VOLUME - 11, ISSUE - 11, NOVEMBER - 2022 • PRINT ISSN No. 2277 - 8160 • DOI : 10.36106/gjra

Original Research Paper

Anaesthesiology

RECURRENT SINUS ARREST DURING LAPAROSCOPIC SACRAL COLPOPEXY IN PATIENTS WITH SICK SINUS SYNDROME : A CASE REPORT

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ABSTRACT Sick sinus syndrome (SSS) is a disease in which the SA node fails to perform the function of pacemaking. ECG shows various rhythms including sinus arrest. We report recurrent sinus arrest during laparoscopic sacral colpopexy in a 77-year-old female patient with SSS. The patient was diagnosed with SSS, but as there were no symptoms, the operation was performed without pacemaker implantation. After induction of anesthesia, vital signs were stable, but sinus arrest repeatedly appeared due to elevated vagal tone during uterine traction. After operation, the patient underwent pacemaker implantation, and to this day, she is doing well without any symptoms. The anesthesiologist should pay close attention to the progress of the operation through much communication with operator during the surgery. Also, we recommend to considering temporary pacemaker implantation for patients with SSS who undergo surgery that can increase vagal tone.

KEYWORDS : sick sinus syndrome; sinus arrest; laparoscopic sacral colpopexy, general anesthesia; pacemaker

INTRODUCTION

Sick sinus syndrome (SSS) is a general term for diseases characterized by abnormal cardiac impulse formation and propagation of the sinoatrial (SA) node. It prevents the SA node from performing normal pacemaker functions (Samelka et al, 2013). It manifests as dysrhythmia, including bradycardia, sinus pause, sinus arrest, and tachycardia–bradycardia syndrome on electrocardiogram (ECG) (Gunes et al, 2017). Sinus arrest is a malfunction of atrial depolarization due to the autonomic cell of the sinus node failing to generate electrical impulses. Consequently, P-QRS-T waves are not observed on ECG, and this lack of waves for over 2 s is defined as sinus arrest (Adachi et al, 2010). Sinus arrest can be caused by excessive vagal tone excitation, medications, hypertension, diabetes mellitus, hyperthermia, and electrolyte abnormalities, such as hyperkalemia. In patients with SSS, sinus arrest may be prolonged and repeated resulting from these causes (Hawks et al, 2021). In particular, propofol and remifentanil, used in general anesthesia, and surgical procedures, such as laparoscopic surgery, general abdominal surgery, and neurosurgery, cause more frequent and prolonged sinus arrest in patients with SSS than those without (Khanna et al, 2020 ; Doyle et al, 1990). As repeated and prolonged sinus arrest can lead to cardiac arrest, caution must be exercised (Ferrer, 1973). SSS cannot be treated with medications alone and requires pacemaker implantation (Samelka et al, 2013).

Here, we report a case of SSS in a patient who did not have adequate indications for pacemaker implantation and showed recurrent sinus arrest during laparoscopic sacral colpopexy under general anesthesia.

Case History

A 77-year-old woman diagnosed with uterus prolapse was admitted to undergo laparoscopic sacral colpopexy. A regular surgery was planned 1 day after admission.

The patient had a history of hypertension, diabetes mellitus, and asthma and was on metformin 1,000 mg, sitagliptin 50 mg, calcium 250 mg, montelukast 10 mg, and candesartan 8 mg daily. Preoperative evaluation included ECG, laboratory tests (complete blood count, prothrombin time, activated partial thromboplastin time, Aspartate Transaminase (AST) / Alanine Transaminase (ALT), blood urea nitrogen level, creatinine level, glomerular filtration rate, Na level, K level, protein level, albumin level, Urinary analysis pulmonary function test, and chest X-ray. ECG showed sinus arrhythmia, while all other tests were unremarkable. Therefore, the patient was consulted by the cardiology department. After ultracardiography (UCG) and 24-h Holter monitoring, the patient was re-consulted. UCG showed an ejection fraction of 60% and normal left ventricular systolic function, and 24-h Holter monitoring ECG showed a minimum heart rate (HR) of 30 bpm, maximum HR of 92 bpm, junctional escape rhythm, premature atrial complex of 3.8%, and longest RR interval of 5.3 s. Based on these findings, the patient was diagnosed with SSS; however, she had no other symptoms, and the indications for a pacemaker did not correlate with her ECG findings. Therefore, a pacemaker was not implanted.

Approximately 30 min preoperatively, the patient received premedication with tabinul 0.2 mg intramuscularly, midacum 1 mg intramuscularly, and gaster 20 mg intravenously and was transferred to the operating room. Noninvasive blood pressure (BP), ECG (lead II), saturation of peripheral oxygen (SpO2), end-tidal carbon dioxide, and bispectral index were monitored. In the operating room before induction of anesthesia, vital signs were as follows: BP, 160/75 mmHg; HR, 55 bpm; SpO2, 95%; and sinus rhythm.

Anesthesia was induced by injecting an intravenous bolus of 1% lidocaine 50 mg and rocuronium 40 mg. Propofol was infused intravenously at 4 mg/kg/h in Schnider model target-controlled infusion mode, and remifentanil was infused intravenously at 4 ng/mL in Minto model target-controlled infusion mode. Subsequently, a 7.0-mm cuffed endotracheal tube was used for tracheal intubation. Vital signs showed no significant changes after intubation.

Anesthesia was maintained with 2 L/min O2, 2 L/min fresh air, 2.5–4.0 mg/kg/h propofol, and 2–4 ng/mL remifentanil. The patient was placed on mechanical ventilation support with volume-controlled ventilation at a tidal volume of 400 mL and a rate of 14 breaths/min. At 14 min after the start of the operation, an HR 32 of bpm and bradycardia were observed.

The patient recovered after injecting intravenous boluses of atropine 0 and 25 mg. Uterine traction was observed 24 min after the operation started, showing symptoms of asystole, which were observed every time the uterus was pulled in the operation field. The intravenous bolus of atropine 0.5 mg did not restore HR to a normal range. HR only recovered when the uterus was released (Figure 1). Other vital signs, such as BP and SpO2 remained stable. The bispectral index was well maintained between 40 and 60. The patient's condition was notified to the surgeon. Propofol infusion was discontinued, and anesthesia was maintained using sevoflurane 2.0 v% instead.

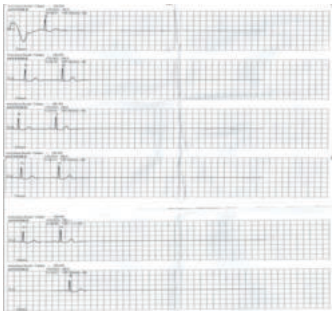

Figure 1: ECGs during uterine traction. This figure shows that sinus arrest occurred repeatedly during uterine traction

At 30 min after the start of the operation, asystole was observed upon uterine traction. However, unlike before, the patient showed findings of hypotension, with a BP of 89/39 mmHg. Upon an intravenous bolus injection of ephedrine 8 mg, HR and BP were 21–33 bpm and 73/41 mmHg, respectively, consistent with bradycardia and hypotension, respectively. After an additional intravenous bolus of ephedrine 4 mg, HR and BP recovered to 80 bpm and 165/85 mmHg, respectively, and uterine traction was no longer observed. The vital signs remained stable. The operation was completed uneventfully.

The patient was extubated using sugammadex 200 mg and moved to the post-anesthesia care unit (PACU). The arterial blood gas analysis conducted at the PACU showed pH of 7.472, pCO2 of 39.3 mmHg, pO2 of 86.5 mmHg, Na level of 143 mEq/L, and K level of 3.32 mEq/L. The patient received an injection of fentanyl 50 g and was transferred to the ward without administration of additional drugs or other symptoms. After consulting the cardiology department again on postoperative day (POD) 1, 24-h Holter monitoring ECG was re-conducted on POD 2. Upon examination, minimum HR of 31 bpm, longest RR interval of 7.3 s, and normal sinus rhythm with junctional escape rhythm were observed. The patient complained of dizziness starting on POD 2. The cardiology department decided that the patient had suitable indications for pacemaker implantation, and the pacemaker was implanted on POD 3. The patient is currently stable and on medications without any complaints.

DISCUSSION

SSS is a disorder characterized by impaired generation of action potential in the SA node and an atrial rate that does not meet the physiological requirements (Dobrzynski et al, 2007). Among the different causes, such as SA node degenerative fibrosis, ion-channel dysfunction, and SA node remodeling, idiopathic degenerative fibrosis is the most common (Demoulin et al, 1978). Additionally, autonomic dysfunction, increased vagal tone, metabolic disturbances, obstructive sleep apnea, antiarrhythmic medication, drugs, and toxins, such as dioxin, can exacerbate SSS. SSS is a progressive disease that is asymptomatic in the early stages and gradually causes symptoms of end-organ hypoperfusion. The most common symptom is syncope, with approximately 50% of patients developing syncope due to cerebral hypoperfusion (Samelka et al, 2013). Other symptoms, such as fatigue and oliguria, may occur. In more severe cases, angina, congestive heart failure, and transient ischemic attack may develop, and some patients may suffer sudden death (Rodriguez et al, 1990).

Currently, no standardized diagnostic criteria for SSS exist. The disease is mainly diagnosed based on symptoms, history, and ECG findings (Homoud, 2022). ECG in patients with SSS often includes the following rhythms: sinus bradycardia, sinus arrest longer than 3 s, SA exit block, and tachycardia–bradycardia syndrome (Adan et al, 2003). However, in addition to ECG or Holter ECG, SSS requires evaluation of symptoms and correlations for the accurate diagnosis.

Permanent pacemaker implantation is the only effective treatment for SSS. Table 1 shows the indications of pacemaker implantation (Table 1). In our case report, the patient's symptoms did not correlate with ECG findings preoperatively and, therefore, the pacemaker was not implanted. However, postoperatively, the patient showed suitable indications and underwent pacemaker implantation.

TABLE-1 Reasons for Pacemaker implantation in SSS

Indicated	Documented symptomatic bradycardia with frequent sympromatic sinus pauses
	Symptomatic chronotropic imcompetence
	Symptomatic sinus bradycardia caused by essential medication
Reasonable	Symptoms of bradycardia and documented HR < 40bpm without documentation of bradycardia during symptom
	Unexplained syncope with dysfunction of SA node found or caused in electrophysiologic study
Considered	Minimal symptom with HR < 40bpm

Source : ACC/AHA/HRS 2008 Guidelines for Device-Based Therapy of Cardiac Rhythm Abnormalities

In our case, the recurrent sinus arrest may be related to the surgical procedure. Laparoscopic sacral colpopexy involves uterine traction during surgery. When the uterus is pulled, the pelvic splanchnic nerve, a parasympathetic nerve that innervates the uterus, is excited and initiates the reflex (Dabbous et al, 2014). Uterine traction stretches the infundibulopelvic ligament that anchors the uterus to the peritoneum, which causes peritoneum traction (Jang et al, 2013). This excites the uterovaginal plexus of the pelvic plexus, leading to the vasovagal reflex (Imai et al, 2022). Additionally, laparoscopy increases the vagal tone. For laparoscopic procedures, the abdominal cavity is expanded with CO2, and the internal contents are separated to artificially create pneumoperitoneum. The CO2 gas in the abdominal cavity increases the intra-abdominal pressure and, subsequently, the vagal tone (Steer et al, 2019). In particular, most gynecological surgeries are conducted in the Trendelenburg position, which easily increases the intra-abdominal pressure and further intensifies vagal tone increase. As previously described, the increased vagal tone aggravates SSS, which caused recurrent sinus arrest in our patient. The patient was diagnosed with SSS preoperatively and did not develop recurrent sinus arrest. When the surgery was started after induction of anesthesia, sinus arrest was not observed. Uterine traction triggered sinus arrest repeatedly, and HR recovered to a normal range after traction.

Drugs for general anesthesia also aggravate SSS. Propofol can cause sinus bradycardia or atrioventricular block, which can have greater effects on SSS compared to sevoflurane (Dabbous et al, 2014). Opioids, such as remifentanil, have vagotonic effects suppressing sympathetic outflow.

Simulation infusion of propofol and remifentanil can lead to sinus arrest (Maruyama et al, 2010). Therefore, after recurrent sinus was observed in the patient, we used sevoflurane instead of propofol with the lowest possible concentration of remifentanil to maintain anesthesia.

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