prisoners in the second phase. There were no statistically significant differences between the subclinical thyrotoxicosis group and the normal TSH group in both the first and second phases of the study. It could be implied that hypokalaemia in prisoners may be affected by other factors aside from intracellular changes due to thyrotoxicosis. Most hypokalaemic cases had renal potassium loss and hypomagnesaemia. The assumption was that hypomagnesaemia reduces the inner correction of ROMK potassium channel secretion in the cortical collecting duct, increases outward conductivity and increases potassium secretion.[14] The aetiology of hypomagnesaemia could be attributed to reduced intake, malabsorption or loss through the gastrointestinal tract or kidney. However, there was no association between hypomagnesaemia and common aetiologies of hypomagnesaemia such as a history of alcohol consumption, diabetes mellitus or diarrhoea in our data.[17]

Table 4 Frequency of hyponatraemia, hypokalaemia, hypocalcaemia, hypophosphataemia, and hypomagnesaemia between subclinical thyrotoxicosis and the normal TSH group

	Subclinical thyrotoxicosis (n=409)	Normal TSH (n=81)	P value
Hyponatraemia (Na<135 mmol/L)	103 (25.2%)	26 (32.1%)	0.197
Hypokalaemia (K<3.5 mmol/L)	22 (5.4%)	6 (7.4%)	0.438
Hypocalcaemia (Ca<8.5 mg/dL)	83 (20.3%)	19 (23.5%)	0.522
Hypophosphataemia (p<2.3 mg/dL)	24 (5.9%)	2 (2.5%)	0.284
Hypomagnesaemia (Mg<0.7 mmol/L)	61 (14.9%)	10 (12.3%)	0.548

Table 5 Review of the literature on the outbreak of thyrotoxicosis caused by the consumption of thyroid tissue contamination in meat

Author, year	Place	Study	Cases, incidence	Investigation	Source of an outbreak
Kinney *et al*, 1984[1]	Nebraska, USA	Case-control study	49 cases, 2.4/1000 persons	▶ Low serum thyroglobulin ▶ Negative antimicrosomal antibodies, antithyroglobulin antibodies	Ground beef
Hedberg *et al*, 1985[2]	Minnesota and Iowa, USA	Case-control study	121 cases, 34/10 000 persons	▶ High concentrations of total iodine, T4, T3 in ground beef from gullet trimming	Ground beef
Matsubara *et al*, 1993[3]	Matsuyama, Japan	Cross-sectional study	159 cases, N/A	▶ Negative antimicrosomal antibodies, antithyroglobulin antibodies ▶ Low- to normal-range serum thyroglobulin ▶ No abnormal findings ultrasound of thyroid glands ▶ Poor uptake of thyroid scintigraphy was noted at the beginning of the study and returned to normal.	Unknown
Parmar and Sturge, 2001[4]	Canada	Case report	1 case, N/A	▶ Normal range erythrocyte sedimentation rate ▶ Negative antinuclear antibody (ANA), antithyroid antibody ▶ Normal range serum thyrotropin binding inhibitor immunoglobulin (TBII) ▶ Low serum thyroglobulin ▶ Poor uptake of radioactive iodine uptake (RAIU)	Beef
Conrey *et al*, 2003–2004[5]	Minas, Uruguay	Case-control study	59 cases, N/A	Case group: minced beef (adjusted OR 13.4; p 0.0136), chorizo (adjusted OR 1.88; p 0.0525)	Minced beef, chorizo
Megías *et al*, 2009[6]	Madrid, Spain	Case report	1 case, N/A	Central hypothyroidism (low-level free T4 and TSH) and turned to T3 toxicosis	Chopped pork
Wartique *et al*, 2017[7]	Brussel, Belgium	Case report	4 cases, N/A	▶ Negative thyroid autoantibodies ▶ Low serum thyroglobulin ▶ Poor uptake of radioactive iodine uptake (RAIU)	Beef neck

N/A, no available data about the incidence of thyrotoxicosis outbreaks.

We found that all the prisoners in the subclinical thyrotoxicosis group had a low normal thyroglobulin level (median 7.79 ng/mL, normal range 3.5–77 ng/mL level), suggesting that the aetiology of subclinical thyrotoxicosis was exogenous thyrotoxicosis rather than endogenous thyrotoxicosis. The presence of antithyroglobulin antibodies, however, could cause a falsely low thyroglobulin level. Due to the limitations approaching the prisoners, we were unable to perform radioactive iodine uptake (RAIU), thyroid ultrasound and thyroid tissue autoantibody measurements to confirm the diagnosis of exogenous thyrotoxicosis. Assessment of the biochemical heterogeneity of TSH and thyroid hormone by using calculated deiodinase activity revealed that the reduction of deiodinase activity favoured exogenous thyrotoxicosis. When the source of exogenous thyrotoxicosis was explored, we found a higher level of thyroid hormone in the frozen meat from prison during the outbreak compared with the control meat.

An outbreak of thyrotoxicosis caused by the consumption of contaminated thyroid tissue in meat was first reported in 1984 in Nebraska, USA.[1] Later, a few case reports and case-controlled studies were published (table 5). Investigations often found a low serum thyroglobulin level, negative test for antithyroglobulin antibodies, no abnormal findings on thyroid gland ultrasound, and poor thyroid uptake, which could be implied as exogenous thyrotoxicosis. Furthermore, there were no previous report on the outbreak of thyrotoxicosis in prisoners or an investigation in a patient who was not transferred into the hospital.

There are several limitations to this study. First, due to the outbreak of thyrotoxicosis found in a large population and many limiting factors including regulations

and policies in the prison, we unfortunately could not obtain data from thyroid scintigraphy, thyroid uptake measurement and thyroid ultrasound. With these limitations, we chose to screen the thyroid function of patients by measuring TSH levels. We proposed that TSH levels should be considered as a screening test for future outbreaks of thyrotoxicosis in limited resource or limited access settings. Second, we were unable to examine the presence of goitre and evaluate radioactive iodine uptake (RAIU). The diagnosis of exogenous thyrotoxicosis includes the absence of goitre, subnormal RAIU and a low serum thyroglobulin level.[15] However, we could imply exogenous thyrotoxicosis by suppressed TSH, lower than normal levels of serum thyroglobulin and reduced calculated deiodinase activity. Third, our study did not measure the antithyroglobulin level, which could interfere with the levels of thyroglobulin.[18] Fourth, we had limitations in collecting an adequate sample of the meat to show a significantly higher level of thyroid hormone. Fifth, this study did not obtain meal data for each group.

According to the available evidence, the aetiology of the thyrotoxicosis outbreak in prison was from contaminated thyroid tissue in the meat. To prevent a future outbreak of thyrotoxicosis, the government agency should import meat from a certified slaughterhouse for prisoners. The meat purchased in prison should be examined for sanitation, freshness and free of contamination. Second, a surveillance system for the outbreak of thyrotoxicosis should be reviewed and developed. Prison guards should be trained for early detection of diseases and notification to the healthcare provider. The prison healthcare system should be regularly revised to include strategies to manage outbreaks in prisons. Finally, prisoners should be concerned about adequate nutrition in their diet in prison. In addition, prisoners who have stayed in prison for a long time should have a health check-up annually.

CONCLUSIONS

The study observed a high frequency of subclinical thyrotoxicosis during the outbreak of thyrotoxicosis among inmates at a prison in Phitsanulok, Thailand. This problem also raises awareness of nutritional problems in prisons and the need for development of a surveillance systems to prevent future outbreaks.

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Patient consent for publication Not applicable.

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