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Review Article

Oxaliplatin-Induced Sinusoidal Obstruction Syndrome: Liver Stiffness Measurement as a Novel Predictor by Elastography

Kazumi Fujioka*

Department of Radiology, Nihon University School of Medicine, Tokyo, Japan

ABSTRACT

Oxaliplatin is an essential component of many chemotherapies protocol for colorectal cancer and colorectal liver metastasis. The use of oxaliplatin in patients with colorectal cancer has been associated with the occurrence of the vascular hepatic lesions such as Sinusoidal Obstruction Syndrome (SOS), Nodular Regenerative Hyperplasia (NRH), and Focal Nodular Hyperplasia (FNH). It is known that SOS impairs liver function and increases morbidity and mortality. Based on the comparison between Liver Stiffness Measurement (LSM) and splenic volume index, recent study indicated that measurement of elasticity using shear wave elastography may noninvasively predict oxaliplatin-induced hepatotoxicity. The recent report also provided the usefulness of MR elastography (MRE) in diagnosing SOS and determining SOS severity without contrast materials in patients with colorectal cancer. In this article, current knowledge and trends of oxaliplatin-induced SOS along with a new indicator, LSM using Ultrasonic (US) elastography and MR elastography have been reviewed. In addition, the significance and role of bevacizumab, VEGF (Vascular Endothelial Growth Factor) inhibitor have been described in oxaliplatin-based chemotherapy. As oxaliplatin is a backbone drug of many regimens for colorectal cancer and colorectal liver metastasis, oxaliplatin-based chemotherapy putatively induces SOS, NRH, and FNH. The assessment of LSM using US elastography and MR elastography may noninvasively show a predictive indicator for oxaliplatin-induced SOS. Though an association between VEGF inhibitor and atherosclerosis status has been suggested, bevacizumab may contribute to protect against the development of oxaliplatin-induced SOS.

Keywords: Oxaliplatin-induced SOS; Liver stiffness measurement; Shear wave elastography; Magnetic resonance elastography; VEGF inhibitor

INTRODUCTION

Oxaliplatin is an essential component of many chemotherapies protocol for colorectal cancer and colorectal liver metastasis. The use of oxaliplatin in patients with colorectal cancer has been associated with the development of the vascular hepatic lesions such as Sinusoidal Obstruction Syndrome (SOS), Nodular Regenerative Hyperplasia (NRH), and Focal Nodular Hyperplasia (FNH) [1,2]. It is known that SOS impairs liver function and increases morbidity and mortality [3]. According to the previous report, several studies have demonstrated that increased splenic volume showed the independent indicator of oxaliplatin-induced SOS [4]. While SOS condition is also induced by Hematopoietic Stem Cell Transplantation (HSCT). To evaluate the Liver Stiffness Measurement (LSM), namely elasticity, elastography has been provided for diagnosis of SOS after HSCT suggesting that this procedure can assist diagnosis of SOS [5,6]. The recent study indicated that the measurement of elasticity using shear wave elastography may noninvasively predict oxaliplatin-induced hepatotoxicity based on the comparison between liver stiffness measurement and splenic volume index [7]. The recent report also provided that MRE is a useful method in diagnosing SOS and determining SOS severity without contrast materials in patients with colorectal cancer [8].

Correspondence to: Kazumi Fujioka, Department of Radiology, Nihon University School of Medicine, 30-1 Oyaguchi-kamicho Itabashi-ku, Tokyo, Japan, E-mail: spbk2xq9@ninus.ocn.ne.jp

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In this article, current knowledge and trends of oxaliplatin-induced SOS along with LSM as a new predictor using elastography have been reviewed. Additionally, the author has described the significance and role of bevacizumab, VEGF inhibitor in oxaliplatin-based chemotherapy setting.

LITERATURE REVIEW

Oxaliplatin-based chemotherapy for colorectal liver metastasis

Recent report has suggested that Colorectal Cancer (CRC) is the third most common cancer. As the liver metastasis developed in about half of CRC cases, a trending of the increased survival rates in patients with Colorectal Liver Metastasis (CRLMs) has been shown due to more effective chemotherapy regimens and new surgical strategies [9]. The US Food and Drug Administration approved the use of oxaliplatin for CRLMs in 2002 [10]. The previous study in the adjuvant treatment of colon cancer trial demonstrated significantly higher disease-free survival and overall survival rates due to the adding oxaliplatin to in fusional FU plus leucovorin [11]. Oxaliplatin-based chemotherapy has been considered as the popular adjuvant therapy for colon cancer [12]. It is known that the typical systemic chemotherapy includes intravenous 5-FU, a fluoropyrimidine with either oxaliplatin or irinotecan (FOLFOX or FOLFIRI regimens) [9]. Oxaliplatin therapy contributes to the reduction of tumor progression by suppressing synthesis of DNA in cancer cells and impairing cell division and growth. Molecular factors including CHK2, SIRT1, c-Myc, LATS2, and FOXC1 have been regarded as regulators of oxaliplatin response in CRC, while the non-coding RNAs effect as master regulator of other molecular pathways in modulating the resistance of oxaliplatin [13].

Oxaliplatin-induced hepatic sinusoidal obstruction syndrome

It is known that chemotherapy-induced hepatopathies include steatosis, steatohepatitis, and SOS [14]. Whereas previous studies have provided benign regenerative nodules as pseudometastatc liver tumors as a late presentation of SOS in children with malignant tumors after high-dose chemotherapy or undergoing HSCT [15,16]. It is known that SOS impairs liver function and increases morbidity and mortality [3]. The use of oxaliplatin in patients with colorectal cancer has been associated with the occurrence of the vascular hepatic lesions such as SOS, NRH, and FNH [1,2]. The author previously reported a case of FNH of the liver accompanied by a marginal hypoechoic zone in the Ultrasonography (US), based on the comparison of the US and pathological features and recently described the trends in focal nodular hyperplasia of the liver along with oxaliplatin-induced this entity [17,18]. In addition, the association between oxaliplatin and NRH development has been shown [19]. Previous report described that the changes of SOS and related local disturbance in hepatic perfusion may cause the occurrence of NRH suggesting that this disturbance may also lead to the development of FNH [18,20]. As oxaliplatin is used as the essential part of chemotherapeutic regimen for CRLMs, oxaliplatin-induced hepatic injury has become a significant concern in patient with metastatic CRC [21]. Several reports described the oxaliplatin-induced SOS in patients with colon and gastric cancers [21,22]. It is known that pathophysiology of SOS is induced by toxic injury to the endothelium of the liver sinusoids [3]. Previous report described that chemotherapy contributes to cause loss of sinusoidal wall integrity with the extravasation of erythrocytes within the Disse space and endothelial cell exfoliation. In result, portal hypertension, liver dysfunction, and destruction of the liver parenchyma with nodular regeneration were observed [20]. Three mechanisms for sinusoidal damage induced by oxaliplatin have been suggested in detail [23]. Firstly, oxaliplatin causes increased porosity of the sinusoidal endothelium and increased cellular fenestrations, leading to the migration of the erythrocyte into the Disse space and formation of perisinusoidal fibrosis. Secondly, NRH easily tends to develop due to the chronic hypoxia. Finally, oxaliplatin generates an obliteration of blood capillaries and areas of parenchymal extinction. Whereas it has been suggested that pathogenesis of oxaliplatin-induced SOS may include oxidative stress, inflammatory damage, liver fibrosis, and platelet aggregation and adhesion [24]. It is well known the relationship between the hepatic steatosis and SOS and oxaliplatin. Additionally, the peripheral neuropathy, liver dysfunction, splenomegaly, and thrombocytopenia are considered as side effects associated with oxaliplatin-induced SOS [23].

The liver stiffness measurement as a novel predictor by US elastography and MR elastography for oxaliplatin-induced SOS.

The predictive parameters for oxaliplatin-induced SOS included the EOB-MRI features, the spleen volume index, hyaluronic acid level, ICG-R15, and AST level [22]. SOS was shown to be related to increased short-term postoperative complications such as blood transfusion, therefore early diagnosis of oxaliplatin-induced SOS is clinically important. Liver biopsy is invasively gold standard tool for the detection of SOS, while splenic volume is a noninvasive parameter of oxaliplatin-induced hepatotoxicity. According to the previous report, several studies have demonstrated that increased splenic volume showed the independent indicator of oxaliplatin-induced SOS [4]. Recent report suggested an increased SV in 81% of the patients using oxaliplatin-based chemotherapy [25]. However, it is suggested that ultrasound modality does not always provide accurate evaluation of the splenic volume. Meanwhile, SOS condition is also induced by HSCT. To evaluate the LSM, elastography has been provided for the diagnosis of SOS after HSCT. Several studies have demonstrated that HSCT recipients developed SOS showing higher LSM by shear wave and transient elastography, suggesting that this procedure can contribute to the diagnosis of SOS after HSCT [5,6]. SOS after HSCT induces a hepatotoxicity in sinusoidal endothelial cells leading to portal hypertension from a pathophysiological viewpoint suggesting that meta-analysis revealed the close association between LSM and hepatic vein pressure gradient.

Oki et al. previously indicated that LSM by US elastography was useful for the detection of oxaliplatin-induced hepatic injury [26]. Recent studies also suggested the usefulness of LSM using

US elastography for oxaliplatin-induced SOS in patients with gastrointestinal cancer [7,27]. Based on the comparison between LSM and splenic volume index, recent study indicated that measurement of elasticity using shear wave elastography may noninvasively predict oxaliplatin-induced hepatotoxicity [7]. Another report suggested that LSM using shear wave elastography may be a diagnostic parameter for oxaliplatin-induced SOS [27]. Meanwhile, Real-Time Tissue elastography (RTE) namely, strain elastography have been also widely used in diagnosing for the superficial regions such as breast, thyroid, testis, and skin lesions. In dermatologic field, the author previously reported that RTE images contribute the accurate diagnosis for unruptured epidermal cyst and superficial fibromatoses [28,29]. With respect to MRI features, reticular hypo-intensity of hepatobiliary phase on gadoxetate/gadoxetic acid (Gd-EOB-DTPA) enhanced MRI is a characteristic feature for the diagnosing SOS [30]. Regarding MRE features, MRE as a phase-contrast MRI technique represents the elasticity of tissues by analyzing the mechanical wave propagation in the tissues showing the detection and staging of the liver fibrosis [8,31]. It is known that liver parenctymal stiffness is dependent on tissue composition such as organizing and vascular components, interstitial pressure, and the pathological status. Destruction of parenchyma, decreased remodeling of Extracellular Matrix (ECM), and distortion of the parenctymal architecture result in the development of the liver stiffness. It is known that inflammation and increased portal venous pressure as other pathologic processes accompanied by fibrosis may also contribute to increased liver stiffness [31]. By using MRE, the prediction of HCC and varices developments, decompensation status, and differentiation of NAFLD and NASH have been studied [31]. The recent report provided that MRE is also useful in diagnosing SOS and determining SOS severity without contrast materials in patients with colorectal cancer [8]. While mapping methods showing the accurate determination of tissue properties by T1 and T2 relaxation times of tissues (T1 and T2 mapping) have been developed [32]. Based on the evidence, LSM using US elastography and MR elastography may noninvasively be a good indicator for SOS. Further study is needed to verify for LSM using elastography in accurate diagnosis of oxaliplatin-induced SOS.

Association between VEGF inhibitor and atherosclerosis status

VEGF produced by cancer cells induces angiogenesis and proliferation for the extension of cancer leading to the proliferation and metastasis of tumor tissue in the microenvironment of cancer. VEGF expression is recognized in many types of cancers such as colorectal cancer, gastrointestinal cancer, liver cancer, and breast cancer, hence angiogenic inhibitors for cancer therapy have been developed. Bevacizumab, as the representative anti-angiogenic agent has been developed as an anti-VEGF human monoclonal antibody and has contributed to the effective treatment for the colorectal cancer. Whereas the angiogenesis inhibitors mainly effect on the vascular endothelial cell and induce vasoconstriction due to the reduction of the vasodilators such as NO and PGI_2 and increased ET-1 leading to the vascular endothelial dysfunction and plaque formation. In result, drug-induced atherosclerosis including hypertension and thrombosis/atherosclerosis has been developed [33,34]. Endothelial dysfunction is the first step for atherosclerosis condition. I will suggest the assessment of vascular endothelial and smooth muscle cell function using Flow-Mediated Vasodilation (FMD) and Nitroglycerin-Mediated Vasodilation (NMD) procedures for assessment of atherosclerosis status in the setting of oxaliplatin-based chemotherapy [35-37]. The clinical study described that in addition to oxaliplatin-based chemotherapy, bevacizumab, VEGF inhibitor decreased the frequency of splenic enlargement and the development of thrombocytopenia [38]. Regarding the animal study, the previous study provided the evidence on the protection effect of VEGF-inhibition against the development of oxaliplatin induced SOS in mice [39]. Though bevacizumab, VEGF inhibitor may induce atherosclerosis status, it may contribute to the protection of the development of SOS. Further study is needed to validate for the optimal treatment of cancer and the effective management.

DISCUSSION

Previous report described that chemotherapy contribute to cause loss of sinusoidal wall integrity with the extravasation of erythrocytes within the Disse space and endothelial cell exfoliation. In result, portal hypertension, liver dysfunction, and destruction of the liver parenchyma with nodular regeneration were observed [20]. The predictive parameters for oxaliplatin-induced SOS included the EOB-MRI features, the spleen volume index, hyaluronic acid level, ICG-R15, and AST level [22]. According to the previous report, several studies have demonstrated that increased splenic volume showed the independent indicator of oxaliplatin-induced SOS [4]. Recent report suggested an increased SV in 81% of the patients using oxaliplatin-based chemotherapy [25]. However, it is suggested that ultrasound modality does not always provide accurate evaluation of the splenic volume. While SOS condition is also caused by HSCT. To evaluate the LSM, elastography has been provided for the diagnosis of SOS after HSCT. Several studies have provided that HSCT cases developed SOS showing higher LSM by shear wave elastography suggesting that this procedure can assist diagnosis of SOS [5,6]. Based on the comparison between LSM and splenic volume index, recent study indicated that measurement of elasticity using shear wave elastography may noninvasively predict oxaliplatin-induced hepatotoxicity [7]. Another report suggested that LSM using shear wave elastography may be a diagnostic parameter for oxaliplatin-induced SOS [27]. By using MRE, the prediction of HCC and varices developments, decompensation status, and differentiation of NAFLD and NASH have been studied [31]. The recent report also provided the usefulness of MRE procedure in diagnosing SOS and determining SOS severity without contrast materials in patients with colorectal cancer [8]. Based on the evidence, LSM using US elastography and MR elastography may noninvasively be a good indicator for oxaliplatin-induced SOS. Meanwhile, VEGF expression is recognized in many types of cancers such as colorectal cancer and gastrointestinal cancer, therefore angiogenic inhibitors for cancer therapy have been developed. Bevacizumab, anti-angiogenic

agent has been developed as an anti-VEGF human monoclonal antibody and has contributed to the effective treatment for the colorectal cancer. Whereas the angiogenesis inhibitors mainly effect on the vascular endothelial cell and induce vasoconstriction leading to the vascular endothelial dysfunction and plaque formation. In result, drug-induced atherosclerosis including hypertension and thrombosis/atherosclerosis has been developed [33,34]. The clinical study showed that the addition of bevacizumab to oxaliplatin-based chemotherapy decreased the frequency of splenic enlargement and the development of thrombocytopenia [38]. Though bevacizumab, anti-angiogenetic agents may induce atherosclerosis status, it may contribute to the protection of the development of SOS. In this article, the current knowledge and trends of oxaliplatin-induced SOS along with a new indicator, LSM using elastography has been reviewed in detail. In addition, the significance and role of bevacizumab, VEGF inhibitor have been described. As oxaliplatin is a backbone drug of many regimens for colorectal cancer and colorectal liver metastasis, oxaliplatin-based chemotherapy putatively induces SOS, NRH, and FNH [39]. The assessment of liver stiffness measurement using shear wave elastography and MRE may show noninvasively predictive indicator for oxaliplatin-induced SOS. Further study is needed to verify for LSM as a new predictor using elastography in accurate diagnosis of oxaliplatin-induced SOS. Clinically, bevacizumab, anti-angiogenetic agents may contribute to the protection of the development of SOS. Although an association between VEGF inhibitor and atherosclerosis status has been suggested. Further research is needed to validate for the optimal treatment of cancer and the effective management in the oxaliplatin-based chemotherapy.

CONCLUSION

As oxaliplatin is an essential component of many chemotherapies protocol for colorectal cancer and colorectal liver metastasis, oxaliplatin-based chemotherapy probably induces sinusoidal obstruction syndrome, nodular regenerative hyperplasia, and focal nodular hyperplasia. The assessment of liver stiffness measurement using shear wave elastography and MR elastography may noninvasively show a novel predictor for oxaliplatin-induced sinusoidal obstruction syndrome. Though a relationship between VEGF inhibitor and atherosclerosis condition has been shown, bevacizumab may contribute to the protection of the development of oxaliplatin-induced sinusoidal obstruction syndrome.

CONFLICT OF INTEREST

Author declares that I have no conflicts of interest.

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None

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