

Splanchnic Venous Thrombosis in Acute Pancreatitis: Does Anticoagulation Affect Outcome?

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Abstract

Background: Splanchnic venous system thrombosis is a well recognized local vascular complication of acute pancreatitis (AP). It may involve thrombosis of splenic vein (SplV), portal vein (PV) and superior mesenteric vein (SMV), either separately or in combinations, and often detected incidentally, indeed some cases present with upper gastrointestinal bleed, bowel ischemia and hepatic decompensation. Incidence is variable depending on study subjects and diagnostic modalities. Pathogenesis is multifactorial centered on local and systemic inflammation. Management involves treatment of underlying AP and its complications. Universal use of anticoagulation may lead to increased risk of bleeding due to frequent need of interventions (radiologic/endoscopic/surgical). Literature on anticoagulation in setting of AP is sparse and at present there is no consensus guideline on it. Current article details our experience on splanchnic venous thrombosis (SVT) in AP in a well defined cohort of patients at a tertiary care center.

Methods: Hospitalized patients with AP from January 2018 to December 2018 were included in the study. Detailed information on demographic, clinical, laboratory, radiologic features, and indication of anticoagulation use were collected prospectively during the index admission. Outcome variables were analyzed at the end of 6 months.

Results: Twenty four out of 105 (22.85%) patients with AP develop SVT. Etiology of AP was alcohol use in 21/24 (87.5%) subjects. Most common vessel involved was isolated SplV in 11/24 (45.8%) patients followed by SplV along with PV and SMV 9/24 (37.50%, $P < 0.001$). Bowel ischemia 4/12 (33.3%), hepatic decompensation 3/12 (25%), triple vessel involvement 4/12 (33.3%) and pulmonary embolism 1/12 (8.3%) were reasons for anticoagulation. There was no statistical

difference with respect to development of varices, collateral formation, recanalization, bleeding and mortality with use of anticoagulation ($P > 0.05$ with respect to all above variables).

Conclusions: SVT is commonly seen in alcohol-induced AP. Anticoagulation does not affect outcomes of SVT. Subset of patients may benefit with anticoagulation.

Keywords: Acute pancreatitis; Splanchnic venous thrombosis; Anticoagulation; Bleeding; Recanalization

Introduction

Acute pancreatitis (AP) is an acute inflammatory process of the pancreas with variable clinical presentations. It is one of the leading causes of hospitalization. In 80-85% patients AP is a mild self-limiting disease without need for specific interventions, while 20-15% patients develop moderate to severe disease with associated local and or systemic complications [1, 2]. Splanchnic venous system thrombosis is a well recognized local vascular complication of AP [3, 4]. It may involve thrombosis of splenic vein (SplV), portal vein (PV) and superior mesenteric vein (SMV), either separately or in combinations. It is often detected incidentally on imaging performed for evaluation of symptoms and/or complications of AP. However, splanchnic venous thrombosis (SVT) may present with hepatic decompensation due to PV occlusion, small bowel ischemia due to SMV occlusion, and upper gastrointestinal bleed from gastroesophageal varices due to SplV and or PV thrombosis [3, 5]. Incidence of SVT in AP ranges from as low as 1.8% to as high as 36.5% [6-9]. Several factors contribute in genesis of SVT, such as direct intimal injury due to inflammation and cellular infiltration, compression by pancreatic/peripancreatic collections leading to venous stasis, systemic activation of hemostasis and hypercoagulable state [9-11]. Management of SVT usually involves treatment of underlying AP and its complications. Spontaneous recanalization is seen in 30% cases with expectant treatment [12]. Universal use of anticoagulation (AC) may not be always beneficial because of frequent need of interventions in setting of AP and therefore the inherent associated risk of bleeding [9]. At present, there is no consensus guideline for use of AC in SVT in the setting of AP. However, the limited

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literature on this subject has suggested the use of AC in presence of PV and/or SMV thrombosis due to risk of hepatic decompensation and bowel ischemia respectively [13]. Currently there is paucity of literature on incidence, associated risk factors, use of AC, and outcome of SVT in the setting of AP. The aims of this study are to look into clinical features and outcome of SVT with AC in a well defined cohort of AP patients at a tertiary care center.

Materials and Methods

The study was conducted at a tertiary care referral center, Topiwala National Medical College and B.Y.L. Nair Charitable Hospital, Mumbai. Institutional Ethics Committee permission was taken for proposed study protocol.

Patient cohort

Eligible patients with AP, as defined by the revised Atlanta criteria [14], hospitalized at our institute from January 2018 to December 2018 were included in the study.

Exclusion criteria

Those patients were excluded from the study: 1) Chronic pancreatitis; 2) Recurrent AP or past history of pancreatitis; 3) Pancreatic or other malignancy; 4) Pregnancy; 5) Chronic liver disease; 6) Age less than 18 years; 7) Unwillingness to provide consent; 8) Intra-abdominal infection; 9) If follow-up information was unavailable.

Data collection

Detailed information on demographic, clinical, laboratory features and radiographic parameters were collected prospectively during the index admission. Data on severity assessment of AP such as, modified computed tomography Index (mCTSI), systemic inflammatory response syndrome (SIRS), revised Atlanta classification system, bedside index of severity in acute pancreatitis score (BISAP), acute physiology and clinical health evaluation score-II (APACHE II) and local and systemic complications were noted. Organ failure defined according to modified Marshall score [15], need for intensive care unit (ICU) admission, indications and type of interventions (radiologic/endoscopic/surgical), type and dose of AC and in-hospital mortality were recorded.

Assessment of SVT

Diagnosis of SVT was based on findings of pancreatic protocol contrast-enhanced computed tomography (CECT), magnetic resonance imaging (MRI), and/or colour Doppler ultrasonog-

raphy of abdomen. SVT was diagnosed when an actual thrombus was detected in the vein or the vein appeared compressed or was not visualized with the presence of collaterals [9, 13]. Portal cavernoma was defined radiologically as the presence of large portoportocollaterals [16].

Follow-up and outcome of SVT

Follow-up information was collected from outpatient department (OPD) records. All patients with SVT were followed up for 6 months. Specific clinical information included duration of AC, type of AC, bleeding, resolution of thrombosis, development of varices, collaterals and portal cavernoma were noted. Follow-up testing/imaging was performed at the disposition of primary care physician and no additional testing was done for study purpose.

Statistical analysis

For continuous variables mean, standard deviation and range, while for categorical variables frequencies and percentages were calculated. Comparisons were made between SVT and non-SVT groups; also between AC and non-AC groups. Independent *t*-test and Chi-square test were used for comparison of continuous variables and categorical variables respectively. *P* value of < 0.05 was set as level of significance.

Results

Out of 105 patients with AP, 24 (22.85%) patients developed SVT. Mean age was 36.62 ± 6.49 and 41.56 ± 13.85 years, with body mass index (BMI) of 20.03 ± 2.25 and 22.49 ± 4.72 kg/m² in SVT and non-SVT groups respectively. There were 19 (79.17%) males and five (20.83%) females in SVT group, and 51 (62.96%) males and 30 (37.04%) females in non-SVT group. Most common etiology was alcohol 21/24 (87.5%) in SVT group (Table 1). None of the patients with gallstones and post-endoscopic retrograde pancreaticholangiography (post-ERCP) pancreatitis developed SVT. Most common vessel involved was SplV followed by SplV along with PV and SMV (Table 2). Mean C-reactive protein (CRP) level was 242.01 ± 172.57 mg/dL in SVT group, as compared to 122.76 ± 82.10 mg/dL in non-SVT group (*P* < 0.001). Pleural effusion and ascites were observed in 21/24 (87.5%) and 12/24 (50%) patients in SVT group, while they were seen in 24/81 (29.62%) and 21/81 (25.92%) patients in non-SVT group. Every patient with SVT developed local complications: pseudocyst in 12 (50%) patients, walled-off pancreatic necrosis (WOPN) in nine (37.5%), acute necrotic collection (ANC) and acute pancreatic fluid collection (APFC) in three (12.5%) patients. Severity scoring systems like SIRS (*P* = 0.004), BISAP (*P* < 0.001), APACHE II (*P* = 0.011), mCTSI (*P* < 0.001) and revised Atlanta classification system (*P* < 0.001) were worse in the SVT group compared to the non-SVT group (Table 1). Organ failure was commonly seen

Table 1. Clinical Features in SVT and Non-SVT Groups

Variables	SVT (n = 24)	Non-SVT (n = 81)	Total (n = 105)	P value
Age (years)	36.62 ± 6.49	41.56 ± 13.85	39.12 ± 13.33	0.09
Sex				
Male	19 (79.17%)	51 (62.96%)	70 (66.67%)	
Female	5 (20.8%)	30 (37.03%)	35 (33.33%)	0.21
BMI (kg/m ²)	20.03 ± 2.25	22.49 ± 4.72	21.93 ± 4.40	0.01
Etiology				
Alcoholic	21 (87.5%)	27 (33.3%)	48 (45.7%)	
Gallstone	0 (0%)	27 (33.3%)	27 (25.7%)	
Idiopathic	0 (0%)	15 (18.5%)	15 (14.3%)	< 0.001
PEP	0 (0%)	3 (3.7%)	3 (2.9%)	
Other	3 (12.5%)	9 (11.1%)	12 (11.4%)	
Amylase (U/L)	493.00 ± 206.92	451.56 ± 150.61	461.03 ± 165	0.282
Hematocrit (%)	38.99 ± 10.74	38.00 ± 6.01	38.23 ± 7.31	0.565
CRP (mg/dL)	242.01 ± 172.57	122.76 ± 82.10	150.02 ± 119.59	< 0.001
BUN (mg/dL)	13.88 ± 9.20	14.04 ± 7.60	14.00 ± 7.94	0.929
Pleural effusion	21 (87.5%)	24 (29.6%)	45 (42.9%)	< 0.001
Ascites	12 (50%)	21 (25.9%)	33 (31.4%)	0.026
Local complications	24 (100%)	42 (51.9%)	66 (62.9%)	< 0.001
APFC	0 (0%)	24 (29.6%)	24 (22.9%)	
Pseudocyst	12 (50%)	9 (11.1%)	21 (20%)	
WOPN	9 (37.5%)	3 (3.7%)	12 (11.4%)	
ANC + APFC	3 (12.5%)	3 (3.7%)	6 (5.7%)	
ANC	0 (0%)	3 (3.7%)	3 (2.9%)	
SIRS	21 (87.5%)	45 (55.6%)	66 (62.9%)	0.004
BISAP	2.00 ± 0.88	1.00 ± 0.91	1.23 ± 0.99	< 0.001
mCTSI	7.25 ± 1.42	4.00 ± 2.32	4.74 ± 2.55	< 0.001
Revised Atlanta classification				
Mild	0 (0%)	36 (44.4%)	36 (34.3%)	
Moderate	18 (75%)	42 (51.9%)	60 (57.1%)	< 0.001
Severe	6 (25%)	3 (3.7%)	9 (8.6%)	
APACHE II	6.75 ± 4.11	4.37 ± 3.88	4.91 ± 4.04	0.011
Organ failure	9 (37.5%)	9 (11.1%)	18 (17.1%)	0.003
Intervention	15 (62.5%)	6 (7.4%)	21 (20%)	< 0.001
Mortality	3 (12.5%)	6 (7.4%)	9 (8.6%)	0.424

SVT: splanchnic venous thrombosis; BMI: body mass index; PEP: post-ERCP pancreatitis; CRP: C-reactive protein; BUN: blood urea nitrogen; APFC: acute pancreatic fluid collection; WOPN: walled-off pancreatic necrosis; ANC: acute necrotic collection; SIRS: systemic inflammatory response syndrome; BISAP: bedside index of severity in pancreatitis; mCTSI: modified computed tomography scan severity index; APACHEII: acute physiology and chronic health evaluation-II.

in the SVT group as compared to the non-SVT group 9/24 (37.5%) vs. 9/81 (11.11%) respectively (P = 0.003). Out of 24 patients in SVT group 15 (62.5%) required interventions, while 6/81 (7.4%) patients in non-SVT group needed interventions for management of local complications. Mortality was observed in 3/24 (12.5%) and 6/81 (7.4%) in SVT and

non-SVT groups respectively.

Clinical features of SVT

Single, double and triple vessels involvement were seen in 11

Table 2. Clinical Features and Outcome Between AC and Non-AC Groups

Variables	Non-AC (n = 12)	AC (n = 12)	Total (n = 24)	P value
Number of veins				
Single	10 (83.3%)	1 (8.3%)	11 (45.8%)	
Double	2 (16.7%)	2 (16.7%)	4 (16.7%)	< 0.001
Triple	0 (0%)	9 (75%)	9 (37.5%)	
Type of vein				
SplV	10 (83.3%)	1 (8.3%)	11 (45.8%)	
SplV + PV	2 (16.7%)	2 (16.7%)	4 (16.7%)	< 0.001
SplV + PV + SMV	0 (0%)	9 (75%)	9 (37.5%)	
Bowel ischemia	0 (0%)	4 (33.3%)	4 (16%)	0.093
Hepatic decompensation	0 (0%)	3 (25%)	3 (12%)	0.217
Bleeding	0 (0%)	3 (25%)	3 (12%)	0.217
Varices	4 (33.3%)	3 (25%)	7 (29.2%)	1.000
Collateral formation	3 (25%)	3 (25%)	6 (25%)	1.000
Portal cavernoma	4 (33.3%)	3 (25%)	7 (29.2%)	0.653
Recanalization	5 (41.7%)	6 (50%)	11 (45.8%)	0.682
Mortality	2 (16.7%)	1 (8.3%)	3 (12.5%)	1.000

AC: anticoagulation; SplV: splenic vein; PV: portal vein; SMV: superior mesenteric vein.

(45.8%), four (16.7%) and nine (37.5%) subjects respectively. Splenic vein was the most commonly involved vessel (24/24, 100%); isolated in 11/24 (45.8%) patients while in combination with PV and or SMV in the remaining patients ($P < 0.001$). AC was started if patients had symptoms of bowel ischemia (defined radiologically by thickened bowel wall, diminished bowel wall enhancement, ileus, pneumatosis, portomesenteric venous gas, pneumoperitoneum) and/or hepatic decompensation (defined by new onset of jaundice, ascites, and hepatic encephalopathy). Bowel ischemia (4/12, 33.3%), hepatic decompensation (3/12, 25%), triple vessel involvement (4/12, 33.3%), and pulmonary embolism (1/12, 8.3%) were reasons for AC in our study subjects. Patients were anticoagulated with intravenous heparin initially followed by oral warfarin with adjustment in dose to keep international normalized ratio 2 - 3.

Outcome of SVT

While on AC, 3/12 (25%) patients developed bleeding. In one case bleeding occurred spontaneously within the pseudocyst with rapid drop in hemoglobin concentration requiring blood transfusion, while in the other two subjects minor bleeding occurred during ultrasonography-guided percutaneous interventions without need of blood transfusion. None of the patient in non-AC group developed bleeding ($P = 0.064$). On follow-up evaluation varices (gastroesophageal and or gastric) were observed in three (25%) and four (33.3%) patients in AC and non-AC group respectively. Collateral vessels were seen in three (25%) patients in both groups, while portal cavernoma were observed in three (25%) in AC group and four (33.3%) subjects in non-AC group (Table 2). Recanalization of throm-

bosed vessels occurred in 6/12 (50%) and 5/12 (41.7%) patients in AC and non-AC group, respectively ($P = 0.682$). There was no statistically significant difference in mortality in both the groups (Table 2).

Discussion

Vascular complication like SVT is well known in AP. However, in view of the limited literature its natural history remains elusive. We have described our experience on this entity at a tertiary care referral center.

Incidence of SVT is widely variable ranging from 1% to 24%. This is due to heterogeneity of the study subjects (mild vs. severe AP, acute vs. chronic pancreatitis), etiologies and imaging modality used for diagnosis (ultrasonography vs. CT scan) [6, 9]. We encountered SVT in 24/105 (22.85%) of our patients with AP. Ahmed et al at tertiary care center found the incidence of SVT in 27.1% subjects of AP, while Easler et al reported SVT in 18% of AP patients evaluated with CECT [9, 17].

Pathogenesis of SVT is multifactorial in which pancreatic and peripancreatic inflammation plays a key role. Inflammation leads to cellular infiltration, edema and systemic activation of hemostasis with consequent deposition of platelet and fibrin thrombi formation [10, 11]. Also disruption of pancreatic tissue leads to activation coagulation; as with compression of vessels by local collections leading venous stasis. Experimental studies have also shown systemic hypercoagulable state in AP due to the effects of inflammatory mediators with increased synthesis of prothrombotic factors from liver with resultant increased risk of splanchnic and extra-SVT [18-21]. In cohort of

127 patients with AP, Gonzelez et al noted co-localized collection in 19 of 20 (95%) patients with splanchnic vein thrombosis [8]. Similarly Easler et al found that all patients with SVT or narrowing had local complications like pancreatic fluid collections and necrosis [9]. All our 24 patients with SVT had one or more of local complications of AP compared to 42/81 (51.9%) in non-SVT group, which is similar to the previous reports. It also explains the high rate of occurrence of SVT if local complications occur during the course of AP [7-9]. We found that markers of disease severity like CRP, BISAP score, APACHEII score, mCTSI and revised Atlanta class were significantly higher in patients with SVT compare to the non-SVT group, which is supported by higher level of inflammatory response in patients with SVT compare to non-SVT.

Most common vessel involved in our study was SplV, seen in all 24 patients, followed by PV in combination with SplV and or SMV, which is concordant to previous studies [7-9]. Splenic vein runs behind the tail and body of the pancreas which joins with SMV to form PV at neck of the pancreas. Inflammatory process may involve all these vessels. Common place of SplV thrombosis may be explained by its close proximity to pancreatic and peripancreatic inflammation and collections. As previous literature has suggested, evolution of SVT may be complicated by portal hypertension, hepatic decompensation and bowel ischemia due to extension of the thrombus into the PV and SMV [6-9]. Gonzelez et al noted derangement in liver function in 1/4 (25%) patients with portal vein thrombosis (PVT) in AP and suggested consideration of AC if PV and SMV involvement is present [8]. Similarly Harris et al showed thrombus extension into SMV resulting in bowel ischemia in 2/45 (4.4%) patients, one of whom died during the course of the disease [7]. In the present study bowel ischemia and hepatic decompensation were observed in 4/24 (16.67%) and 3/24 (12.5%) patients respectively during the course of AP. Limited literature suggests rate of spontaneous recanalization in up to 30% especially for SplV thrombosis [10, 12]. Also 10-year recurrence-free survival is highest for isolated SplV thrombosis, however this may not be true in case of PV thrombosis and/or SMV thrombosis [22]. Earlier studies have shown a high mortality in acute mesenteric vein thrombosis [23, 24]. At present there is no consensus guideline on AC in SVT in the setting of AP; and use of AC is mainly derived from AC in setting of extra-hepatic PV thrombosis and mesenteric thrombosis [13]. In earlier studies reasons for starting AC were heterogeneous. In our study indications for initiation of AC were involvement of PV and/or SMV with or without SplV involvement and pulmonary embolism. We identified a group of patients with SVT in which AC needs to be considered. With respect to outcomes of SVT including occurrence of varices, collateral vessels, cavernoma formation, recanalization and mortality we did not observe statistically significant difference between AC and non-AC group which are consistent with previous literature (Table 3, [7-9]). This also exemplifies the predominant role of inflammation in occurrence of SVT. Risk/benefit ratio may not always favor the routine use of AC in AP; firstly underlying inflammation rather than thrombophilia plays a predominant role, secondly SplV is the most commonly involved vessel which has a high rate of spontaneous recanalization and 10-year recurrence-free

survival; thirdly most of these patients are associated with local pancreatic collections and thereby potential candidates for interventions (radiological/surgical/endoscopic), and AC use may increase bleeding risk in this settings. Possible beneficial intervention for SVT in this setting may be early drainage of infected local pancreatic collection once its wall matures. However subset of patients with SVT may benefit from AC use; especially those having involvement of SplV with extension into PV and or SMV, associated with bowel ischemia, hepatic decompensation and underlying thrombophilia disorder. Considering the availability of limited literature, routine use of AC in every patient with SVT may not be advisable. More studies involving large number of subjects with this entity will clarify this issue and robust recommendations can be made in future.

Limitations

In present study most common etiology in SVT group was alcohol use, we could not eliminate this inherent bias as alcohol use is common in our region. It is possible that these patients might be having underlying subclinical liver disease which adversely affected coagulation system and predisposes to SVT. Results of our observation may not be applicable to other etiologies of AP. Secondly thrombophilia testing was not carried out in all patients, and it is possible that some of our patients had an underlying thrombophilic disorder. However, recent study evaluating the role of thrombophilia in AP did not find any significant difference between SVT and non-SVT group, and abnormal procoagulant parameters were more common in non-SVT patients [17]. We suggest thrombophilia testing to be considered in patients with multiple vessels thrombosis, especially if associated with extra-splanchnic thrombosis as it guides the duration of AC [13]. Another limitation is the relatively small sample size. Despite these limitations, in view of the limited literature, our experience conveys an important message on incidence, clinical features, management and outcomes of SVT in AP which will help the clinician in decision making and may form the basis for future studies.

Conclusions

SVT is seen more commonly in alcohol-induced AP. It is associated with the presence of local complications of AP. AC use may not always affect the outcome. Subset of patients with SVT may benefit from AC.

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Financial Disclosure

None to declare.

Table 3. Literature on Splanchnic Venous Thrombosis and Anticoagulation in Acute Pancreatitis

Author	Harris et al, 2013 [7]	Gonzalez et al, 2011 [8]	Easler et al, 2014 [9]	Present study
Study design	Retrospective	Prospective Retrospective	Prospective Retrospective	Prospective
Sample size	2,454	127	162	105
Rate of SVT occurrence	45/2,454 (1.8%)	24/127 (18.9%)	22/162 (14%)	24/105 (22.8%)
Predominant etiology	Gallstone	Alcohol	Gallstone	Alcohol
Local complications	Peripancreatic collection in 24/45 (53.33%); necrotizing pancreatitis in 26/45 (57.78%)	19/22 (86.36%)	21/22 (95.45%)	24/24 (100%)
Clinical features of SVT				
Most common vessel involved	Splenic vein in 30/45 (67%)	Splenic vein 14/20 (70%)	Splenic vein 19/22 (86%)	Splenic vein 24/24 (100%)
Bowel ischemia	2/45 (4.4%)	1/20 (5%)	-	4/24 (16.67%)
Hepatic decompensation	-	1/20 (5%)	-	3/24 (12.5%)
Bleeding	7/45 (15.56%)	-	2/22 (9.09%)	3/24 (12.5%)
Development of collateral/varices/portal cavernoma	Collaterals or varices 21/45 (46.67%)	Collaterals in 10/20 (50%); portal cavernoma in 3/20 (15%)	Collaterals in 19/22 (86%); varices in 6/22 (27%)	Collaterals in 6/24 (25%); varices in 7/24 (29.17%); portal cavernoma in 7/24 (29.17%)
Recanalization	5/45 (11.11%)	7/20 (35%)	2/22 (9%)	11/24 (45.83%)
Mortality	3/45 (6.67%)	1/20 (5%)	1/22 (5%)	3/24 (12.5%)
Number of patients anticoagulated	17/45 (37.78%)	4/20 (20%)	6/22 (27.27%)	12/24 (50%)
Indication for anticoagulation	Acute PV thrombosis, thrombus extension, DVT or pulmonary embolism	Acute PVT with or without SplV thrombosis or thrombus extension	Deep vein thrombosis, stroke	Bowel ischemia, hepatic decompensation, SplV + PV + SMV thrombosis, pulmonary embolism
Outcome with AC				
Bleeding	2/17 (12%)	-	2/6 (33.33%)	3/12 (25%)
Development of collateral, varices and portal cavernoma	Varices or collaterals 6/17 (35%)	-	Collaterals 5/6 (83.33%)	Collaterals in 3/12 (25%); varices in 3/12 (25%), portal cavernoma in 3/12 (25%)
Recanalization	2/17 (12%)	2/4 (50%)	0/6 (0%)	6/12 (50%)

SVT: splanchnic venous thrombosis; SplV: splenic vein; PV: portal vein; SMV: superior mesenteric vein; AC: anticoagulation; DVT: deep vein thrombosis; PVT: portal vein thrombosis.

Conflict of Interest

None to declare.

Informed Consent

Obtained.

Author Contributions

Study concepts: PRJ, SU, SN, PD, SJ, AM, PR; Study design: PRJ, SU, SN, PD, SJ, SR, QC; definition of intellectual content: PRJ, SU, SN, PD, SJ, SR, QC; literature search: PRJ, SU, SN, PD, PR, SR, QC; clinical studies: PRJ, SU, SN, PD, SJ, AM, PR; data acquisition: PRJ, SU, SN, SJ, AM, PR, SR, QC; data analysis: PRJ, SU, SN, PD, SJ, AM, PR, SR, QC; statistical analysis: PRJ, SU, PD, SJ, AM, PR, SR, QC; manuscript preparation: PRJ, PR, SR, QC; manuscript editing: SU, SN, PD, SJ, AM, PR, SR, QC; manuscript review: SN, PD, SJ, AM, PR.

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