

A 7 years old girl with abdominal lump and per-vaginal bleeding - A rare presentation of Hypothyroidism.

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Abstract

Hypothyroidism is a common endocrine disorder resulting from decreased secretion of thyroid hormone. The diagnosis of hypothyroidism is suggested from the clinical and laboratory findings. Its occurrence in childhood is rare. Here we present a case report on this disease with rare presentation of abdominal lump and pervaginal bleeding in childhood. A 7 years old girl admitted in tertiary level hospital with abdominal lump and irregular per vaginal bleeding. Abdominal swelling was gradually increasing in size & associated with lower abdominal pain for last 5 months. On examination the girl was moderately anaemic, mildly oedematous, distended lower abdomen and a mass in left iliac region. Her thyroid function test, serum FSH, serum LH, serum Prolactin was done and high FSH, LH, Prolactin levels were found. USG of lower abdomen revealed bulky uterus and bilateral ovarian cysts. MRI of Brain showed feature of pituitary microadenoma. Finally the patient was diagnosed as **primary hypothyroidism** and bilateral follicular ovarian cyst with pituitary adenoma developed as its consequence. The case is reported for clinical awareness & to share our experience.

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Introduction

Hypothyroidism is a common endocrine disorder resulting from decreased secretion of thyroid hormone and female to male ratio is 6:1. Causes are autoimmune, iatrogenic, congenital, secondary. Thyroid gland produces predominantly thyroxine (T4) & a small amount of triiodothyronine (T3). Production of T3 & T4 in the thyroid is stimulated by thyrotropin (thyroid stimulating hormone-TSH) a glycoprotein released from the thyrotroph cells of anterior pituitary in response to the hypothalamic tripeptide, thyrotrophin releasing hormone. In hypothyroidism due to disease of the thyroid gland, low T3 & T4 are associated with high circulatory TSH level. The anterior pituitary is very sensitive to minor changes in thyroid hormone levels within the normal range. TSH is usually regarded as the most useful investigation of thyroid function. In primary hypothyroidism, TSH level rises but in secondary hypothyroidism TSH level is normal or low. TSH producing pituitary tumour is a rare condition. Administration of TRH results in elevation of TSH in thyroid hormone resistance and not in TSH-oma. But an MRI scan of pituitary may be necessary to exclude macroadenoma^{1,2}.

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Hyperprolactinaemia is a common biochemical abnormality, the cardinal features are galactorrhoea and hypogonadism. Pituitary tumours can cause hyperprolactinaemia not only by their ability to secrete prolactin but also if they are large enough to compress the infundibular stalk & thus interrupt the tonic inhibitory effect of hypothalamic dopamine or prolactin secretion ("disconnection" hyperprolactinoma).

Upper normal limit of serum prolactin-500mu/l (14 ng/ml). Level between 1000-5000 mu/l likely to be either drugs or microprolactinoma or due to "disconnection" hyperprolactinoma. Level more than 5000mu/l highly suggestive of macroprolactinoma³.

Ovarian cysts are a common cause for gynecological surgery. Some cysts only result from reproductive endocrine dysfunction and may resolve without surgery after endocrine correction⁴.

Possible explanation of follicular cysts of ovaries-

- Ovulatory dysfunction due to altered estrogen metabolism resulting formation of cyst.
- Hypothalamic Pituitary Ovarian (H.P.O.) axis dysfunction has impact on ovaries.
- TSH, FSH, LH all shares common alfa chain that confer specificity.
- Cross reaction of TSH produce FSH & LH like activity which is responsible for luteinization or as follicular cyst.
- Bilateral ovarian cysts may develop in long standing untreated primary hypothyroidism due to elevation of FSH⁵.

Case report

A 7 years old girl hailing from, Trishal, Mymensingh from below average socioeconomic condition admitted into the department of Gynaecology, Mymensingh Medical College Hospital, Bangladesh with the complaints of irregular per vaginal bleeding for 2 years, her mother stated that amount of bleeding was variable. She noticed that blood

loss was gradually increasing both in amount & duration. Gradually she developed pallor & generalized weakness. She had a swelling in lower abdomen which was gradually increasing in size & was associated with lower abdominal pain for last 5 months and was dull in character. There was no associated bladder or bowel complaints, no history of headache, fever, vomiting, and dimness of vision or convulsion. She did not give any history of gum bleeding, blood vomiting or passage of black stool per rectum or any skin rash.

General examination revealed the girl was ill looking, body built below average, moderately anaemic, BMI- 13.44, mildly oedematous, pulse- 94/min, regular. BP-90/60 mm of Hg, respiratory rate-16 breath/min, temp.- 98.4°F, thyroid gland not enlarged, no abnormal pigmentation on skin.

Per abdominal examination revealed distended lower abdomen, flanks not full, umbilicus centrally placed & slightly everted. On palpation a mass in left iliac region, size about (8x5cm) Surface – Irregular, margin - Ill defined, consistency – firm, restricted movement, non tender. Lump was dull on percussion.

Per-Vaginal examination average amount of bleeding was present on inspection.

Per rectal examination a large cystic mass felt in pouch of Douglas. **Examination of Eye** revealed—within normal limit but fundoscopy showed- mild bilateral disc swelling.

Investigation revealed, All routine biochemical parameter including coagulation profile found normal except Hb%-40%, ESR – 40 mm in 1st hour,

Chest X-Ray P/A view revealed—Normal

Study

Thyroid function tests:

Serum T3 - 0.29 nmol/ L (0.8-3.16nmol/L)

Serum T4 - 13.21 nmol/L (52-173nmol/L)

Serum TSH - >90 mIU/L (0.3-5 mIU/L)
 Serum estradiol—104.06 pg/ml (31-417 pg/ml)
 Serum FSH—22 IU/L (<1.16 IU/L)
 Serum LH - 9.92 IU/L (0.56-1.91 IU/L)
 Serum Prolactin - 1497.96 mIU/L (<460 mIU/L)
 Serum Cortisol— 263.60 ng/ml (147-251 ng/ml)
 Basal Human GH - 0.55 ng/ml (0.05-5 ng/ml)
 Serum ACTH - 10 pg/ml (8.3-57.8 pg/ml)
 Serum Calcium - 9.4 mg/dl (8.1-10.4 mg/dl)
 X-Ray Skull L/V - revealed no abnormality

Thyroid Scan on Scan with 99m Technetium - shows normal size thyroid gland with homogenous radiotracer concentration.

USG of lower abdomen revealed Uterus bulky in size (8.5x3.5 cm). Uterine tissue texture is normal. Cavity is empty. There is a multiloculated cystic mass (5x3.5 cm) in right adnexa. Another cystic mass (8x5 cm) with septation seen in left adnexa.

CECT of the Lower Abdomen-There is an isodense mass (3.1 x 2.8 cm) with well defined capsule in right adnexal region. There is another similar type of mass (7.5 x 5 cm) in left adnexal region.

The masses show mild homogenous enhancement after administration of water soluble i/v contrast medium.

CT Scan of Brain (Contrast) -There is well defined rounded enhancing mass in pituitary fossa (9.8 x 9.4 mm). Rests of brain parenchyma normal. Ventricles are not dilated. Midline structures are not shifted. Posterior fossa structure are normal. Skull bones revealed no abnormality. **Comment-Pituitary microadenoma.**

MRI of Brain- Pituitary fossa is enlarged. Pituitary gland is enlarged (8.8x8.6 mm). The lesion is iso to hypo intense in T1W1 image & hyper intense in T2W1 image. Pineal gland including stalk seems to be normal.

Comment- Pituitary microadenoma. Finally the patient was diagnosed as **Primary Hypothyroidism.**



Fig-1: Physical appearance of this patient

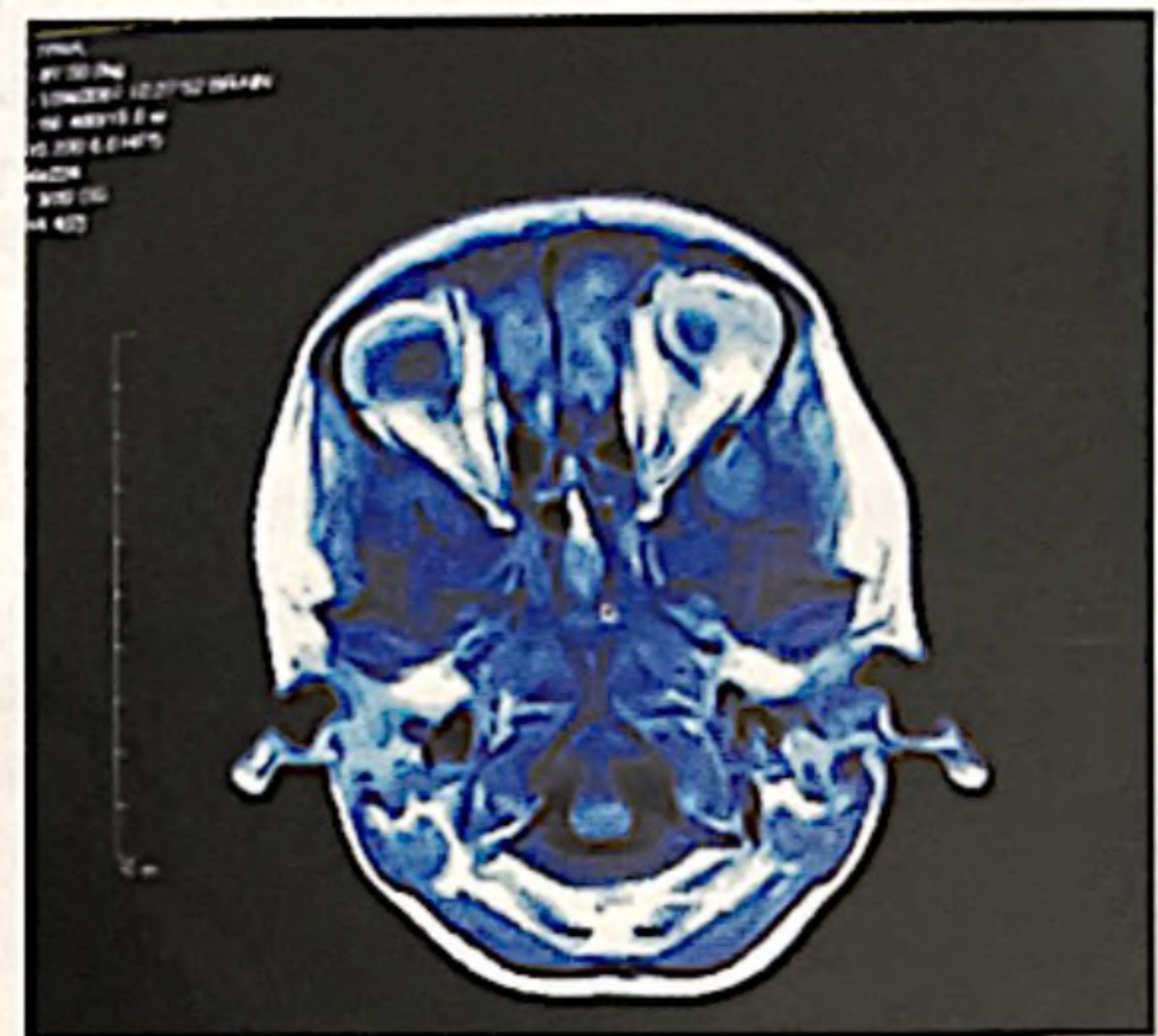


Fig-2: MRI of brain in axial view of T2 image showed enlarged pituitary gland (8.8x8.6 mm).

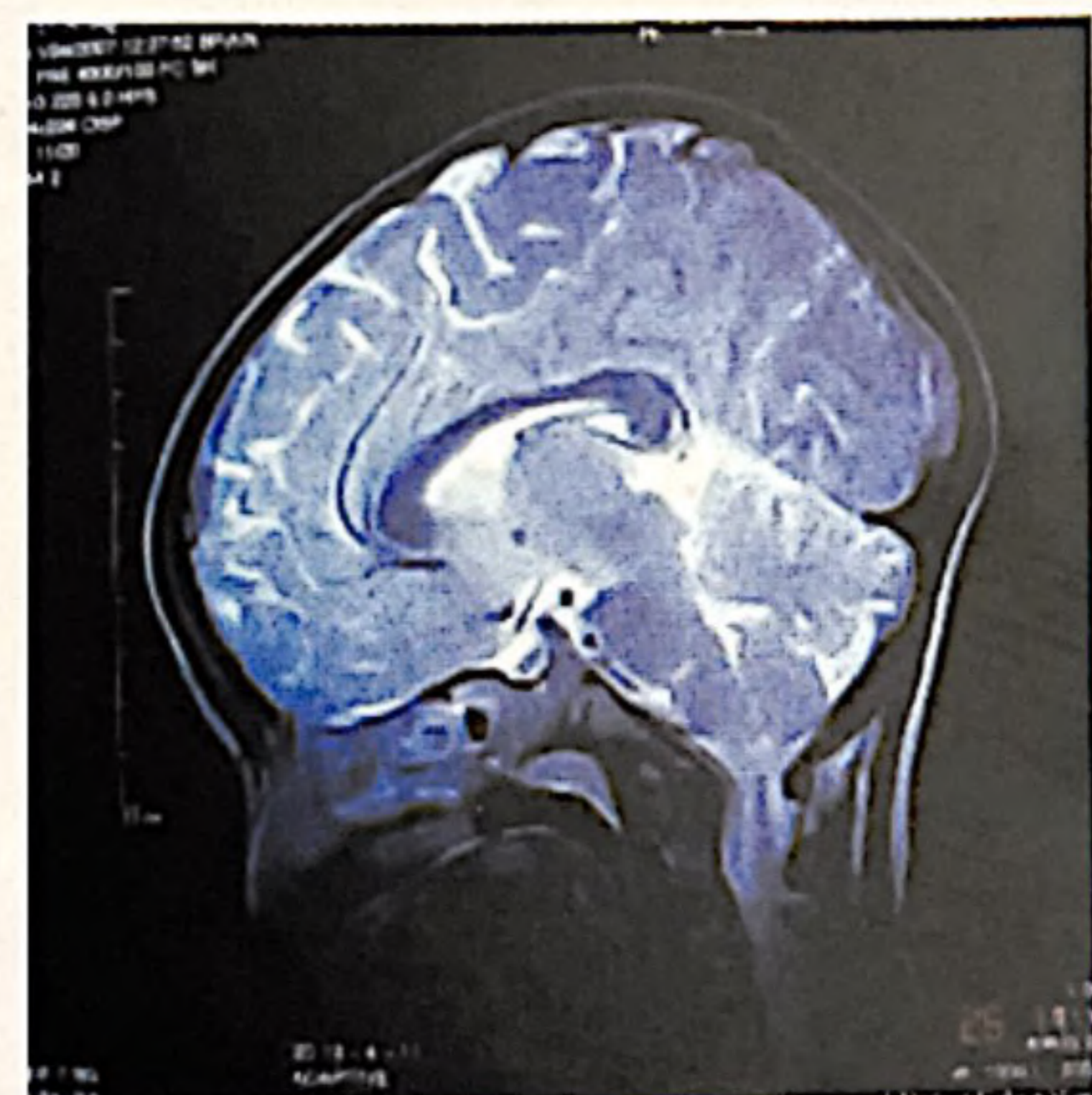


Fig-3: MRI of brain in sagittal view of T2 image showed enlarged pituitary gland (8.8x8.6 mm).

Thyroid replacement therapy was started with Thyroxine 100 microgram and patient was followed up time to time and showed dramatic response. After 3 month, her symptoms subside and follow up ultrasonography revealed complete resolution of Ovarian cysts. & MRI scan revealed regression of size of pituitary tumour.

Discussion

Ovarian cysts are a common cause for gynecological surgery. Owing to the complexity of ovarian composition and function, the etiology of ovarian cysts can vary greatly, including benign or malignant tumors, endometriosis and inflammation, etc. Some cysts only result from reproductive endocrine dysfunction and may resolve without surgery after endocrine correction. Ovarian cysts may be demonstrated on ultrasonography due to elevation of follicle stimulating hormone (FSH)⁶. Ovarian hyperstimulation is such a condition. With multiple follicles developing, the ultrasound images of the ovaries appear similar to multilocular cystadenoma with multiple septa. Usually, ovarian hyperstimulation syndrome (OHSS) is caused by iatrogenic superovulation. Excessive exogenous FSH stimulates multiple follicular growth simultaneously^{7,8}. Ectopic gonadotrophin adenoma secreting FSH can also present with multiple follicular cysts in ovaries^{9,10}. Without considering these endocrine disorders as a possible etiology, clinicians are likely to assume a diagnosis of neoplasm, leading to unnecessary ovarian surgery. Other authors have also described improvement of symptoms in such patients, with normalization of thyroid function tests and resolution of ovarian cysts, after treatment for hypothyroidism^{11,12}. Thyroid replacement therapy shows dramatic response with complete resolution of the ovarian cysts and clinical symptoms. No need of surgical intervention for this type of cysts as has been documented by other authors^{13,14}.

In severe primary hypothyroidism of long duration, the sella turcica may be enlarged due to thyrotrope hyperplasia.

References

1. s D.K., Dewhurst Textbook of Obstetrics and Gynecology, 7th edition, Blackwell Publishing, OXFORD university perss.pp.377-398.
2. Decherney A. H., Nathan L., Goodwin T.M., Laufer N., Current Diagnosis & Treatment Obstetrics & Gynaecology, 10th Edition, 2003, McGraw-Hill Medical Publishing Division, pp.654-661.
3. Stranchan M.W.J., Walker B.R., Davidson's Principles & Practice of Medicine, 20th edition, 2007, Elsevier, pp792-794.
4. Shu J., Xing L., Zhang L., Fang S., Huang H., Ignored adult primary hypothyroidism presenting chiefly with persistent ovarian cysts: a need for increased awareness, *Reproductive Biology and Endocrinology* 2011 V- 9 pp.119-121.
5. Mittal A., Mehta V., Mahendru R., Ovarian disease secondary to hypothyroidism in a prepubertal girl, *Ann Trop Med Public Health*, 2011 V-4(2) pp. 107-109.
6. Brook C.G.D., Dattani M.T. Handbook of clinical pediatric endocrinology, 2nd edition, John Wiley & Sons Ltd, The atrium, Southern gate, West Sussex, PO198SQ, UK, pp-137.
7. Vasseur C., Rodien P., Beau I., Desroches A., Gerard C., et al.: A chorionic gonadotropin-sensitive mutation in the follicle-stimulating hormone receptor as a cause of familial gestational spontaneous ovarian hyperstimulation syndrome. *N Engl J Med* 2003, 349(8) pp.753-759. INCLUDEPICTURE "http://www.rbej.com/sfx_links?getImage" * MERGEFORMATINET
8. Montanelli L., Delbaere A., Di Carlo C., Nappi C., Smits G., et al: A mutation in the follicle-stimulating hormone receptor as a cause of familial spontaneous ovarian hyperstimulation syndrome. *J Clin Endocrinol Metab* 2004, 89(3) pp.1255-1258. INCLUDEPICTURE "http://www.rbej.com/sfx_links?getImage" * MERGEFORMATINET
9. Shimon I., Rubinek T., Bar-Hava I., Nass D., Hadani M., et al: Ovarian hyperstimulation without elevated serum estradiol associated with pure follicle-stimulating hormone-secreting pituitary adenoma. *J Clin Endocrinol Metab* 2001, 86(8) pp. 3635-3640. INCLUDEPICTURE "http://www.rbej.com/sfx_links?getImage" * MERGEFORMATINET
10. Burgos J., Cobos P., Vidaurrazaga N., Prieto B., Ocerin I., et al : Ovarian hyperstimulation secondary to ectopic secretion of follicle-stimulating hormone. Literature review prompted by a case. *Fertil Steril* 2009, 92(3) pp.1165-1168. INCLUDEPICTURE "http://www.rbej.com/sfx_links?getImage" * MERGEFORMATINET
11. Tho SP., Hanly M., Moretuzzo RW., McDonough PG., Massive ovarian enlargement in primary hypothyroidism. *Fertil Steril* 1997(67) pp.169-71.
12. Yamashita Y., Kawamura T., Fuzikawa R., Mochizuki H., Okubo M., Regression of both pituitary and ovarian cysts after administration of thyroid hormone in a case of primary hypothyroidism. *Intern Med* 2001(40) pp.751-5.
13. Riddlesberger MM Jr., Kuhn JP., Munschauer RW., The association of juvenile hypothyroidism and cystic ovaries. *Radiology* 1981(139) pp.77-80.
14. Ajlouni K., A case of ovarian enlargement in severe primary hypothyroidism and review of literature. *Ann Saudi Med* 2006(26) pp.66-8.