Is general anesthesia a risk for myocardium? Effect of anesthesia on myocardial function as assessed by cardiac troponin-i in two different groups (isofluran+N_2O inhalation and propofol+fentanyl iv anesthesia)

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Background and objectives: Peroperative myocardial infarction (MI) is the most common cause of morbidity and mortality. What is the role of general anesthesia in this process? Is general anesthesia a risk for myocardial infarction? The present study was designed to determine whether the measurement of serum levels of cardiac troponin I (cTnI), a highly sensitive and specific marker for cardiac injury, would help establish the diagnosis of myocardial infarction in two different types of anesthesia.

Method: Elective abdominal hysterectomy was planned with the permission of the ethic committee in 40 patients who were 20–45 years range, in ASA-I group, and have a Goldman Cardiac Risk Index-0. The patients were divided into two groups. Isoflurane + N_2O was administrated to first group, and Propofol + Fentanyl to second group. cTnI levels were determined before anesthesia, after induction before surgery and 9 hours after the second period respectively.

Results: There was no significant difference between the groups by the means of demographic properties, hemodynamic parameters and cTnI levels, and the cTnI levels were determined under the basal levels in all samples.

Conclusion: General anesthesia is not a risk for myocardial infarction to state eliminating risk factors and protection hemodynamia cardiac.

Keywords: cardiac troponin-I, myocardial infarction, isofluran + N_2O inhalation anesthesia, propofol + fentanyl intravenous anesthesia.

Introduction

Peroperative myocardial infarction (MI) which is detected by troponin I is the most common cause of morbidity and mortality (Adams et al 1994). What is the role of general anesthesia in this process? Is general anesthesia a risk for MI?

Anesthesia directly causes myocardial depression and also effects vascular tonus and myocardial contractibility by autonomic nervous system or by hypoxia, hypercapnia, and acidosis depress myocardium (Corial et al 1998; Miller et al 1988). Apart from anesthesia, complications such as surgery, bleeding, infection, fever, and emboli also increase cardiac load. In the absence of cardiac disease, either myocardium may not tolerate this additional stress and failure or ischemia may develop. In order to decrease postoperative mortality and morbidity, risks must be determined and prevented.

We tried to determine the cardiac injury, in three periods (pre-, per- and postoperative), and we realized that it has no side effects after using isofluran + N_2O inhalation

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and propofol + fentanyl intravenous anesthesia in healthy patients who underwent noncardiac surgery.

Method

The study was conducted with the permission of the faculty etiquette committee. All patients were informed and consented. Elective abdominal hysterectomy was planned in 40 patients who were 20–45 years range, in ASA-I group and Goldman Cardiac Risk Index-0. The patients were admitted to operation room without premedication, monitorization, and vital parameters (TA, pulse, arterial O_2 saturation) were recorded. Two venous catheters were placed on each hand, blood samples were taken from one, and fluid was given into the other. The patients were divided into two groups. Each group was formed by 20 patients. Inhalation anesthesia was administrated to the first group, and intravenous anesthesia to the second. After induction with thiopental sodium (4–7 mg/kg) + fentanyl (1 μg/kg), inhalation anesthesia was maintained by isofluran 1.5% + N_2O/O_2 (50%/50%). In the second group, after induction with propofol (3 mg/kg) and fentanyl (5 μg/kg), anesthesia was maintained by propofol (0.2 mg/kg/min) and fentanyl (0.2 μg/kg/min). The patients in this group were ventilated by 50% O_2 and 50% air. Muscle relaxation was provided by vecuronium bromide (0.1 mg/kg) in both groups, and mechanical ventilation was applied as follows: V_T: 8–10 ml/kg, f: 10–12 min, P_{max} 20–30 mbar, PEEP: O mbar, $T_{1/E}$: 1/1.7 monitorization and ventilation was obtained from DRÄGER Cato 8040 anesthesia machine and perfusion by Compact B BRAUN perfuser.

Three blood samples were taken from patients, before anesthesia, after induction before surgery and 9 hours after second period. Blood samples were taken into sterile tubes which did not contain EDTA and heparin in order to avoid low results, and they were centrifuged and stored in −20 °C.

Cardiac troponin I levels were calculated by ng/ml with solid phase, two-site chemluminescent immunometric assay method in Immulite Automated Analyser. Normal Range: <0.4ng/ml. Statistical analysis was performed by SPSS for Windows (Microsoft, Redmond, Washington DC). All results are expressed as mean ±SD except as stated otherwise. p values under 0.05 ($p < 0.05$) were accepted statistically significant. T-test was used as the groups have parametric and independent conditions.

Results

Groups were similar according to age, body weight and gender (Table 1).

Table 1 Demographic properties (mean ± standard error)

Group	Inhalation (n = 10)	Intravenous (n = 10)	p
Age (year)	39.27 ± 8.45	40.27 ± 1.70	0.747
Body weight (kg)	68.64 ± 1.74	69.73 ± 3.3	0.778
Gender (f/m)	20/0	20/0	

Except for the pulse rates under anesthesia, there was no difference in the preanaesthetic and anaesthetic hemodynamic parameters of two groups ($p > 0.05$) (Table 2). The pulse rates difference under anesthesia was attributed to the bradycardic effects of anaesthetic agent that was used in intravenous group.

The cTnI levels were presented in Table 3. With $p > 0.05$, there was no difference in troponin levels between groups. As the value for the troponin levels between preanaesthetic/anaesthetic and anaesthetic/postanaesthetic was $p > 0.05$, there was no significant difference found (Table 4).

Discussion

We tried to determine the cardiac injury rate using cTnI blood levels through two different anesthesia techniques in preanaesthesic, anaesthetic, and postanaesthetic (9th hour) periods. Patients were selected from ASA-I group who have no cardiac risk factor, no drug usage, without metabolic and endocrine disorder. They were divided into the inhalation and intravenous group randomly. Demographically, no difference was found between two groups. Isofluran + N_2O were preferred because of minimal cardiac depressive effects and propofol + fentanyl for best suppression in stress response. Muscle relaxation was ensured by vecuronium bromide in both groups, which had no cardiac side effects. Anesthetic pulse rates were determined lower comparing the hemodynamic parameters of both groups. This contributed to the vagal effect of fentanyl. As arterial blood pressure and O_2 saturation were not different at the rare time, it was estimated that this effect did not influence cardiac hemodynamia. Troponin I blood samples collected in three periods was found in basal levels. There was no significant difference between the groups statistically.

Under physiological conditions, myocardium works to balance the O_2 supply and needs O_2, but in critical states such as anesthesia, myocardium exposes physiologic and pharmacologic distress. The increased O_2 need is compensated by tachycardia, increased contractility, and left ventricle end-diastolic pressure. As myocardium satisfies the O_2 need, in diastole this mechanism may cause ischemic attacks

Table 2 Hemodynamic parameters

Before induction			
Group	**Inhalation**	**Intravenous**	**P**
SBP (mm/Hg)	125.55 ± 4.36	128.18 ± 3.52	0.643
DBP (mm/Hg)	76.73 ± 2.17	76.73 ± 2.17	0.272
Pulse (/min)	82.00 ± 2.10	75.45 ± 2.71	0.071
O_2 Saturation (%)	96.55 ± 0.49	96.18 ± 0.48	0.604
Before surgery, after favorable anesthetic conditions			
Group	**Inhalation**	**Intravenous**	**P**
SBP (mm/Hg)	128.27 ± 5.68	131.91 ± 4.20	0.612
DBP (mm/Hg)	82.09 ± 1.81	80.91 ± 2.39	0.698
Pulse (/min)	88.00 ± 3.00	77.09 ± 3.06	0.019*
O_2 Saturation (%)	96.91 ± 0.34	97.00 ± 043	0.870

and myocardial injury. An anesthetic agent causes imbalance in this compensation mechanism. They may depress myocardium directly or changing the vascular tonus and myocardial contractility in the autonomic nervous system, changing cardiac rhythm-rate or hypoxia, hypercapnia and acidosis. Apart from anesthesia, cardiac load is increased by surgery, bleeding, infection, fever, and complications such as pulmonary emboli. Especially, among the older population, the incidence of ischemic heart disease in intensive care unit is higher. Developing surgery and anesthetic opportunities ensure more patients are operated upon and their mean lifetime increases. This means more patients with cardiac problems and anesthesia administration. Either in the absence of cardiac disease, anesthetic and surgical additional stress may cause failure ischemia (Coriat et al 1990).

The most common complication is myocardial necrosis and 3%–5% of patients are seen under risk. Many of them are silent subendocardial lesions, but in a shorter time, this may have life threatening effect on surgery patients. Cardiac evens usually occur after 48 hours from surgery. In the early postoperative period, increased left ventricle load and metabolic disorders were common, especially, the release of cathelocoliginergic mediators and increased hypercoaguability related to the effects of anesthesia on circulation and surgery stimulus. This change demolishes myocardial energy balance and causes left ventricular failure development. For this reason, in order to reduce mortality and morbidity related to cardiac complications, risks must be determined and measures must be taken (De Hert et al 1989, 2003; Lowenstein 1989; Priebe 2005; Weitz 2001).

Table 3 Cardiac troponin i levels (Normal range <0.4 ng/ml)

	Inhalation (ng/ml)	**Intravenous (ng/ml)**	**P**
Preanesthetic Troponin:	0.24 ± 0.03	0.21 ± 0.01	0.261
Anesthetic Troponin:	0.23 ± 0.02	0.20 ± 0.00	0.199
Postanesthetic Troponin:	0.21 ± 0.01	0.20 ± 0.01	0.206

Table 4 Comparison of troponin levels between groups

Groups	**P (Inhalation)**	**P (Intravenous)**
Preanesthetic-anesthetic:	0.692	0.343
Anesthetic-postanesthetic	0.476	0.406

In the past 20 years, creatinine kinase mb (CK-MB) isoenzyme, which has been issued as a marker for this purpose, may also be positive in traumatic injuries, renal insufficiency, strokes and mypathies, has not been informative. In diagnosis Wu et al suggest that after 6–24 hours later from the onset of acute myocardial infarction (AMI) CK-MB has great sensitivity but in 24–48 hours the sensitivity is reduced (Wu 1996; Benoit 2001). Although myoglubin is valuable in the early period, it is not specific to cardiac injury. Cummins et al (1987) reported cTnl releasing in AMI. Many acceptable studies were performed about the sensitivity and specify of cTnl (Adams et al 1990, 1993) Diagnosis and postoperative management is presently based on assessment of cTnI (Coriat 2001). The European and American Cardiology Societies (ESC/ACC 2000) have established cTnI as the gold standard for diagnosis of AMI and risk stratification for adverse cardiac events.

Experimental and clinical studies have extensively shown that all volatile anesthetic agents decrease myocardial loading conditions and contractility. Even the newer inhalational agents such as desflurane and sevoflurane seem to exhibit a similar dose-dependent depression of myocardial function (De Hert et al 2001). Those depressant effects decrease myocardial oxygen demand and may, therefore, have a beneficial role on the myocardial oxygen balance during myocardial ischemia. Recently, experimental evidence has demonstrated that in addition to these indirect protective effects, volatile anesthetic agents also have direct protective properties against reversible and irreversible ischemic myocardial damage (De Hert 2006).

As a result, general anesthesia is not a risk for MI to date, but eliminates risk factors and protection against cardiac hemodynamia.

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