Document heading doi: 10.21276/apjhs.2018.5.4.21 **Case report**

Anaesthesia for emergency exploratory Laparotomy in a Patient with undiagnosed Thyrotoxicosis: A Case Report

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Abstract

OF, a 25-year-old female with a history of 3 previous unsuccessful pregnancies was referred from a peripheral hospital with a diagnosis of ectopic gestation. She had a thyroid swelling. Initial pulse rate and blood pressure were 168 beats per minute and 130/90 mmHg respectively. She was premedicated with oral atenolol 100 mg on suspicion of undiagnosed thyrotoxicosis and pulse rate and blood pressure dropped to 110 beats per minute and 100/60 mmHg respectively. A right salpingectomy was carried out under endotracheal anaesthesia with muscle relaxation.A thyroid function test done postoperatively revealed a raised T3 and T4 with reduced thyroid stimulating hormone (TSH). She had a successful recovery and was discharged home. Issues and anaesthetic management challenges specific to patients with undiagnosed thyrotoxicosis coming for emergency surgery are discussed.

Keywords: Anaesthesia, emergency, laparotomy, thyrotoxicosis, ectopic-pregnancy.

Introduction

Ectopic pregnancy is defined as implantation in any site other than the decidua of the uterus and occurs in about 1-2%of pregnancies globally.[1] Ninety eight percent of ectopic pregnancies occur in the fallopian tubes.[2] Ampullary end, isthmus and interstitial part in that order are the leading sites for ectopic implantation. Very rarely, the fertilized ovum embeds in the ovary (primary ovarian pregnancy) or in the peritoneal cavity (e.g. on the broad ligament or omentum), giving rise to the abdominal pregnancy. Cases of repeated tubal pregnancy are common and account for about 7 percent of all ectopic pregnancies but the majority of women who have had an ectopic pregnancy will have a normal intrauterine pregnancy the next time they conceive.[3] Coexisting intra and extra uterine pregnancy (heterotopic pregnancy) are not unknown.[4]

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Multinodular goitre is one of the most common problems of the thyroid gland, especially among women aged 30 to 50.[5-7] It is seven times more common in women than in men (7:1).[7]Five percent of women have thyrotoxic effects 3 – 6 months postpartum and tend to have recurrences with subsequent pregnancies.[8]About 30-60% of women with hyperemesis gravidarum have gestational hyperthyroidism.[7,8] This condition is characterized by elevated free thyroid (FT_4) values, suppressed TSH levels, minimal thyroid enlargement, variable evidence of clinical hyperthyroidism, and absent thyroid antibodies[9] Gestational hyperthyroidism is due to the thyroid-stimulation effects of human chorionic gonadotropin (HCG) and is most likely to arise in the setting of elevated HCG concentrations (eg, molar or multiple pregnancies). Treatment is usually not needed because spontaneous recovery occurs after the first trimester.[5]Thyrotoxicosis is characterized by excess circulating thyroid hormones (T_4 and or T_3) either as a result of hyperfunction of the thyroid or exposure to exogenous thyroid hormones.[10] Thyroid function tests are not routine laboratory tests; they are only carried out when there is a need, and more often in non-urgent circumstances.[6,10]

The diagnosis of ectopic pregnancy in a thyrotoxic patient sometimes needs a high index of suspicion.[2]

This case therefore highlights the need for vigilance in pre-anaesthetic review of patients, and for consideration of a differential diagnosis of thyrotoxicosis in patients with unduly high heart rates, even when the high heart rate may be explained by the clinical illness[10]

Case Presentation

OF, a 25-year-old P_3^{+2} female with thyroid swelling presented at University of Port Harcourt Teaching Hospital (UPTH) with complaints of bleeding per vaginam, severe abdominal pain as well as dizzinessof one week duration. The onset of patient's symptoms was insidious. The patient missed her period but thought it was the usual irregular period that was normal with her. After one-month amenorrhoea, she noticed a sharp continuous pain in her left illac fossa radiating to the whole of her lower abdomen. Pain was not relieved by oral acetaminophen, hyoscine and ibuprofen. Two days after the onset of pains, she noticed bleeding per vaginam which was associated with dizziness and fainting spells.

She has had 2 mid trimester spontaneous abortions in the past one year. Two years before her presentation at UPTH she had intra uterine fetal death from an unknown cause. In these past pregnancies she had her antenatal care in a maternity home. She had been treated twice in the past for pelvic inflammatory disease.Her thyroid swelling was not noticed until she presented at UPTH, but she had noticed an uncomfortable palpitation in the past one year which was associated with excessive sweating, hotness of the body, loss of weight and increased appetite.

Physical examination revealed an anxious, acutely ill looking young woman in obvious painful distress. She was moderately pale but anicteric and afebrile. Her pulse rate was 168 beats per minute, blood pressure was 130/90 mmHg; respiratory rate was 24 cycles per minute and heart sounds were normal. Her chest was clear clinically. Examination of the neck revealed enlargement of the right lobe of the thyroid gland measuring 7cm by 7cm. Musculoskeletal examination showed sweaty skin and fine tremor of out stretched hands. Abdominal examination revealed lower abdominal fullness and marked generalized tenderness. Deep organs could not be palpated because of marked guarding. Based on clinical findings a diagnosis of ruptured ectopic pregnancy in a patient with possible thyrotoxicosis was made, and she was booked for emergency exploratory laparotomy. Results of investigations were as shown below:

Packed cell volume (PCV) (%): 32 (normal range 40-50).

Urinalysis: Cloudy, nil glucose, nil protein, nil leucocyte. Urine pregnancy test was positive.

Pelvic ultra-sonography showed a retroverted, normal sized and essentially empty uterus. No gestational sac or product of conception were seen in-utero. Leftt adnaexae harboured a thick-walled mass of suspicious identity. There was massive collection of fluid (? haemperitonium) in the pouch of Douglas and the pelvis.

Anaesthetic Management

At the pre-anaesthetic visit, history and investigations were essentially as previously recorded. She was pale, afebrile, anicteric with a pulse rate of 170 beats per minute. The blood pressure was 110/70 mmHg, heart sounds were normal and there was no mumur. Temperature was 37.2^0C, respiratory rate was 22 breaths per minute and breath sounds were vesicular with no added sound. There was an anterior neck swelling. Airway assessment was Mallampati II and physical status based on the American Society of Anesthesiologists (ASA) classification was class III. The planned procedure was explained to the patient to allay anxiety and rapport was established. Informed consent was obtained and a request was made for 2 units of blood to be grouped and cross-matched.Ranitidine, 50 mg and atenolol, 100 mg were prescribed to be administered intravenously and orally (with a sip of water) respectively. Her vital signs were monitored every 5 minutes by the nurse. When reviewed 50 minutes later, her blood pressure was noted to be 100/60 mmHg and pulse rate 110 beats per minute. She was resuscitated with 1 litre of normal saline.

Prior to her arrival to the operating theatre, routine checks were carried out on the anaesthetic machine, suction machine, monitors and laryngoscopes. Equipment for difficult intubation including a range of cuffed endotracheal tubes sizes 6.5, 7.0 and 8.0, a stylet and LMA size 3 were all assembled. A malleable stylet was inserted into the lumen of the most likely tube to be used (7.0). The tubes and LMA were all lubricated. Drug dosages were calculated and drawn up in labelled syringes.

In the operating room, standard routine monitoring was established. The baseline vital signs were a pulse rate of 118 beats per minute, blood pressure of 100/60 mmHg and respiratory rate of 24 breaths per minute.

Temperature was 37.4 degree centigrade. ECG showed normal sinus rhythm corresponding to the heart rate, while oxygen saturation was 100% on room air. The urethra was catheterized with a size 16 Foley's catheter.

The patient was preoxygenated for 5 minutes with 100% oxygen by facemask at a flow rate of 6 liters per minute. Anaesthesia was then induced with sleep dose of iv sodium thiopentone (200 mg). Cricoid pressure was applied as soon as the patient lost consciousness and 100 mg of iv suxamethonium was administered followed by second dose of sodium thiopentone (50 mg) and tracheal intubation with a size 7.0 cuffed portex orotracheal tube. Correct placement of the tube was confirmed by auscultation of the chest and normal capnographic tracing (4 kPa) before it was secured with adhesive tape.Anaesthesia was maintained with 0.2% to 0.4% isoflurane in 100% oxygen (6 litres per minute). Intravenous (iv) atracurium 25 mg was administered to achieve muscle relaxation. Analgesia was achieved with 100 mg of iv tramadol. Ventilation was controlled manually at about 18 breaths per minute via a circle breathing system.

Intraoperatively the heart rate ranged between 90 - 100 beats per minute, arterial blood pressure measured non-invasively was between 100 - 130 mmHg systolic, 60 - 90 mmHg diastolic, oxygen saturation remained 100% while ECG showed sinus rhythm. Capnograph tracing was normal and end tidal carbon dioxide ($EtCO_2$) was between 4-5 kPa. Axillary temperature ranged between 34.8 and 35.2^{0}C.The total estimated blood loss was 1.6 litres and about 4 litres of normal saline was infused.

At the end of the surgery which lasted 40 minutes, isoflurane was discontinued. The oropharynynx was cleared with a sterile catheter. When the patient was fully conscious and able to sustain a head lift for 5 seconds, the endotracheal tube was removed. Administration of 100% oxygen by face mask was continued for 5 minutes at a flow rate of 6 litres per minute before she was transferred to the ICU for further observation.

Surgery and Findings

Massive haemoperitoneum of about 1.5 litres was evacuated. Ruptured, left tubal ectopic pregnancy was seenand left salpingectomy was done. Haemostasis was achieved and the abdominal surgical incision was closed in layers- vicryl to rectus sheath and vicryl-rapide to skin.

Postoperative Management

In the ICU, vital signs were monitored every 15 minutes. The pulse rate was between 90 and 110 beats per minute, blood pressure was between 100/60 mmHg to 130/90 mmHg, temperature was maintained at 37.0^{0}C and arterial oxygen saturation was between 99 and 100%. Five percent dextrose in 0.9% saline infusion was continued at the rate of 1 litre 8 hourly for the first 48 hours and postoperative analgesia was achieved with intramuscular pentazocine 30 mg 6-hourly for 48 hours. She received systemic antibiotics (gentamycin, metronidazole and ampiclox) for 5 days.

She was encouraged to sit out of bed from the first day after surgery and to start ambulation on the second day. Graded oral feeds were commenced on the 2nd day and postoperative haemoglobin estimated on the same day was 9 gm/dl. The patient remained stable in the ICU until 36 hours later when she started complaining of severe palpitations. Her heart rate ranged between 136 and 148 beats per minute, blood pressure ranged between 96/60 and 85/50 mmHg while SpO_2 was between 85 and 94%. She had tolerated oral feeds well, and oral atenolol 100 mg was administered to her with good result after she was reviewed by the medical team. Thyroid function test done confirmed the suspicion of thyrotoxicosis. (Triiodothyronine (T_3) 3.0 (normal 0.51-1.58 ng/ml), thyroxine (T_4) 200 (normal l53-121 ng/dl), thyroid stimulating hormone (TSH) 0.53 (normal 0.44-3.45 iu/ml). She was commenced onoral carbimazole 10 mg 8 hourly and propranolol 20 mg 8 hourly. The urethral catheter was removed after 36 hours. She was transferred to the ward after 48 hours and discharged home on the 9th postoperative day. She was followed up by the surgical team and had a successful thyroidectomy done 6 months later.

Discussion

The incidence of ectopic pregnancy is directly related to the prevalence of salpingitis[1]. It has been suggested that there is a higher incidence of ectopic gestation in patients using intrauterine contraceptive device and in women taking progesterone only contraceptive pills.[1] In both cases there may be interference with tubal peristalsis. It is, however possible that some of the patients had pre-existing salpingitis, but the statistical evidience is uncertain.[1] This may be true in this patient with a previous history of pelvic inflammatory disease.

The incidence of ectopic pregnancy in United Kingdom is about 1 in every 80 pregnancies but in some urban areas, the rate may be as high as 1 in 50. Ectopic

pregnancy may be classified as unruptured (tubal) pregnancy, chronic or slowly-leaking ectopic pregnancy and ruptured ectopic pregnancy.[2] In an amenorrhoeic patient, the sudden onset of severe abdominal pain, recurrent vaginal bleeding, haemodynamic instability and distended tender abdomen are suggestive of ruptured ectopic pregnancy.

Diagnosis was made based on the above history and confirmed by ultra-sonography and pregnancy test. Needle aspiration is controversial.[1,2]If left untreated, about half of ectopic pregnancies will resolve without treatment. These are the tubal abortions. The advent of methotrexate treatment for ectopic pregnancy has reduced the need for surgery[11]; however, surgical intervention is still required in cases where the fallopian tube has ruptured or is in danger of doing so. This intervention may be achievedvia laparoscopy or laparotomy[2] as performed here.

Thyrotoxicosis though reported as one of the most common endocrine disorders in an otherwise healthy young population is not common in this environment as found by Soyannwo et al. in their review of medical diseases in patients coming for surgery under anaesthesia.[12] There are eight known causes of thyrotoxicosis namely: Graves's disease, toxic nodular goitre, thyroiditis, thyroid carcinoma, excessive production of thyroid stimulating hormone, excess extraneous thyroid hormone, neonatal and transient neonatal / postpartum and iodide induced hyperthyroidism[6]. Studies have shown that thyrotoxicosis during pregnancy is most often due to Graves disease.[13,14]The two signs that are truly 'diagnostic' of Graves' disease (i.e., not seen in other hyperthyroid conditions) are ophthalmopathy (thyroid eye disease) and non-pitting edema (pretibial myxedema).[13,14] The ophthalmopathy will include dry eyes, irritation, and blurriness, swelling of the eyelids, lid retraction, lid lag and exophthalmos.

The mechanism of action of thyroid hormones is by binding of T3 and T4 to the thyroid receptors in the cells of different organs in the body. This complexity permits thyroid hormones to produce their many different effects in the body. The thyroid receptor is expressed on the follicular cells of the thyroid gland (the cells that produce thyroid hormone), and the result of chronic stimulation is an abnormally high production of T3 and T4.[9,14] This in turn causes the clinical symptoms ofhyperthyroidism, and the enlargement of the thyroid gland visible as goiter such as seen here. The clinical spectrum of thyrotoxicosis ranges from asymptomatic biochemical abnormalities to life-threatening crisis with multi-system dysfunction and a high mortality rate.[13] Depending on the severity of thyrotoxicosis clinical presentation can be quite subtle.[16] Other major manifestations in hyperthyroidism are recurrent abortion, intra uterine fetal death, palpitations, and thyroid swelling.[27] Classical symptoms of thyrotoxicosis which included excessive sweating, heat intolerance, weight loss and increased appetite were also seen in this patient. Constitutive signs such as restlessness, sweaty skin and fine tremor of out stretched hands were also elicited. The combination of high pulse rate, palpitations, anterior neck swelling and other symptoms and signs point more to thyrotoxicosis. The patient admitted having uncomfortable palpitations in the past 1 year. There was no proptosis and other eye signs were not elicited in this patient. This may be explained by the fact that the thyrotoxicosis was not yet advanced. Her apex beat was located at the 5th left intercostal space midclavicular line which is an evidence that cardiomegaly a common complication of advanced thyrotoxicosis had not yet set in. A high pulse rate and sweaty skin may also exist in shocked patients, anxiety states, and thyrotoxicosis.

The greatest fear in the thyrotoxic patient is the precipitation of a thyroid storm which may be triggered in susceptible patients by thyroid as well as non-thyroid surgery, stress and infection,[18,19] resulting in the failure of the patient's metabolic, thermoregulatory and cardiovascular compensatory mechanisms.[19] Hyperthyroid patients who require emergency surgery are given esmolol, guided by changes in pulmonary artery wedge pressure.[9] The use of IV potassium iodide and a broad spectrum beta blocker such as propranolol or atenolol either orally or intravenously is also acceptable.[7] It is believed that surgery should be avoided in any patient whose thyroid function is clinically abnormal. Therefore, only a life-threatening emergency as seen here should preclude making the patient pharmacologically euthyroid before surgery.[20] Ideally, the patient should be rendered euthyroid by commencing on antithyroid therapy for about 4 – 8 weeks using carbimazole 5-15mg 3 times daily and propranolol 160-480mg daily before surgery.[21]

The anaesthetic management for emergency exploratory laparotomy in a patient with undiagnosed thyrotoxiocosis poses a serious challenge to every anaesthetist. Vasoactive drugs usually preferred in anaesthesia for ruptured ectopic may predispose the patient to thyrotoxic storm which is life-threatening. Therefor meticulous pre-anaesthetic assessment,

judicious use of drugs and close monitoring of patient are essential for safe anaesthetic care.

Ideally intravenous beta blockers like propranolol, esmolol, nadolol or sotalol should be administered to these patients. It has been established that propranolol can be used for rapid relief of symptoms of thyrotoxicosis, and it is often used together with anti-thyroid drugs and radioactive iodine to render the patient euthyroid and also give a sense of well being.[20,22] In the absence of above mentioned drugs oral atenolol was used, and found to be effective. However, atenolol has a long duration of action upto 24 hours and being a beta blocker may attenuate the compensatory sympathetic response to hypovolaemia which may be deleterious to this patient. Close monitoring of vital signs perioperatively as was done here was necessary.

General anaesthesia, with rapid sequence induction is often the anaesthetic technique of choice for patients with full stomach coming for emergency surgery. However, in the uncontrolled hyperthyroid patient for abdominal surgery, combination of general anaesthesia with a regional technique[23] has been suggested as a better approach. Variela et al. combined general anaesthesia and spinal with preservative free morphine (the latter for postoperative analgesia) with good effect.[24] Regional anaesthesia however may potentiate hypovolaemia and worsen the tachycardia. No controlled study has demonstrated clinical advantage of any anaesthetic drug over another for surgical patients who are hyperthyroid.[20] A review of cases performed at the University of California, San Francisco, from 1968 to 1982 revealed that virtually all anaesthetic drugs and techniques have been used without adverse effects even being remotely attributable to the drug or technique.[25] Drugs that stimulate the sympathetic nervous system with consequent exaggeration in elevation of blood pressure and heart rate like ketamine, pancuronium, atropine, were avoided in this patient.

Thiopentone may be the induction agent of choice, since it possesses some antithyroid activity at high doses.[9,10] Hyperthyroid patients can be chronically hypovolaemic and vasodilated and are prone to an exaggerated hypotensive response during induction.[9] To avoid exaggerated hypotensive response during induction, the patient was properly resuscitated with crystalloids in the accident and emergency ward before transfer to the theatre. Although hyperthyroid patients display accelerated drug biotransformation, caution was taken in the administration of sodium thiopentone. A sleep dose of sodium thiopentone was given to avoid excessive hypotension during induction of anaesthesia. Propofol has also been found useful for induction of anaesthesia in hyperthyroid patientsbecause it impairs the normal arterial baroreflex response to hypotension and changes in heart rate are insignificant in healthy patients.[9] A bolus dose may be used for induction, followed by an infusion for maintenance of anaesthesia.

Adequate depth of anaesthesia should be ensured before laryngoscopy and surgery in order to prevent tachycardia, hypertension and ventricular dysrhythmias. Ebegue reportedthatsecond dose of thiopentone attenuates the haemodynamic response to laryngoscopy and intubation.[26] Second dose of thiopentone was used here and was found effective. Esmolol and other beta-adrenergic antagonists available could be used both to attenuate this response, as well as control heart rate perioperatively in thyrotoxic patients.[27] Adequate analgesia perioperatively should also be ensured using drugs like fentanyl, sufentanil, alfentanil, pentazocine and tramadol. Fentanyl, sufentanil and alfentanil if available could also be used to obtund pressor response to laryngoscopy.[27] Experienced assistance is important when there is anticipated difficult intubation.

There was no contraindication to the use of suxamethonium. And, suxamethonium is characterized by rapid onset and short duration of action which is necessary in patients with full stomach and anticipated difficult intubation. Rocuronium, vecuronium, mivacurium and cis-atracurium are cardio-stable but do not guarantee short action in the case of a difficult intubation moreover, they were unavailable. Atracurium besylate was used because it is relatively cardio-stable unlessdoses above 0.5mg/kg are used.[28] It may cause hypotension due to histamine release but a slow rate of injection minimizes these effects.[28] Muscle relaxants should be administered cautiously because hyperthyroidism is associated with an increased incidence of myopathies and myasthenia gravis.[9] Monitoring of neuromuscular blockade is therefore indicated.

Isoflurane is more cardio-stable when compared to halothane and hence was used for the patient. Sevoflurane and desflurane where available should be used because they are more cardio-stable when compared to isoflurane. Halothane and enflurane should be avoided because hyperthyroid patients display accelerated drug biotransformation and may theoretically be more susceptible to hepatic injury from

halothane or kidney toxicity from enflurane.[9] Halothane also causes dysrhythmias in the presence of high plasma levels of circulating catecholamines.[29]

The residual neuromuscular blockade was not reversed because atracurium besylate which undergoes pH and temperature dependent Hofmann's degradation was used and also the patient even though peripheral nerve stimulator was not available showed evidence of full recovery by head up lift for 5 seconds and also obeyed command.

The most serious threat to hyperthyroid patients in the postoperative period is thyroid storm which may occur 6–24 hours postoperatively. Thyroid storm is characterised by hyperpyrexia, tachycardia, altered consciousness (e.g. agitation, delirium, and coma) and hypotension. It mimicks malignant hyperthermia but unlike the latter, thyroid storm is not associated with muscle rigidity, elevated creatine kinase, or a marked degree of lactic and respiratory acidosis. The monitoring of this patient was continued postoperatively in the intensive care unit to detect onset of thyroid storm.

Standard monitoring in the theatre and ICU was adequate for the patient; however invasive monitoring, where available is preferred because of the anticipated swings in blood pressure and haemorrhage. Invasive monitoring was continued for 24 hours postoperatively by Variela et al.[24]However, this was not performed because of the emergent presentation of the patient as no time was available for application of invasive monitors. There is no substitute for vigilance and careful monitoring of the anaesthetised patient by the anaesthetist.

Surgical thyroidectomy is simply the removal of all or part of the thyroid gland, which immediately reduces the levels of thyroid hormone in the body. Surgical thyroidectomy is also used to treat thyroid cancer and to remove large goitres or nodules that obstruct breathing or for cosmetic reasons. Thyroidectomy is typically considered a safe form of treatment, however up to seven percent of all thyroid surgeries result in some form of facial nerve damage, while up to three percent of patients are left with paralysis of both vocal cords.[21] Another possible side-effect is damage to the parathyroids, four tiny glands located close to the thyroid gland. This patient had successful thyroidectomy 6 months later without complications.

Conclusion

The successful anaesthetic management of a patient with undiagnosed thyrotoxicosis presenting for emergency laparotomy requires understanding of the pathophysiology and haemodynamic changes characteristic of the disease process. A high index of suspicion is necessary for accurate diagnosis and effective perioperative management as symptoms and signs may be blurred by the presenting illness. Close monitoring of this patient after preoperative administration of oral atenolol was highly indicated for a favourable outcome. The expertise of the anaesthetist in the provision of pain relief, management of haemodynamic derangement, fluid balance and familiarity with the drugs used are essential for safe anaesthesia.

References

1. Panelli D, Phillips C, Brady P. Incidence, diagnosis and management of tubal and nontubal ectopic pregnancies: a review. Research and Practice 2015; 1: 15.
2. Murray H, Baakdah H, Bardell T. Diagnosis and treatment of ectopic pregnancy. CMAJ 2005; 173(8):905-912.
3. Zhang J, Li F, Sheng Q. "Full-term abdominal pregnancy: a case report and review of the literature". Gynecol Obstet Invest2008; 65(2): 139–141.
4. Farquhar CM. Ectopic Pregnancy. Lancet 2005; 366: 583.
5. Abalovich M, Amino N, Barbour LA, Cobin RH, De Groot LJ, Glinoer D. Management of thyroid dysfunction during pregnancy and postpartum: an Endocrine Society Clinical Practice Guideline. J Clin Endocrinol Metab. 2007; 92(8):S1-47.
6. Farling PA. Thyroid disease. Br J Anaesth 2000; 85: 15-20.
7. ACOG Practice Bulletin. Clinical management guidelines for obstetrician-gynecologists. Thyroid disease in pregnancy. Gynecol 2002; 100(2):387-396.
8. Azizi F. Treatment of post-partum thyrotoxicosis. J Endocrinol Invest 2006; 29(3):244-247.
9. Morgan GE Jr, Mikhail MS. Editors. Anaesthesia for patients with endocrine disease in: clinical anesthesiology 2nd edition. Lange medical books/Mcgraw-Hill New York 1996; pp640-641.
10. Mato CN, Johnson UU, Odagme MT. Anaethesia in the undiagnosed thyrotoxic

patient: A case report. Sahel Med J 2007; 10:63-66.

11. Clark L, Raymond S, Stanger J, Jackel G. "Treatment of ectopic pregnancy with intraamniotic methotrexate--a case report". Aust New Zealand J Obstet Gynaecol 1989; 29 (1): 84–85.

12. Soyannwo OA, Bamgbade OA and Odutola OO. Medical Diseases and anaesthesia. Afr J Anaesth Intens Care 1996; 2:51-56.

13. Brent GA. Clinical practice. Graves' disease. N Engl J Med. 2008; 358(24):2594-605.

14. Lazarus JH, Kokandi A. Thyroid disease in relation to pregnancy: a decade of change. Clin Endocrinol 2000; 53(3):265-278.

15. Cawood T, Moriarty P, O'Shea D. "Recent developments in thyroid eye disease". Br Med J 2004; 329 (7462): 385–390.

16. So PC. Unmasking thyrotoxicosis during anaesthesia. Hong Kong Med J 2001; 7: 311-314.

17. Houck JA, Davis RE, Sharma HM. Thyroid-stimulating immunoglobulin as a cause of recurrent intrauterine fetal death. Obstet Gynaecol 1988; 71:1018-1019.

18. Burch H, Wartofsky L. Life threatening thyrotoxicosis. Endocrinol Metab Clin North Am J 1993; 22:263-274.

19. Nicoloff J. Thyroid storm and myxedema coma. Med Clin North Am J 1985; 69: 1005-1012.

20. Michael F. Roizen and Lee A. Fleisher. Anaesthetic implications of concurrent diseases. In: R.D. Miller, Editor. Millers Anaesthesia, 6th edition Elsevier, Churchill Livinstone, Philadelphia 2005; pp1046-1047.

21. Singer PA, Cooper DS, Levy EG, Ladenson PW, Braverman LE, Daniels G. Treatment guidelines for patients with hyperthyroidism and hypothyroidism. Standards of Care Committee, American Thyroid Association. JAMA 1995; 273(10):808-812.

22. Nachum Z, Rakover Y, Weiner E, Shalev E. Graves' disease in pregnancy: prospective evaluation of a selective invasive treatment protocol. Am J Obstet Gynecol 2003; 189(1):159-165.

23. Robson NJ. Emergency surgery complicated by thyrotoxicosis and thyrotoxic periodic paralysis. Anaesth 1985; 40:27-31.

24. Variela A, Yuste A, Villazalla R, Garrido J, Lorenzo A. Spinal anaesthesia for emergency abdominal surgery in uncontrolled hyperthyroidism. Acta Anaesthesiol Scand 2005: 49: 100–103.

25. Roizen MF, Becker CE: Thyroid storm: A review of cases at the University of Califonia, San Francisco. Calif med 1971; 115:5.

26. Ebegue AF. Second dose thiopentone attenuates the haemodynamic response to laryngosopy and intubation. Middle East J Anesthesiol 2001; 16:81-90.

27. Morgan GE Jr, Mikhail S, editors. Anaesthesia for patients with cardiovascular disease In: Clinical Anesthesiology 2nd edition. Lange medical books/Mcgraw-Hill. New York 1996; 346.

28. Scott RP, Savarese JJ, Basta SJ. Atracurium: Clinical strategies for preventing histamine release and attenuating the haemodynamic response. Br J Anaesth 1985; 57:550-553.

29. Lowry LD. Safe use of epinephrine with halothane anesthesia. Laryngoscope 1977; 87: 1277-1280.

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