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Case Report

Persistent intraoperative sinus tachycardia: threat of thyroid storm? A case report

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ABSTRACT

Sinus tachycardia in a patient undergoing surgery under general anaesthesia is not an uncommon experience in anaesthesia practice. Causes of intraoperative sinus tachycardia can be multi-factorial. Operative causes are pain, surgical stimulation and light depth of anaesthesia. Pharmacological factors include administration of catecholamines, atropine, or ketamine. Medical factors such as sepsis, hypovolaemia, heart failure, anaemia, and thyrotoxicosis should also be considered. We report a case of inadequately controlled hyperthyroidism undergoing surgery for a spinal cord tumour under general anaesthesia who developed unexplained tachycardia intraoperatively.

Keywords: Unexplained intraoperative tachycardia, Thyroid storm

INTRODUCTION

Sinus tachycardia in a patient undergoing surgery under general anaesthesia is not an uncommon experience in anaesthesia practice. Intraoperative tachycardia has been shown to be a risk factor in the occurrence of postoperative complications, more so in older age groups. Rose *et al*. conducted a large, prospective study to determine the contribution of various risk factors for postoperative cardiac events. Both intra- and postoperatively, tachycardia (HR >120 beats/min for longer than ten minutes) and hypertension (systolic BP >200 mmHg for longer than five minutes) were found to be associated with an increased incidence of postoperative cardiac events.[1]

Causes of intraoperative sinus tachycardia can be multi-factorial. Operative causes are pain, surgical stimulation and light depth of anaesthesia. Pharmacological factors include administration of catecholamines, atropine, or ketamine. Medical factors such as sepsis, hypovolaemia, heart failure, anaemia, and thyrotoxicosis should also be considered.[2]

Thyroid storm is the most severe form of thyrotoxicosis and is a life threatening condition. Though it's incidence during thyroid surgery has gone down, it may occur in a patient of hyperthyroidism in the face of additional stress of any infection, diabetes, heart failure, trauma, or indeed any acute stress including that of coincidental surgery.[2] We report a case of inadequately controlled hyperthyroidism undergoing surgery for a spinal cord tumour under general anaesthesia who developed unexplained tachycardia intraoperatively and was successfully managed.

CASE REPORT

A 26 year female diagnosed to have spinal cord tumor was posted for laminectomy and excision of tumor. She had complaints of progressively increasing pain and weakness in lower back and both lower limbs since

5months. She gave history of having hyperthyroidism since 4yrs and was on treatment previously but had discontinued it on her own. On admission her thyroid profile was as follows - T_3 1.92 ng/ml (0.52-1.9), T_4 14.85 µg/dl (4.4-11.6) and TSH 0.05µIU/ml (0.39-6.16). She was put on tab. carbimazole 20 mg BD and tab. propranolol 40 mg BD. After about 20 days her thyroid profile values were T_3 1.13 ng/ml (0.52-1.9), T_4 10.2µg/dl (4.4-11.6) and TSH 0.05µIU/ml (0.39-6.16). In preanaesthetic examination her vitals recorded were HR 84/min, BP 116/70 mmHg and RR 14/min. Local examination revealed a diffuse swelling in anterior aspect of neck of size 7x8 cm that was firm and moved with swallowing. She also had exophthalmos with lid lag and lid retraction. CNS examination revealed power grade 3 in both lower limbs; sensations and DTR were normal. Other systems were normal.

Other blood investigations were normal. X-ray neck AP view showed deviation of trachea to left; there was no evidence of compression in lateral view. MRI study of lumbo-sacral spine revealed well defined intradural extramedullary lesion at L1-L2 level compressing adjacent cauda equina nerve roots (?meningioma, ?nerve sheath tumor). In view of this compression and the values of T3 and T4 becoming normal, she was accepted for anaesthesia even though the TSH was still low (subclinical hyperthyroidism) as ASA III. She was advised to take the morning dose of carbimazole as well as propranolol with a sip of water as per institutional protocol.

After shifting to operating room monitors like NIBP, ECG, SpO_2 probe were attached. Baseline pulse was 72/min, BP 116/72 mmHg, SpO_2 100% on room air and axillary temperature was 37°C Patient was preoxygenated and premedicated with inj. Dexamethasone 8 mg, inj. ranitidine 50 mg, inj ondensatron 4 mg, inj. midazolm 1.5 mg and inj. fentanyl 100 µg intravenously. She was induced with inj thiopentone 300 mg and inj vecuronium 5 mg. On laryngoscopy 10% lignocaine was sprayed over vocal cords and the patient was intubated with cuffed armoured ET tube number 7.5. After confirming equal bilateral air entry, patient was maintained on O_2+nitrous oxide in the ratio of 50:50 and isoflurane 1 % via circle absorber and ventilated with the parameters of Volume control, tidal volume 400 ml, respiratory rate of 12 breaths/min, PEEP of 3 cm of water. With these settings the peak airway pressure was 15-16 cm of water. Continuous cardiac monitoring for rate and rhythm, NIBP, SpO_2 and $ETCO_2$ monitoring were done and she was turned prone for the surgery.

Twenty minutes after the start of procedure the patient started developing sinus tachycardia (>110/min). Systolic BP increased to >130 mmHg. Plane of anesthesia was deepened with inj. fentanyl 50 micrograms and increasing concentration of isoflurane. Further top-up of vecuronium 2 mg was also given. Fluid replacement was checked and found ok. Fluid challenge bolus of 200 ml was also given. Tachycardia did not settle; hence inj. thiopentone 2 mg/ml infusion was started and titrated according to her BP and pulse rate. There was no significant response and after some time, heart rate increased more to >120/min with the BP being maintained. Inj. metoprolol 2.5 mg was given. The tachycardia came down to 116 beats/min but again increased to about 128/min. 1 mg of additional metoprolol had no effect. We could not monitor the nasopharyngeal temperature as we did not have a suitable working probe but the skin temperature never increased above 37°C. The $ETCO_2$ was between 32 to 36 mmHg throughout the case. At this point of time after enquiring from her relatives, we discovered that she had not taken her morning doses of carbimazole and propranolol as prescribed. So we gave tab. carbimazole 20 mg crushed and mixed with 20 ml saline through the nasogastric tube that we had already placed as per institutional protocol. We had already used beta blocker metoprolol, and so did not give propranolol. About 40 min after administration of carbimazole, the patient's tachycardia gradually settled to about 90/min though the isoflurane concentration, the speed of the thiopentone drip were the same and no further fentanyl was given. Thereafter the heart rate remained stable between 80 to 90 beats/min and the thiopentone drip was gradually withdrawn and isoflurane also decreased. About an hour later the surgery was completed. The patient was turned supine and reversed with inj. Neostigmine 2.5 mg + inj. glycopyrolate 0.4 mg intravenously. Inj. xylocard 1% was given to prevent stress response to extubation. Extubation was uneventful. Post-operatively patient was observed in ICU and remained stable.

DISCUSSION

Our patient developed intraoperative tachycardia which was initially sought to be treated with additional doses of fentanyl, vecuronium and increasing the concentration of inhalational agent. A drip of thiopentone was started with the thought of deepening the level of anaesthesia and also for the additional benefit of decrease in the peripheral conversion of T_4 to T_3. Adequate fluid administration and a good urine output with a stable BP indicated adequate hydration. β blockers were the next line of treatment. Though there was a brief decrease in the heart rate from 124/min to 116/min, it again increased to 128beats/min and after that there was no appreciable response to additional 1 mg of metoprolol. Thyrotoxic patients appear to have a change in the affinity of catecholamines for their receptors or a change in receptor number or a modification of a postreceptor mechanism.[3] Propranolol is the most commonly used beta-blocker to treat thyrotoxicosis and has been the mainstay beta-blocker therapy to prepare thyrotoxic patients for surgery.[4] An advantage of propranolol compared to other beta-blockers is that it blocks peripheral conversion of T_4 to T_3 at high doses. Intravenous propranolol can also be given in the event oral intake is limited post-operatively. A disadvantage is its short half-life requiring high doses and frequent administration of up to four times daily to

maintain therapeutic plasma levels. This can limit medication compliance and increases the risk of perioperative thyroid storm or hyperthyroid symptoms if doses are missed or inadequate doses are prescribed.[4] Difficulties surrounding the use of propranolol led to a number of studies of other beta-blockers, in particular, more cardioselective agents such as metoprolol. Results suggest that metoprolol is as effective as propranolol. Furthermore, there is also a stronger association between plasma metoprolol levels and clinical efficacy than with propranolol. Metoprolol also offers a simpler twice-daily dosing regimen and can be given intravenously when oral administration is limited.[4]

Corticosteroids produce a rapid fall in T_3 and T_4 and a rise in reverse T_3 when administered to hyperthyroid Grave's disease patients, suggesting a direct inhibition of glandular secretion of thyroid hormone as well as the inhibition of peripheral monodeiodination.[5]

Thiopentone also decreases the peripheral conversion of T_4 to T_3. Our patient had received dexamethasone and thiopentone infusion. At this point we questioned the relatives to confirm that the patient had received the morning doses of carbimazole as well as propranolol and learnt that she had omitted to take both the drugs as she was fasting. We had prescribed the morning doses as per our institutional protocol and assumed that she had taken them. We did not confirm this before the induction of anaesthesia as we should have.

When sinus tachycardia occurs in a patient of thyrotoxicosis, thyroid storm is a possibility that has to be considered. We then gave 20 mg of carbimazole through the nasogastric tube with the thought that there should not be a longer gap in her antithyroid medication. Langley and Burch have stated that as drugs are metabolised faster in hyperthyroid patients, the drug dosing frequency may actually have to be increased in such patients.[3] Carbimazole acts by prevention of organification of iodine and coupling of iodotyrosines, hence decreasing the synthesis of T_4 and T_3. It does not block the peripheral conversion of T_4 to T_3 and so we did not expect a rapid action of carbimazole. To our pleasant surprise, after about 40 to 45 min of administration of carbimazole, the sinus tachycardia responded even though the patient was receiving the same concentration of isoflurane and thiopentone as before, without any additional dose of metoprolol. Peak plasma concentration of Methimazole is achieved within 30 mins to 3 hours after ingestion and the half-life is from 3 to 6 hours.[6]

Since the persistent tachycardia did not respond to deepening of anaesthesia, additional analgesia, IV fluid challenge, and there was no hypoxaemia or hypercarbia, we feel that it was due to excessive thyroid activity. It is speculated that thyroid hormones have direct effects on the conduction system, possibly via cellular changes in cation transport, including a decrease of atrial excitation threshold, an increase of sinoatrial node firing, and a shortening of conduction tissue refractory time.[7] The heart rate reached 120-128/min and not more. But it needs to be considered on the background of the patient already being treated with β blockers. Was it the beginning of a storm? The cardinal features of a thyroid storm are tachycardia, fever, increase in $ETCO_2$, CNS symptoms like delirium, confusion and irritability and GIT symptoms of hypermotility. All these symptoms have been described in a non-anaesthetised patient breathing spontaneously. When the patient is on IPPV under anaesthesia, there may not be any hypercarbia. In our patient the $ETCO_2$ did not increase beyond 36 mmHg at any point during the course of anaesthesia. The peripheral temperature did not increase beyond 37°C. The temperature of an anaesthetised patient does tend to drop in the air conditioned cold OT environment. Hence we may not have appreciated any increase in the patient's temperature and only an oesophageal probe would have given us the true increase in core body temperature if it had occurred. The management of thyroid crisis must proceed as follows (in this order): (1) protect airway, obtain vascular access and stabilize haemodynamics; (2) initiate specific therapy aimed at ameliorating the proximate cause of the crisis (e.g., antibiotics for pneumonia); and (3) reduce thyroid hormone levels and treat the adrenergic signs of thyrotoxicosis.[8]

The point at which thyrotoxicosis transforms to thyroid storm is controversial, and is, to some degree, subjective. In an effort to standardize and objectify thyroid storm somewhat, as compared with severe thyrotoxicosis, Burch and Wartofsky have delineated a point system assessing degrees of dysfunction in various systems (thermoregulatory, central nervous, gastrointestinal, and cardiovascular). The Burch-Wartofsky score of our patient was 25 (tachycardia of 120-129/min i.e., 15 points and presence of a precipitating factor i.e., 10 points). A score of 25 to 44 is suggestive of impending storm. Clinically, it is prudent to assume that someone with severe thyrotoxicosis has impending thyroid storm, and to treat them aggressively, rather than focus on specific definitions.[9] Malignant hyperthermia is a differential diagnosis of thyroid storm. In our case, we never considered it as the $ETCO_2$ did not increase beyond 36 mmHg, nor did the temperature of the patient increase.

So PC has also reported a case where the patient had intraoperative tachycardia without fever or hypercarbia, which, on investigations postoperatively was diagnosed to have thyrotoxicosis.[10] We did not come across any mention in literature that could explain why the tachycardia in our patient was controlled after administration of carbimazole. In fact it is mentioned that thionamides will provide little immediate relief when used to start the treatment of thyroid crisis.[8] There is a possibility that as the patient was already receiving carbimazole, the dose that we gave through the nasogastric tube was useful to control the thyroid hyperactivity. It may be that β-blockade was more effective after the increase in the plasma level of

carbimazole and hence its antithyroid activity. But this remains our speculation.

CONCLUSION

We conclude from our case that it is necessary to confirm that in patients on thionamides who are taken for surgery, the applicable doses of antithyroid medications have been taken preoperatively. An impending thyroid crisis should be considered if unexplained tachycardia occurs intraop. Besides β-blockers, the management in this situation should include the thionamide which can be given through the nasogastric tube.

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