Incidence and Risk Factors for Symptomatic Myocardial Ischemia After Posterior Decompressive Surgery for Degenerative Lumbar Disorders

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Research Article

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Abstract

Background: This study aimed to determine the incidence of symptomatic myocardial ischemia (PSMI) after posterior decompressive surgery for degenerative lumbar disorders and identify its risk factors.

Methods: 256 patients who underwent the posterior decompressive surgery and were discharged from the hospital between January 2011 and December 2016 were enrolled in this study, according to the inclusion and exclusion criteria. Subjects were divided into two groups by the occurrence of PSMI: the PSMI group and the N-PSMI group. Three sets of factors were analyzed to investigate potential risk factors for PSMI. Patient characteristics included age, gender, body mass index, smoking, drinking, venous thrombosis of lower extremities, comorbidities, visual analog scale, hemoglobin, electrolytes. Preoperative cardiac functional variables included electrocardiogram, ventricular ejection fraction, history of ischemic attack, previous cardiac surgery. Perioperative variables included surgical strategy, surgery time, blood loss, intraoperative aortic injury, intraoperative maximum heart rate, arterial blood pressure, postoperative temperature, daily liquid intake and output volume, electrolytes glucose, drug allergy, and postoperative visual analog scale. Univariate analyses followed by the multivariate logistic regression model were employed to identify the risk factors for PSMI.

Results: PSMI was developed in 23 of 256 patients (8.9%) before discharge. Univariate analyses revealed that patients with onset of PSMI were older, more likely to have undergone cardiac surgery, with less daily liquid intake volume, and less intravenous infusion ($p < 0.05$). Logistic regression analysis revealed that age > 52 years, heart rate > 81 Bpm, daily liquid intake < 2140 mL, and cardiac surgery history were independently associated with PSMI after posterior decompressive surgery for degenerative lumbar disorders.

Conclusions: The incidence of PSMI after posterior decompressive surgery for degenerative lumbar disorders was 8.9%; senior, fast heart rate, insufficient daily liquid intake, and cardiac surgery history are potential risk factors and should be carefully evaluated before an operation.

Background

Degenerative lumbar disorders (DSD), including lumbar disc herniation, lumbar stenosis, lumbar spondylolisthesis, are disabling [1]. Often accompanied by symptoms such as low back pain (LBP), lower extremity pain, and weakness, DSD lead to substantial years lived with disability (YLDs) [1, 2]. According to GBD 2019 Diseases and Injuries Collaborators, LBP was the leading cause of YLDs, responsible for 63.7 million YLDs, or 7.4% of total global YLDs in 2019 [3]. During the past decade, related YLDs has increased by 13.1%, indicating an increasing healthcare burden [3]. Due to a decrease in fertility rates and an increase in life expectancy, the global population is undergoing an upward shift in its age structure [4]. The increasing aging of the population poses unique challenges for the health care system and spinal physicians. On the one hand, this is due to the more prevalent age-related degenerative lumbar diseases; on the other hand, this is because the elderly population is often comorbid with multiple chronic conditions [4, 5]. Degenerative lumbar disorders have become an extensive reason to undergo lumbar spinal surgeries [1, 6]. However, the surgical treatment of DSD is not without risk of perioperative complications, particularly for those aging patients [5].

Postoperative cardiac events are frequent complications for patients undergoing various non-cardiac surgery [7], contributing to substantial morbidity and mortality and excessive hospital costs and length of stay [8–11]. Their incidence ranged from 1–16%, depending on the study population, the procedure types, and the definition of myocardial events [10, 12–14]. The clinical symptoms may vary from silent heart attacks with electrocardiogram abnormality alone to symptomatic myocardial ischemia with electrocardiogram abnormality, the elevation of isoenzyme, as well as severe clinical symptoms, such as chest pain, pressure within the anterior mid-chest, sometimes with radiation of pain to the neck, lower jaw, teeth, anns, or elbows [15]. For the patients with postoperative symptomatic myocardial ischemia (PSMI), most present no serious consequence, and the management could be not challenging. However, some cases may be life-threatening after onset, and their impact on the long-term quality of life should never be underestimated. Postoperative myocardial ischemia is associated with a 55% lower survival rate over the first five years postoperatively [16].

Multiple previous studies investigated the incidence and risk factors of myocardial ischemia after non-cardiac surgeries. In a cohort of 16,363 patients undergoing vascular surgery, the incidence of postoperative myocardial ischemia was 3%, with risk factors including procedure type and history of coronary artery disease [16]. In a prospective cohort of 2,018 patients undergoing 2,546 non-cardiac procedures, the incidence of acute myocardial infraction reached 5.8% [10]. Results from a large retrospective cohort of 30,339 patients who underwent spine surgery reported the incidence of perioperative cardiac complications (cardiac arrest or myocardial infarction) was 0.34% [11]. They found that old age, insulin-dependent diabetes, perioperative anemia, and history of cardiac diseases and treatments were risk factors for cardiac arrest or myocardial infarction [11]. To the best of our knowledge, few studies have investigated the incidence and risk factors of symptomatic myocardial ischemia (SMI) after posterior decompressive surgery for degenerative lumbar disorders. Therefore, the purpose of this study was to identify the incidence of postoperative symptomatic myocardial ischemia (PSMI) in-hospital after posterior decompressive surgery for degenerative lumbar diseases and to identify its risk factors. This study would like to provide references for spinal surgeons in decision-making and surgical planning for the most vulnerable patients.

Methods

Patients

This study is a retrospective study. The inclusion criteria included a) Degenerative lumbar disorders, including lumbar disc herniation, lumbar stenosis, lumbar spondylolisthesis; b) Posterior decompressive surgery with/without instrumented fusion; and c) Detailed and complete medical data, including history, comorbidity. The exclusion criteria included a) Patients treated for nondegenerative disorders, such as trauma, tumor, infection, inflammation, metabolic disease, rheumatoid arthritis; b) Patients treated with anterior or lateral lumbar fusion surgery, minimally invasive lumbar fusion surgery, or c) Non-cardiac-cause death in hospital. By retrieving the medical records from January 2011 to December 2016, 256 patients (152 female and 104 male) who met both the inclusion and exclusion criteria were retrospectively reviewed. The mean age was 51.8 ± 11.3 years (range from 26 to 78 years).

Clinical data collection and evaluation

Postoperative symptomatic myocardial ischemia in-hospital was defined as the combination of clinical symptom, Electrocardiogram abnormality, as well as elevation of isoenzyme, including creatine kinase-MB (CK-MB) isoenzyme, myoglobin (MYO), cardiac troponin I (cTnl), brain natriuretic peptide (BNP). Patients present with a broad spectrum of symptoms, including chest pain, radiative pain of the shoulder and back. Electrocardiogram abnormality includes either new Q waves in at least two leads that were 0.04 seconds in duration and 1 mm in depth, new ST segment depression of 1 mm or more, or new T-wave inversion. According to the occurrence of PSMI in-hospital, patients were divided into two groups: the PSMI group and the N-PSMI group.

To investigate risk factors for the occurrence of postoperative symptomatic myocardial ischemia, three sets of factors were analyzed: a) Patient characteristics include age, gender, body mass index (BMI), smoking, drinking, venous thrombosis of lower extremities, comorbidities (hypertension, diabetes, chronic obstructive pulmonary disease, immunosuppressive disorder, liver disease includes patients with a documented cirrhosis or chronic hepatitis, kidney disease, bleeding disorders), preoperative visual analog scale (VAS) for pain evaluation (back and leg pain), preoperative laboratory values of hemoglobin, electrolytes (K^+, Na^+, Ca^{2+}), and glucose. b) Preoperative cardiac functional variables include ECG, echocardiography (ventricular ejection fraction), history of ischemic attack (MI without current symptoms, stable angina, or unstable angina), previous cardiac surgery, including heart stent implantation, coronary bypass graft procedure (CABG). c) Perioperative variables include surgical strategy (decompressive surgery only or combined with instrumented fusion), surgery time, blood loss, intraoperative aortic injury, intraoperative maximum heart rate and arterial blood pressure (systolic and diastolic blood pressure), postoperative temperature, daily liquid intake (including intravenous infusion, oral intake) and output volume (including somatosensory evaporation, excreted by the kidney and intestinal discharge, wound drainage), electrolytes (K^+, Na^+, Ca^{2+}), glucose, drug allergy, postoperative VAS for incision, back and leg pain.

Statistical analysis

Data analyses were conducted using Statistical Product and Service Solutions software (version 13; SPSS, Chicago, IL). Continuous variables were described as mean ± standard deviation (SD), and categorical variables were expressed as frequency and percentages. Differences in patient demographics were determined by Student's t-test for continuous variables and chi-square or Fisher's exact test for dichotomous data as appropriate. Variables with *p* values smaller than 0.05 in the univariate analyses and several variables selected by experts entered into a multivariate logistic regression model. We computed the odds ratio (OR) with its 95% confidence interval (CI) for each variable. *P*-value of less than 0.05 was considered as statistically significant.

Results

Postoperative symptomatic myocardial ischemia was developed in 23 of 256 patients (8.9%) before discharge and was enrolled as the PSMI group. The other 233 patients who presented no postoperative symptomatic myocardial ischemia were enrolled as the N-PSMI group. The mean duration from immediate postoperative to the onset of PSMI was 3.4 days.

There was no statistically significant difference between the two groups in patient characteristics of gender, BMI, smoking, drinking, lower extremity venous thrombosis, comorbidities, VAS-back, VAS-leg, hemoglobin, electrolytes, glucose. However, subjects in the PSMI group were statistically older than those in the N-PSMI group (Table 1).

Table 1
Patient characteristics between the PSMI and N-PSMI group

Variables	**PSMI group**	**N-PSMI group**	***p* value**
	(n = 23)	(n = 233)	
Age, years (SD)	64.9 ± 8.9	50.5 ± 10.7	< 0.001
Sex, n (%)			0.550
Female	15(65.2)	137(58.8)	
Male	8(34.8)	96(41.2)	
BMI, Kg/m^2 (SD)	24.9 ± 1.7	24.5 ± 2.4	0.397
Smoking, n (%)			0.692
Yes	6(26.1)	57(24.5)	
No	17(73.9)	176(75.5)	
Drinking, n (%)			0.771
Yes	5(21.7)	51(21.9)	
No	18(78.3)	182(78.1)	
Lower extremity venous thrombosis, n (%)			NA
Yes	0(0)	2(0.9)	
No	23(100.0)	231(99.1)	
Comorbidities, n (%)			
Hypertension	16(69.6)	126(54.1)	0.154
Diabetes	9(39.1)	78(33.5)	0.585
Chronic obstructive pulmonary disease	2(8.7)	17(7.3)	0.683
Immunosuppressive disorder	1(4.3)	8(3.4)	0.577
Liver disease	1(4.3)	7(3.0)	0.534
Kidney disease	2(8.7)	5(2.1)	0.123
Bleeding disorders	0(0)	2(0.9)	-
VAS- back pain, points (SD)	4.9 ± 1.6	4.7 ± 1.6	0.695
VAS- leg pain, points (SD)	4.9 ± 1.6	4.7 ± 1.6	0.601

PSMI symptomatic myocardial ischemia, *BMI* body mass index, *VAS* visual analog scale, *mEq/L* milliequivalents per liter, *mmol/L* millimoles per liter, *SD* standard deviation

Variables	PSMI group	N-PSMI group	*p* value
Hemoglobin, g/L (SD)	123.0 ± 11.8	125.2 ± 8.8	0.287
Electrolytes			
K+, mEq/L (SD)	4.2 ± 0.5	4.3 ± 0.4	0.108
Na+, mEq/L (SD)	142.2 ± 2.9	143.1 ± 2.3	0.061
Ca+, mg/dL (SD)	2.3 ± 0.2	2.3 ± 0.1	0.272
Glucose, mmol/L (SD)	5.7 ± 1.1	5.8 ± 0.9	0.697

PSMI symptomatic myocardial ischemia, *BMI* body mass index, *VAS* visual analog scale, *mEq/L* milliequivalents per liter, *mmol/L* millimoles per liter, *SD* standard deviation

No statistically significant difference was observed for preoperative cardiac functional variables of ECG, ventricular ejection fraction, history of ischemic attack between the two groups. However, subjects in the PSMI group were more likely to have undergone cardiac surgery than their counterparts in the N-PSMI group (Table 2).

Table 2
Preoperative cardiac function between the PSMI and N-PSMI group

Variables	PSMI group	N-PSMI group	*p* value
	(n = 23)	(n = 233)	
ECG			0.779
Normal	18	190	
Myocardial ischemia	5	43	
Ventricular ejection fraction, percentage (SD)	66.4 ± 8.2	67.6 ± 9.6	0.577
History of ischemic attack			0.086
No	16	196	
MI without current symptoms	3	17	
Stable angina	2	12	
Unstable angina	2	8	
Previous cardiac surgery			< 0.001
No	11	217	
Heart stent implantation	5	12	
CABG	7	4	

PSMI symptomatic myocardial ischemia, *ECG* electrocardiogram, *MI* myocardial infarction, *CABG* coronary bypass graft procedure

There was no statistically significant difference between the two groups in perioperative variables of surgical strategy, surgery time, blood loss, intraoperative aortic injury, intraoperative maximum heart rate, and arterial blood pressure, postoperative temperature, electrolytes, Glucose, drug allergy, postoperative VAS for incision, back and leg pain, daily liquid oral intake, the liquid output volume of excreted by the kidney and wound drainage. However, the overall daily liquid intake and intravenous infusion were statistically smaller in the PSMI group than in the N-PSMI group (Table 3).

Table 3
Perioperative characteristics between the PSMI and N-PSMI group

Variables	**PSMI group**	**N-PSMI group**	***p* value**
	(n = 23)	(n = 233)	
Surgical strategy, n (%)			0.877
Decompressive surgery only	7(30.4)	53(22.7)	
TLIF	11(47.8)	85(36.5)	
PLIF	15(62.5)	95(40.8)	
Surgery time, minutes (SD)	152.6 ± 13.6	156.6 ± 18.4	0.310
Blood loss, mL (SD)	524.8 ± 71.4	546.1 ± 71.7	0.176
Intraoperative aortic injury, n (%)			NA
Yes	0(0)	0(0)	
No	23(100.0)	233(100)	
Intraoperative maximum heart rate, bpm (SD)	84.0 ± 9.7	80.6 ± 10.2	0.117
Systolic blood pressure, mmHg (SD)	130.0 ± 7.3	132.8 ± 8.7	0.141
Diastolic blood pressure, mmHg (SD)	81.0 ± 10.2	83.7 ± 10.9	0.256
Postoperative temperature, °C (SD)	37.6 ± 3.8	38.9 ± 2.0	0.745
Electrolytes			
K+, mEq/L (SD)	4.2 ± 0.4	4.1 ± 0.5	0.284
Na+, mEq/L (SD)	142.1 ± 2.7	141.7 ± 3.1	0.574
Ca+, mg/dL (SD)	2.3 ± 0.1	2.3 ± 0.2	0.245
Glucose	5.8 ± 1.1	5.8 ± 1.1	0.813
Drug allery, n (%)			0.315
Yes	1(4.3)	3(1.3)	
No	22(95.7)	230(98.7)	
VAS-incision pain, points (SD)	4.9 ± 1.8	4.6 ± 1.5	0.339
VAS-back pain, points (SD)	4.5 ± 1.4	4.7 ± 1.3	0.532
VAS-leg pain, points (SD)	4.4 ± 1.4	4.6 ± 1.5	0.556

PSMI symptomatic myocardial ischemia, *TLIF* transforaminal lumbar interbody fusion, *PLIF* posterior lumbar interbody fusion, *bpm* beats per minute, *VAS* visual analog scale

Variables	PSMI group	N-PSMI group	*p* value
Daily liquid intake volume, mL (SD)	1750.4 ± 149.2	2178.6 ± 170.9	< 0.001
Intravenous infusion	1234.8 ± 149.6	1646.1 ± 155.0	< 0.001
Oral intake	515.7 ± 71.6	532.5 ± 72.9	0.291
Daily liquid output volume, mL (SD)	1658.3 ± 90.3	1663.2 ± 91.1	0.809
Somatosensory evaporation	500 ± 0	500 ± 0	NA
Excreted by kidney	726.5 ± 93.1	734.9 ± 90.7	0.674
Intestinal discharge	300 ± 0	300 ± 0	NA
Wound drainage	131.8 ± 9.9	128.3 ± 11.1	0.141

PSMI symptomatic myocardial ischemia, *TLIF* transforaminal lumbar interbody fusion, *PLIF* posterior lumbar interbody fusion, *bpm* beats per minute, *VAS* visual analog scale

The following variables entered into the multivariate model: age, gender, BMI, smoking, drinking, venous thrombosis of lower extremities, comorbidities, preoperative VAS-back, VAS-leg, K^+, Na^+, Ca^{2+}, Glucose, ECG, ventricular ejection fraction, history of ischemic attack, previous cardiac surgery, surgical strategy, postoperative temperature, daily liquid intake, output volume, postoperative K^+, Na^+, Ca^{2+}, Glucose, drug allergy, postoperative VAS for incision, back and leg pain. When included in a binary logistic regression model, age > 52 years, heart rate > 81 Bpm, daily liquid intake < 2140 mL and cardiac surgery history were independently associated with the onset of postoperative symptomatic myocardial ischemia after posterior decompressive surgery for degenerative lumbar disorders (Table 4).

Table 4
Factors associated with PSMI after posterior decompressive surgery for degenerative lumbar disorders

Variable	Odds ratio [95% CI]	*p* value
Age	0.04 [0.006–0.253]	0.001
Heart rate	0.09 [0.019–0.443]	0.003
Daily liquid intake	4.88 [1.179–20.236]	0.029
Cardiac surgery history	44.09 [7.232–268.870]	< 0.001

PSMI symptomatic myocardial ischemia

Discussion

In the current study, most patients who underwent posterior decompressive surgery for degenerative lumbar disorders achieved satisfactory clinical outcomes. However, 8.9% of the patients experienced postoperative symptomatic myocardial ischemia, resulting in the decreased overall surgical outcome and patient satisfaction. Most of these events occurred within the first four days after surgery, with the risk declining sharply after the third postoperative day. Age > 52 years, heart rate > 81 Bpm, daily liquid intake < 2140 mL, and cardiac surgery history were independently associated with postoperative symptomatic myocardial ischemia after posterior decompressive surgery for degenerative lumbar disorders. These results were not confounded by other variables potentially associated with PSMI.

Due to the frequently degenerative nature of the lumbar spine, the afflicted population tends to be older, often with multiple medical comorbidities, such as hypertension, diabetes, cardio-cerebrovascular disease, pulmonary disease, etc. Even in the absence of comorbid conditions, older patients demonstrate diminished physiologic reservation compared to the younger counterparts, including cardiac, renal, pulmonary, and immunologic functions contributing to an increased vulnerability to external insults. General anesthesia is a routine method in posterior decompressive surgery for degenerative lumbar disorders. Intraoperative decreases in pressure occur almost universally during inhalation anesthesia because halothane, isoflurane, and enflurane all produce a dose-related depression of myocardial contractility. The compensatory ability for hemodynamic changes (hypotension, bradycardia, and decreased pulmonary capillary wedge pressure) is diminished in elderly patients. The hemodynamic changes induced by anesthesia induction and revival procedure, intraoperative mass blood loss, may cause myocardial infarction. In the current study, age older than 52 years is found to be a potential risk factor for myocardial infarction, partly consistent with Goldman's findings, in which age over 70 years was identified as a risk factor for postoperative myocardial infarction [17]. The ten-year difference in age between our study and Goldman may be due to the racial disparity and the difference in diagnosis included in the two studies. Nitroglycerin was proved to prevent intraoperative myocardial infarction by improving coronary arterial blood flow in 1980 [18]. However, intraoperative prophylactic use of nitroglycerin in high-risk patients may have negligible benefits or may even be harmful, leading to cardiovascular decompensation through decreased preload [19]. Several studies demonstrate that nitroglycerin administered as a prophylactic against myocardial infarction caused hemodynamic changes that may have led to an increased incidence of intraoperative myocardial infarction due to the altered hemodynamic responses [20, 21]. However, older patients should not be excluded from the benefit of surgery, given that their overall improvements of Oswestry Disability Index (ODI) and pain relief were equivalent to those of patients without postoperative myocardial infarction. Given the correlation of age and the risk of postoperative myocardial infarction, a thorough understanding of the interplay between postoperative myocardial infarction and surgical outcomes will be vital to optimizing clinical decision-making and appropriate patient counseling before surgery. For spinal surgeons and anesthesiologists, avoidance of significant hemodynamic changes during the perioperative period should be followed.

Previous studies have demonstrated that patients with prior myocardial infarction, particularly those who had an infarction within six months before surgery, are at increased risk for postoperative infarction. Those with angina alone were not [22–25]. For patients with pre-existing coronary artery disease undergoing non-cardiac surgery, the risk of cardiac complications is known to be higher [16, 26]. Most of the degenerative lumbar disorders do not need an emergency operation; comprehensive preoperative assessment of surgical and anesthesia risks, especially the cardiovascular and respiratory function, is necessary as time permits. Based on the understanding of the above point, patients with myocardial infarction or angina within six months before are not recommended to receive surgery unless in particular circumstances in our medical center. However, we did find that patients who received cardiac surgical intervention six months ago, including coronary artery bypass grafting (CABG) and percutaneous coronary intervention (PCI), assume a greater risk of postoperative myocardial infarction. There are two possible explanations for it. First, while at rest, the heart receives 4% of the cardiac output, although 10% of the systemic oxygen consumption is cardiac [27]. It results in a high degree of oxygen extraction across the coronary vascular bed. The heart increases its myocardial oxygen supply by increasing its oxygen extraction and increasing the total coronary blood flow. Under normal conditions, this increase can be up to five times the resting flow. Both the CABG and PCI are the recommended treatment methods for severe coronary artery disease involving multiple vessels or the left main stem; the patients always present poor cardiac function and lower compensatory capacity [28]. Second, revascularization with CABG and PCI are options for patients presenting with angina pectoris on optimal medical therapy. However, the repeat revascularization rate is 25.9% after PCI and 13.7% after CABG, both of which are not much higher. Delay of revascularization could result in irreversible loss of cardiac function. The surgical intervention could mechanically expand the narrowed coronary vessels but not improve vascular biological performance. Kappetein et al. [29] revealed that major adverse cardiac and cerebrovascular events (MACCE) were 26.9% in the CABG group and 37.3% in the PCI group after five years of follow-up. In other words, surgical intervention may temporarily relieve myocardial ischemia symptoms but not prevent the recurrence of myocardial infarction permanently.

The intraoperative blood loss varied from 300 mL to 1000 mL in the surgical treatment for degenerative lumbar disorders, depending on the surgery time, surgeon's experience, blood coagulation status, etc. Humans have a multitude of survival compensatory neuroendocrine mechanisms, which allow them to tolerate up to 30% of circulating blood volume loss before changes in traditional vital signs are evident, such as heart rate, blood pressure, respiratory rate, and oxygen saturation [30, 31]. Postoperatively, fluid resuscitation or blood transfusion should be implemented to maintain hemodynamic stability. The liquid intake and output volume need to be balanced. Gold JP et al. demonstrated that patients with larger hourly urine output did have a higher rate of ischemic complications, although this did not reach significance [32]. In the current study, the overall liquid output-intake difference of more than 800 mL was a potential risk factor for postoperative symptomatic myocardial ischemia.

In contrast, the amount of intraoperative blood loss is not. Postoperatively, if the liquid intake was not enough or the fluid output was excessive (use of mannitol or diuretic, somatosensory evaporation), the insufficient blood volume may induce a series of compensatory adaptations in the body, especially the increase of venous return and rapid heartbeat. Heart rate is an essential determinant of myocardial oxygen consumption: the faster the heart rate, the greater the amount of oxygen consumed per given time period. It is because the number of contractions per minute increased. Slogoff and Keats' showed a twofold increase in the frequency of ischemia associated with a heart rate above 100 bpm. By analyzing the ST segment trends, it appears that heart rate increase precedes ST depression, which suggests a cause-and-effect relationship [30]. Moreover, the autonomic nervous system plays a significant role in the pathophysiology of perioperative ischemia. There is evidence that sympathetic activation has an essential role in the onset of adverse cardiac events [30]. Adrenergic activity and plasma catecholamine levels change considerably in the postoperative period, which may predispose myocardial ischemia by altering the relationship between myocardial oxygen demand and supply [33].

There were several potential limitations in this study. First, this is a single-center study with a limited sample size. Only 256 patients were enrolled. Therefore, the study may be prone to type two error and underpowered to detect the significance of some potential risk factors. Thus, a multi-center large sample study is required. Second, only Chinese Han individuals were included in this study, and ethnic variation was not covered.

Conclusions

Our study revealed that older age (more than 52 years), fast heart rate (more than 81 Bpm), insufficient daily liquid intake (less than 2140 mL), and cardiac surgery history were potential risk factors for postoperative symptomatic myocardial ischemia after posterior decompressive surgery for degenerative lumbar disorders. Based on the above findings, patients of older age or with cardiac surgery history should be fully informed of the risk of postoperative symptomatic myocardial ischemia. It is essential for surgeons to control the heart rate and prevent insufficient daily liquid intake in postoperative patient management.

List Of Abbreviations

PSMI

symptomatic myocardial ischemia; Bpm:beats per minute; DSD:degenerative lumbar disorders; YLDs:years lived with disability; GBD:Global Burden of Diseases, Injuries, and Risk Factors Study; SMI:symptomatic myocardial ischemia; CK-MB:creatine kinase-MB; MYO:myoglobin; cTnl:cardiac troponin I, BNP:brain natriuretic peptide; BMI:body mass index; VAS:visual analog scale; CABG:coronary bypass graft procedure; SD:standard deviation; OR:odds ratio; CI:confidence interval; ODI:Oswestry Disability Index; PCI:percutaneous coronary intervention; MACCE:major adverse cardiac and cerebrovascular events; MI:myocardial infarction; TLIF:transforaminal lumbar interbody fusion; PLIF:posterior lumbar interbody fusion; ECG:electrocardiogram

Declarations

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Authors’ contribution

All authors contributed to drafting, revising the manuscript, and gave final approval of the version to be published. WJL designed the study, collected and analyzed the data, and drafted the initial manuscript. SSZ and YL took part in the interpretation of the results. HYH critically reviewed the manuscript for important intellectual content.

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Availability of data and materials

All data generated or analyzed during this study are included in this published article.

Ethics approval and consent to participate

The data did not contain any information that may disclose the privacy of patients. Analysis of data did not do any harm to the patients involved. The study did not have a commercial purpose. The study protocol was approved by the ethics committee of the First College of Clinical Medical Science, China Three Gorges University and Yichang Central People's Hospital. The informed consent from the participants was waived by the Institutional Review Board (IRB) of the First College of Clinical Medical Science, China Three Gorges University and Yichang Central People's Hospital.

Consent for publication

Not applicable.

Competing interests

The authors declare that they have no competing interests.

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References

1. Ravindra VM, Senglaub SS, Rattani A, Dewan MC, Härtl R, Bisson E, Park KB, Shrime MG. Degenerative Lumbar Spine Disease: Estimating Global Incidence and Worldwide Volume. Global Spine J. 2018, 8:784–94.

2. GBD 2019 Diseases and Injuries Collaborators. Global burden of 369 diseases and injuries in 204 countries and territories, 1990–2019: a systematic analysis for the Global Burden of Disease Study 2019. Lancet. 2020, 396:1204–22.
3. GBD 2019 Demographics Collaborators. Global age-sex-specific fertility, mortality, healthy life expectancy (HALE), and population estimates in 204 countries and territories, 1950–2019: a comprehensive demographic analysis for the Global Burden of Disease Study 2019. Lancet. 2020, 396:1160–203.
4. Fehlings MG, Tetreault L, Nater A, Choma T, Harrop J, Mroz T, Santaguida C, Smith JS. The Aging of the Global Population: The Changing Epidemiology of Disease and Spinal Disorders. Neurosurgery. 2015, 77 Suppl 4:S1–5.
5. Mannion AF, Fekete TF, Porchet F, Haschtmann D, Jeszenszky D, Kleinstück FS. The influence of comorbidity on the risks and benefits of spine surgery for degenerative lumbar disorders. Eur Spine J. 2014, 23 Suppl 1:S66–71.
6. Caruso R, Pesce A, Martines V, Wierzbicki V, Piccione E, Paolini S, Lanciano T. Assessing the real benefits of surgery for degenerative lumbar spinal stenosis without instability and spondylolisthesis: a single surgeon experience with a mean 8-year follow-up. J Orthop Traumatol. 2018, 19:6.
7. Sellers D, Srinivas C, Djaiani G. Cardiovascular complications after non-cardiac surgery. Anaesthesia. 2018, 73 Suppl 1:34–42.
8. Douleh DG, Yarlagadda M, Shen MS, Williams G, Mousavi I, Sethi MK. Drivers of hospital length of stay in 56,000 orthopaedic trauma patients: The impact of postoperative cardiac events. J Clin Orthop Trauma. 2017, 8:45–9.
9. van Waes JA, Nathoe HM, de Graaff JC, Kemperman H, de Borst GJ, Peelen LM, van Klei WA. Myocardial injury after noncardiac surgery and its association with short-term mortality. Circulation. 2013, 127:2264–71.
10. Puelacher C, Lurati Buse G, Seeberger D, Sazgary L, Marbot S, Lampart A, Espinola J, Kindler C, Hammerer A, Seeberger E, *et al*. Perioperative Myocardial Injury After Noncardiac Surgery. Circulation. 2018, 137:1221–32.
11. Bovonratwet P, Bohl DD, Malpani R, Haynes MS, Rubio DR, Ondeck NT, Shultz BN, Mahal AR, Grauer JN. Cardiac Complications Related to Spine Surgery: Timing, Risk Factors, and Clinical Effect. J Am Acad Orthop Surg. 2019, 27:256–63.
12. Badner NH, Knill RL, Brown JE, Novick TV, Gelb AW. Myocardial infarction after noncardiac surgery. Anesthesiology. 1998, 88:572–8.
13. Lopez-Jimenez F, Goldman L, Thomas EJ, Kuntz KM, Sacks DB, Lee TH. Predictive value of creatine kinase (CK)-MB for diagnosis of acute myocardial infarction after major noncardiac surgery. Arch Med Res. 1998, 29:33–7.
14. Grobben RB, van Waes JA, van Klei WA, Nathoe HM. Asymptomatic myocardial infarction after non-cardiac surgery; importance of routine testing of troponin concentration. Ned Tijdschr Geneeskd. 2013, 157:A5915.

15. Leaman DM, Davis D. Diagnosis and management of myocardial ischemia in the postoperative period. Surg Clin North Am. 1983, 63:1081–9.
16. Simons JP, Baril DT, Goodney PP, Bertges DJ, Robinson WP, Cronenwett JL, Messina LM, Schanzer A. The effect of postoperative myocardial ischemia on long-term survival after vascular surgery. J Vasc Surg. 2013, 58:1600–8.
17. Goldman L, Caldera DL, Southwick FS, Nussbaum SR, Murray B, O'Malley TA, Goroll AH, Caplan CH, Nolan J, Burke DS, *et al*. Cardiac risk factors and complications in non-cardiac surgery. Medicine (Baltimore). 1978, 57:357–70.
18. Abrams J. Nitroglycerin and long-acting nitrates. N Engl J Med. 1980, 302:1234–7.
19. Jacobs AK, Kushner FG, Ettinger SM, Guyton RA, Anderson JL, Ohman EM, Albert NM, Antman EM, Arnett DK, Bertolet M, *et al*. ACCF/AHA clinical practice guideline methodology summit report: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. J Am Coll Cardiol. 2013, 61:213–65.
20. Mutch WA, Culligan JD, Cote DD, Thomson IR. Hemodynamic effects of intravenous nitroglycerin: importance of the delivery system. Anesth Analg. 1982, 61:927–32.
21. Moon HS, Lee SK, Choi YS, In CB, Choi EJ. The effect of nitroglycerin on hemodynamic changes during laparoscopic low anterior resection. Korean J Anesthesiol. 2011, 61:388–93.
22. von Knorring J. Postoperative myocardial infarction: a prospective study in a risk group of surgical patients. Surgery. 1981, 90:55–60.
23. Narducci ML, Pelargonio G, Rio T, Leo M, Di Monaco A, Musaico F, Pazzano V, Trotta F, Liuzzo G, Severino A, *et al*. Predictors of postoperative atrial fibrillation in patients with coronary artery disease undergoing cardiopulmonary bypass: a possible role for myocardial ischemia and atrial inflammation. J Cardiothorac Vasc Anesth. 2014, 28:512–9.
24. Aldweib N, Negishi K, Hachamovitch R, Jaber WA, Seicean S, Marwick TH. Impact of repeat myocardial revascularization on outcome in patients with silent ischemia after previous revascularization. J Am Coll Cardiol. 2013, 61:1616–23.
25. Dorr R, Thiele H. Diagnostics and therapy of chronic myocardial ischemia: the when and how of diagnostic work-up and therapy. Herz. 2013, 38:327–8.
26. Ashton CM, Petersen NJ, Wray NP, Kiefe CI, Dunn JK, Wu L, Thomas JM. The incidence of perioperative myocardial infarction in men undergoing noncardiac surgery. Ann Intern Med. 1993, 118:504–10.
27. Zelis R, Liedtke JA, Leaman DM, Babb JD, Roberts BH. Angina pectoris. Diagnosis and treatment. Postgrad Med. 1976, 59:179–88.
28. Mohr FW, Morice MC, Kappetein AP, Feldman TE, Stahle E, Colombo A, Mack MJ, Holmes DJ, Morel MA, Van Dyck N, *et al*. Coronary artery bypass graft surgery versus percutaneous coronary intervention in patients with three-vessel disease and left main coronary disease: 5-year follow-up of the randomised, clinical SYNTAX trial. Lancet. 2013, 381:629–38.

29. Kappetein AP, van Mieghem NM, Head SJ. Revascularization options: coronary artery bypass surgery and percutaneous coronary intervention. Cardiol Clin. 2014, 32:457–61.
30. Chan GS, Middleton PM, Lovell NH. Photoplethysmographic variability analysis in critical care–current progress and future challenges. Annu Int Conf IEEE Eng Med Biol Soc. 2011, 2011:5507–10.
31. Cottingham CA. Resuscitation of traumatic shock: a hemodynamic review. AACN Adv Crit Care. 2006, 17:317–26.
32. Charlson ME, MacKenzie CR, Gold JP, Ales KL, Topkins M, Fairclough GJ, Shires GT. The preoperative and intraoperative hemodynamic predictors of postoperative myocardial infarction or ischemia in patients undergoing noncardiac surgery. Ann Surg. 1989, 210:637–48.
33. Longhitano S, Coriat P, Agro F. Postoperative myocardial infarction: pathophysiology, new diagnostic criteria, prevention. Minerva Anestesiol. 2006, 72:965–83.