Effectiveness and Safety of Radioactive Iodine Therapy in Childhood Graves' Disease in Khon Kaen, Thailand

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Background: *Graves' disease (GD) is the most common cause of hyperthyroidism in children and adolescents. Treatments consist of medication, radioactive iodine (RAI) therapy and surgery. Currently, radioactive iodine therapy is the first line treatment in many medical centers.*
Objective: *To evaluate the effectiveness and safety of RAI therapy in childhood GD.*
Material and Method: *A retrospective study was performed in 46 GD patients, aged at onset ≤15 years, who had undergone RAI therapy at the age ≥10 years. Goiter grading, evidence of hypothyroidism, severity of ophthalmopathy, RAI dosage and side effects of RAI therapy were evaluated.*
Results: *The cure rate was 95.6%. All participating patients had goiter reduction (p = 0.005). Hypothyroidism was induced in 33 (71.7%) and 11 (23.9%) patients after the first and second RAI therapy. The total RAI dosage was significantly higher in the patients with failure response (p = 0.001). The average time to induce hypothyroidism after the first RAI therapy was 127.5 (IQR: 94.5-223.0) days. All of the patients had improvement of ophthalmopathy and none had thyroid carcinoma during the follow-up period of 42.5 (IQR: 17-52) months.*
Conclusion: *Radioactive iodine therapy is effective and safe in the treatment of children and adolescents with Graves' disease.*

Keywords: *Graves' disease, Radioiodine therapy, Childhood, Adolescent*

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Graves' disease (GD) is the most common cause of hyperthyroidism in children and adolescents[1,2]. Current therapy of GD includes treatments with antithyroid drugs (ATDs), thyroidectomy and radioactive iodine (RAI) therapy. ATDs are commonly used as the first line therapy in childhood GD. Only 20% to 30% of pubertal cases and 15% of prepubertal cases had long-term remission with ATDs therapy[3,4]. Risk of minor and serious life threatening adverse side effects are associated with ATDs therapy, especially of propylthiouracil hepatotoxicity and antineutrophil cytoplasmic antibody (ANCA) induced vasculitis in various organs[1,5,6]. Children with methimazole therapy also develop both minor and major side effects, including Stevens-Johnson syndrome and vasculitis[7]. Surgery of a total or subtotal thyroidectomy was recommended in children younger than five years of age or those with large thyroid glands. Both hypothyroidism and relapse hyperthyroidism after surgery were reported[8,9]. Radioactive iodine was introduced for the treatment of GD in children in some institutes or medical schools in the United States and European countries[5,10-13]. The goal of RAI therapy for pediatric GD is to induce hypothyroidism[14,15]. Srinagarind Hospital, Khon Kaen University, is the sole university hospital in the northeast Thailand which provides RAI therapy for GD patients. Therefore, the present study was conducted to evaluate the efficacy and long-term follow-up safety of RAI therapy in childhood GD aged at onset ≤15 years and who received RAI at the age ≥10 years.

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Material and Method

A retrospective review of 46 medical records of childhood onset GD with RAI therapy from 1 January

2009 to 31 December 2014 was performed. Inclusion criteria were patients with the onset of GD at the age of ≤15 years who attended at the Pediatric Endocrinology Unit, Srinagarind Hospital, Khon Kaen University, Khon Kaen, Thailand. The study was approved by the Ethics Committee of the Faculty of Medicine, Khon Kaen University. The diagnosis of GD was based on the presence of goiter and/or ophthalmopathy combined with elevation of a total and/or free thyroxine (T4) and/or triiodothyronine (T3) concentrations, suppressed thyrotropin (TSH) levels, and evidence of thyroid autoantibodies. Palpations of goiters were performed using WHO criteria for goiter classification grading (Table 1)[16]. According to the European Group on Graves orbitopathy (EUGOGO) recommendations, Graves orbitopathy (GO) was classified into three groups based on the severity: mild, moderate to severe, and sight-threatening GO (Table 2)[17]. RAI therapy was performed on childhood patients with GD aged ≥10 years at the Division of Nuclear Radiology, Department of Radiology, Srinagarind Hospital, Faculty of Medicine, Khon Kaen University. The doses of RAI therapy were calculated using estimated thyroid weight in grams as assessed by a nuclear medicine radiologist (Table 3). First line RAI therapy was performed in GD patients, aged at onset ≥10 years with the parents' and patient's informed consents. Second line therapy was used in patients with failure medical treatment after six months or relapse. All patients receiving pre-treatment with ATDs, need to stop taking the medications three to five days before RAI treatment and were then given a beta-blocker. Acute side effects of RAI were followed-up by nuclear radiologists after the first two weeks of therapy. The thyroid function tests and clinical assessments of hyper/hypothyroidism, goiter grading and severity of GO were performed every two months and the patients were reviewed for treatment outcomes at the six months after the first RAI therapy. The second RAI therapy was introduced to the patients who had persisted hyperthyroidism for six months after the first RAI therapy.

Statistical analysis

SPSS and Microsoft Excel programs were used to analyze descriptive statistics, including average, percentage, median, mean and standard deviations (SD).

Results

Forty-six childhood onset GD patients,

Table 1. WHO classification of goiter

Grade	Characteristics of goiter
0	No goiter found, the thyroid impalpable and invisible.
1	An enlarged thyroid that is palpable, but not visible on inspection with the neck in a normal position. An enlarged thyroid is noticeable during swallowing. Grade 1 also includes a nodular goiter if the thyroid is not visible on inspection.
2	An enlarged thyroid is visible when the neck is in a normal position, corresponding to an enlarged thyroid found with palpation.

Adapted from WHO/UNICEF/ICCIDD, 1994[16]

Table 2. Severity classification in Graves orbitopathy (GO)

Classification	Graves orbitopathy (GO)
Sight-threatening GO	Patients with dysthyroid optic neuropathy (DON) and/or corneal breakdown.
Moderate to severe GO	Patients have any one or more of the following: lid retraction ≥2 mm, moderate to severe soft tissue involvement, exophthalmos ≥3 mm above normal for race and gender, inconstant, or constant diplopia.
Mild GO	Patients have only one or more of the following: minor lid retraction (<2 mm), mild soft tissue involvement, exophthalmos <3 mm above normal for race and gender, transient or no diplopia, and corneal exposure responsive to lubricants.

Adapted from Bartalena et al (2008)[17]

involving five (10.9%) males and 41 (89.1%) females, were included in the study. The average age at RAI therapy was 11.7±1.4 (mean ± SD) years. All patients had diffuse goiters, of which 11 (23.9%) patients had grade 1 goiters, and 35 (76.1%) had grade 2 goiters. The demographic data of the patients are shown in Table 4. RAI was the first line therapy in 17 (37.0%) patients and 29 (63.0%) patients received RAI as second line therapy. No patients developed thyroid storm or other minor acute side effects after receiving RAI. The duration of follow-up after RAI therapy was 42.5 (interquartile range, IQR: 17-52) months. Hypothyroidism was induced, after the first RAI therapy, in 33 (71.7%) patients, with a time to develop hypothyroidism of 127.5 (IQR: 94.5-223.0) days. Thirteen (28.3%) patients had persisted hyperthyroidism and required a second RAI therapy. Only two (4.4%) patients still had hyperthyroidism after the second RAI therapy (Table 5). Thyroxine replacement was prescribed in all patients with hypothyroidism, a low dose of 1.1 (IQR: 0.4-2.0) mcg/kg/day was successful to induce euthyroidism.

GO or exophthalmos were presented in 36 (78.3%) patients. Mild GO was found in 24 (52.2%) patients, and 12 (26.1%) patients with moderate to severe GO. After RAI therapy, GO persisted in 24 (52.2%) patients and only four (8.7%) patients had moderate to severe GO. No GO increased from 10 (21.7%) to 22 (47.8%) patients. All patients had reduction of goiters ($p = 0.005$). Thyroid gland sizes and GO before and after RAI therapy are shown in Table 6.

Table 7 shows the contributing factors that influenced acquired hypothyroidism or euthyroidism after treatment with single and more RAI therapy. There were no statistically significant differences ($p>0.05$) in age, sex, thyroid gland size, GO, pre-treatment antithyroid drug dose between patients who received single and more RAI therapy. A higher free T4 level (7.77 vs. 4.70 ng/dL) and a higher dose of RAI (10 vs.

Table 3. Raidioactive iodine (I-131) dosage according to estimated thyroid gland size*

Estimated thyroid gland (gm)	Radioactive iodine (I-131) dose (mCi)
<50	10
50-99	15
100-149	20
150-199	25
>200	30

* 80% of the patients received radioactive iodine dosage according to estimated thyroid gland size. The dose of 30 mCi of I-131, however, was used in 20% of the patients who had very high free T4 (≥5.0 ng/dL) level and hyperthyroid symptoms with enlarged thyroid glands, grade 2.

Table 4. Baseline characteristics of the participating Graves' disease children

Characteristics	n = 46
Age (mean ± SD) years	11.7±1.4
Gender (number, %)	
Males	5, 10.9
Females	41, 89.1
Height (mean ± SD) cm	148.3±11.3
Weight (mean ± SD) kg	40.7±12.4
Thyroid function test at first diagnosis	
Serum TSH (median) mU/L	0.010 (IQR: 0.005-0.110)
Serum T4 (mean ± SD) mcg/dL	20.69±5.72
Serum free T4 (median) ng/dL	6.02 (IQR: 4.14-7.77)
Choice of treatments	
First line therapy (number of patients, %)	17, 37.0
Second line therapy (number of patients, %)	29, 63.0
Antithyroid drug doses before starting radioactive iodine treatment	
PTU (median) mg/kg/day	5.1 (IQR: 2.5-6.4)
Methimazole (median) mg/kg/day	0.6 (IQR: 0.2-0.6)
Duration of antithyroid-drugs treatment before starting radioactive iodine therapy (median) days	455 (IQR:261-778)

PTU = propylthiouracil; IQR = interquartile range

Table 5. Outcomes of radioactive iodine therapy

Outcomes	n = 46
First RAI dosage (median) mCi	12 (IQR: 9-15)
Time to hypothyroidism or euthyroidism after the first RAI therapy (median) days	127.5 (IQR: 94.5-223.0)
Number of hypothyroid patients after the first RAI therapy (numbers of patients, %)	33, 71.7
Number of GD patients who required the second RAI therapy (numbers of patients, %)	13, 28.3
Number of GD patients with hyperthyroidism after the second RAI therapy (numbers of patients, %)	2, 4.4
Duration of follow-up after RAI therapy (median) months	42.5 (IQR: 17-52)
L-thyroxine dose to attain and keep euthyroidism in acquired hypothyroidism (median) -mcg/kg/day	1.1 (IQR: 0.4-2.0)

IQR = interquartile range

Table 6. Thyroid gland size and ophthalmopathy of the participating children with Graves' disease before and after RAI therapy

	Before RAI therapy No. of patients (%)	After RAI therapy No. of patients (%)	*p*-value*
Goiter (n = 46)			0.005
Grade 0	0	23 (50.0)	
Grade 1	11 (23.9)	22 (47.8)	
Grade 2	35 (76.1)	1 (2.2)	
Graves ophthalmopathy (GO) (n = 46)			>0.05
No GO	10 (21.7)	22 (47.8)	
Mild GO	24 (52.2)	20 (43.5)	
Moderate to severe GO	12 (26.1)	4 (8.7)	
Sight-threatening GO	0	0	

* Using McNemar's Chi-Square test, *p*-value <0.05 = statistically significant differences.

23 mCi, $p = 0.001$) were observed in those who required more than single RAI treatment.

No late effects of I-131 such as thyroid carcinoma and gonadal and hematological complications were observed. All patients had normal growth and puberty but no conceptions were reported.

Discussion

The prevalence of Graves' disease (GD) in children is much lower than in adults. In Srinagarind Hospital, the prevalence of GD in adults is about ten times that of childhood GD (598 vs. 59 subjects) during the same period of study time. Radioactive iodine (RAI) therapy for GD patients is limited in some university hospitals. Almost all Thai GD patients are treated with antithyroid medications and there is no clinical practice guidelines (CPG) for management of childhood GD in Thailand. Previously, RAI therapy for GD was concerned with the possibility of secondary malignancy. No thyroid cancer or leukemia, however, was detected after 36 years of follow-up in childhood RAI therapy of GD[12] in the United States. In Great Britain, a survey of RAI usage in the young GD patients showed a percentage of the total increase from 0.2% in 1990 to 1.5% in 2008 and the youngest age receiving RAI treatment fell from 19 to 11 years[18]. RAI should not be given to children under five years of age but it is acceptable in children between five and ten years of age depending on the dose. In children with GD who have high free T4 estimates >5 ng/dL and, high TSH receptor antibody levels, surgery is contraindicated. In addition, when it is not possible to report side effects of ATDs, RAI therapy should be considered[15]. To prevent short-term worsening of hyperthyroidism or thyroid storm, pre-treating with ATDs and beta-blockers until T4 normalizes before proceeding with RAI is recommended[19]. Fewer than 10% of children complained of mild tenderness over the thyroid in the

Table 7. Comparisons between subjects with treatment success and failure after the first RAI therapy*

Parameters	Acquired hypothyroidism or euthyroidism after the first RAI therapy (n = 33)	GD patients required the second RAI therapy (n = 13)	*p*-value
Age (mean ± SD) years	11.8±1.3	11.3±1.7	>0.05**
Gender			>0.05***
Males (number of patients, %)	5, 10.9	0	
Females (number of patients, %)	28, 60.8	13, 28.3	
Total (number of patients, %)	33, 71.7	13, 28.3	
Time before starting radioactive iodine therapy from diagnosis (median) days	392 (IQR: 133-737)	545.5 (IQR: 364.5-1084)	>0.05****
Total RAI dosage (median) mCi	10 (IQR: 8-12)	23 (IQR:13-25)	0.001***
Gland size grading (median)	2	2	>0.05***
Ophthalmopathy grading (median)	1 (mild GO)	1 (mild GO)	>0.05***
Thyroid function tests at first diagnosis			
Serum TSH (median) mU/L	0.006 (IQR: 0.005-0.050)	0.021 (IQR: 0.002-0.110)	>0.05****
Serum free T4 (median) ng/dL	4.70	7.77	>0.05****
Choice of treatments			>0.05****
First line therapy (number of patients, %)	15 (45.5%)	2 (15.4%)	
Second line therapy (number of patients, %)	18 (54.6%)	11 (84.6%)	
Antithyroid drug doses before starting radioactive iodine treatment PTU (median) mg/kg/day	5.0 (IQR: 2.2-6.7)	5.2 (IQR: 4.9-5.5)	>0.05****

* Treatment success = the cure of hyperthyroidism with acquired hypothyroidism or euthyroidism after the first RAI therapy, treatment failure = RAI therapy more than once to induce acquired hypothyroidism or euthyroidism
** Using independent t-test
*** Using Chi-squared test
**** Using Mann-Whitney U test
PTU = Propylthiouracil, IQR = Interquartile range
p-value <0.05 = statistically significant differences

first week after RAI therapy[20]. Other acute side effects of RAI therapy such as redness or swelling in the neck region, ageusia, sialadenitis, and radiation-induced thyroiditis have been reported in a small number of children[21,22] but there were none in the present study. After RAI treatment, there is a theoretical risk in young children to develop thyroid cancer when there is residual thyroid tissue. Nearly all patients in the present study developed hypothyroidism and successfully kept euthyroidism with low doses of thyroxine replacement. I-131 doses used to achieve hypothyroidism was based on a gland size. In some centers, administration of a fixed dose of about 150 μCi/g of I-131 to all children yielded good outcomes[23,24]. There is an evidence showing 95% of patients achieved hypothyroidism with I -131 doses of 220 to 275 μCi/g[20] but the outcome is still imprecise due to individual variations in the sensitivity of the thyroid to radioiodine. In the present study, I-131 doses were calculated and used based on estimated gland size and a fixed dose of 30 mCi was used for the subjects with severe hyperthyroidism and high free T4 levels. Thyroid gland size using WHO grading classifications of goiters was significantly reduced in the present study. Ultrasonography is considered the most reliable method to determine the accurate thyroid volume or gland size in many studies[24,25]. In this present study, 95.6% of the subjects ended up hypothyroidism, of which 71.7% were after the first and 23.9% were after the second RAI treatment. Progression to euthyroidism or hypothyroidism after RAI therapy varied from 60.0-90.0% in some studies[21,22]. In a recently published meta-analysis of 1,874 patients in 29 trials, the overall cure rate using RAI reached 49.7%[26]. Factors possibly contributing to post-RAI hypothyroidism are: dosage of I-131, age, gender, size of the gland, initial serum free T4, free T3, TSH levels, circulating levels of TSH receptor antibodies (TRAb), radioiodine uptake, and duration of disease[20,27,28]. TRAb were not verified in this present study, the necessary technology has been

available only in the last couple of years at Khon Kaen University Hospital. The free T4 level was clinically higher and the total RAI dosage was higher statistically between success and failure GD patients after the first RAI therapy in this present study. The failure group required fixed high dosage due to severe hyperthyroidism with very high Free T4. A previous study in adults suggests that the use of RAI as second line therapy or pre-treatment with antithyroid drugs (PTU) leads to a higher failure rate[29]. A recent study of pediatric Graves' disease showed that prior carbimazole treatment does not alter the outcomes of RAI therapy[30]. Multivariate analyses showed no statistical significance of single contributing factor for the development of hypothyroidism in the present study. The weak point is due to a small sample size.

In female adolescents, reduction of goiters and GO are important factors for cosmetic reasons. About half of the subjects had no goiter and GO after six months of RAI therapy in the present study. GO is an inflammatory eye disease associated with autoimmune thyroid disorders. Radioiodine therapy is one of the risk factors for GO in adults[31]. Other identified risk factors included smoking, high pre-treatment of T3 values, high serum pre-treatment TRAb levels and hypothyroidism following RAI treatment[32]. Smoking is the most important known risk factor for worsening of GO in adults. No smoking subjects and no aggravation of GO after RAI therapy were found in this present study. Progression to puberty as determined by Tanner stage V was adequate after RAI therapy. All patients had improvement of GO and none had thyroid carcinoma during the follow-up period of 42.5 (IQR: 17-52) months. The follow-up period should be continued as long as possible to detect long-term sequelae of RAI therapy in childhood GD.

Conclusion

Radioactive iodine therapy is effective and safe in the treatment of children and adolescent Graves' disease.

What is already known in this topic?

The effectiveness of RAI therapy in GD and long-term outcomes were well-known in adults.

What this study adds?

RAI therapy is suitable as the first line therapy in children with GD. High RAI dosage should be used in patients with severe hyperthyroidism. Graves ophthalmopathy is likely to improve when compared with adults.

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Potential conflicts of interests

None.

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ประสิทธิผลและความปลอดภัยของการรักษาผู้ป่วยเด็ก Graves' disease ด้วยไอโอดีนกัมมันตรังสีในจังหวัดขอนแก่น

ทรงพล เกษสุวรรณ, ภัทร วิริมย์รัตน์, อวยพร ปะนะมณฑา, มนัส ปะนะมณฑา, สรรพัชญ์ ไพรีพินาศ

ภูมิหลัง: *Graves' disease เป็นโรคที่พบได้บ่อยที่สุดของภาวะไทรอยด์เป็นพิษในวัยเด็ก วิธีการรักษาประกอบไปด้วยการใช้ยา การรักษาด้วยไอโอดีนกัมมันตรังสีและการผ่าตัด ในปัจจุบันการใช้ไอโอดีนกัมมันตรังสีถือเป็นการรักษาอันดับแรกในบางศูนย์การแพทย์*

วัตถุประสงค์: *ประเมินประสิทธิผลของการรักษาและความปลอดภัยของผู้ป่วยเด็ก Graves' disease ที่ได้รับการรักษาด้วยไอโอดีนกัมมันตรังสี*

วัสดุและวิธีการ: *ศึกษาแบบย้อนหลังจากเวชระเบียนผู้ป่วย Graves' disease 46 รายที่ได้รับการวินิจฉัยที่อายุ ≤15 ปี และได้รับการรักษาด้วยไอโอดีนกัมมันตรังสีที่อายุ ≥10 ปี ข้อมูลที่บันทึกและประเมินได้แก่ ขนาดของคอพอก ความรุนแรงของตาโปน ขนาดของไอโอดีนกัมมันตรังสี และผลข้างเคียงของการรักษา*

ผลการศึกษา: *หลังการรับไอโอดีนกัมมันตรังสีผู้ป่วยทุกคนมีคอพอกขนาดเล็กลง (p = 0.005) พบภาวะพร่องไทรอยด์ฮอร์โมน 44 (95.6%) ราย 33 (71.7%) ราย หลังได้รับ 1 ครั้ง 11 (23.9%) ราย หลังได้รับ 2 ครั้ง และขนาดของไอโอดีนกัมมันตรังสีที่ใช้สูงกว่า (p = 0.001) ในผู้ป่วยที่ไม่ตอบสนองต่อการรักษาครั้งแรก ระยะเวลาหลังได้รับไอโอดีนกัมมันตรังสีครั้งแรกจนเกิดภาวะพร่องไทรอยด์ฮอร์โมนเท่ากับ 127.5 (IQR: 94.5-223) วัน หลังการรักษาผู้ป่วยทุกคนมีตาโปนดีขึ้น จากการติดตามผู้ป่วยเป็นระยะเวลานาน 42.5 (IQR: 17-52) เดือน ไม่พบการเกิดมะเร็งของต่อมไทรอยด์*

สรุป: *การใช้ไอโอดีนกัมมันตรังสีในการรักษาผู้ป่วยเด็ก Graves' disease ได้ผลดีและปลอดภัย*