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2

3 **TITLE:** Splenic artery embolization for traumatic and non-traumatic splenic injury

4

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25

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27 submission.

28

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31

32

33 **ABSTRACT**

34

35 **Aims**

36 The aim of this retrospective study was to identify clinical factors associated with the  
37 clinical outcome of both traumatic and non-traumatic patients who underwent splenic  
38 artery embolization (SAE) in the treatment of splenic hemorrhage.

39

40 **Methods**

41 Of 84 patients with blunt splenic injuries identified at our institution, 43 patients  
42 underwent SAE for management of bleeding. Additionally, 14 patients underwent  
43 SAE for non-traumatic splenic injuries. The following factors were assessed to  
44 determine their relationship to procedure outcomes: age, Shock Index (SI),  
45 hemoglobin (Hb), hematocrit (Ht), prothrombin time (PT), systolic blood pressure  
46 (BP), BP changes during SAE, blood transfused by the end of SAE. For blunt splenic  
47 injuries, the American Association for the Surgery of Trauma (AAST) grade and  
48 Injury Severity Score (ISS) were also assessed.

49

50 **Results**

51 The overall good clinical outcome rate was 79.1% (34/43) for traumatic patients and  
52 92.8% (13/14) for non-traumatic patients. Lower Hb, lower Ht, lower BP, less  
53 increase in BP during SAE and increased requirement for blood transfusions by the  
54 end of SAE were associated with poor clinical outcome of the patients with blunt  
55 splenic injury.

56

57 **Conclusion**

58 We revealed several factors associating with the success rate for SAE. These results  
59 may indicate the treatment of choice in patients with traumatic and non-traumatic  
60 splenic injuries.

61

62 **Keywords:** Splenic artery, Embolization, Trauma, Hemorrhage

63

64

**65 INTRODUCTION**

66 The spleen is one of the most commonly injured abdominal organs after abdominal  
67 trauma [1]. Several decades ago, splenectomy was the sole treatment for traumatic  
68 splenic injury [2], leaving asplenic patients particularly vulnerable to infection with  
69 encapsulated organisms. Surgery still remains the gold standard for treating patients  
70 with splenic injuries with hemodynamic instability, and it has constituted up to 50% of  
71 cases [3].

72 Splenectomy is also the first choice for the treatment of atraumatic splenic injury. On  
73 the other hand, with blunt traumatic splenic injury, non-operative management has  
74 been employed as an alternative in hemodynamically stable patients [2, 4-8] and is  
75 becoming the new standard for treatment [9], not only for patients with abdominal  
76 multiorgan injuries [10] but also for children [11]. Among nonoperative approaches,  
77 transcatheter artery embolization (TAE) has been widely used to control bleeding in  
78 patients with abdominal injuries, as it can rapidly assure hemostasis. In 1981,  
79 Sclafani presented 4 patients with splenic injuries, in whom angiography and splenic  
80 artery embolization (SAE) were applied [12]. After that, many studies have confirmed  
81 the effectiveness of SAE in hemodynamically stable patients with blunt traumatic  
82 splenic injuries, showing that SAE was able to increase the success rate of non-  
83 operative management [13,14]. Criteria for non-operative management includes 1)  
84 the restoration of hemodynamic stability with minimal fluid resuscitation; and 2) the  
85 absence of significant associated injuries requiring surgical intervention. However,  
86 controversy remains regarding the indications for SAE [1] [5] [6] [8] [14,15].  
87 Moreover, a number of studies have suggested that various clinical factors should be  
88 used to guide the choice of treatment modality but still fail to reach any conclusions  
89 [16, 17].

90 SAE for patients with non-traumatic splenic injury has also not yet been established  
91 with definite value. We retrospectively reviewed the medical records of patients with  
92 traumatic splenic injury and non-traumatic splenic injury who received SAE,  
93 compared the factors of both groups and assessed the outcome of SAE to clarify  
94 clinical factors that are associated with clinical outcome of the patients who  
95 underwent this procedure.

96

97 **MATERIALS AND METHODS**

98 This retrospective study obtained the approval of our institutional review board. We  
99 confirmed that the patients or the legal representatives of the patients in this study  
100 were given a comprehensive written statement of information about the clinical  
101 study, including information on SAE, and their consent was documented in the  
102 clinical records. We reviewed the records of 84 consecutive patients who were  
103 admitted to the Emergency Center of Yokohama City University between January  
104 1996 and April 2015 for blunt splenic injuries with or without injuries to other organs.  
105 Patients who required emergency surgery for gastrointestinal tract injury or those  
106 with severe hemodynamic instability did not undergo angiography. Those patients  
107 with stable hemodynamics, implying a lack of significant bleeding, also did not  
108 undergo angiography. Thus, 49 of 84 patients underwent angiography. Of these 49  
109 patients, four patients had an injury grade that could not be classified due to  
110 incomplete documentation in the clinical chart, and those patients were excluded  
111 from the study. An additional 2 patients were also excluded from the study; in one  
112 case, TAE was performed to stop pancreatic hemorrhage after splenectomy, and in  
113 the other case, the patient underwent SAE for delayed splenic rupture after a period  
114 of conservative management in another hospital. The remaining 43 patients (33  
115 males and 10 females) were included in this study. The patient age range was 8-77  
116 years (mean±SD, 37.6±19.1). Inciting events for splenic injury included traffic  
117 accidents (n=27), falls (n=11), and assaults (n=3). Two other cases included hit by  
118 falling down and uncertain origin. Additionally, we reviewed the records of 15  
119 consecutive patients who were admitted to our institution for non-traumatic splenic  
120 injuries and received SAE. One patient who was actually bleeding from pancreatic  
121 artery was excluded from the study. The remaining 14 patients (10 males and 4  
122 females) were included in this study. The patient age range was 41-80 years  
123 (67.4±9.4). Inciting events for non-traumatic splenic injury included rupture of a  
124 splenic artery aneurysm, vascular malformation, tumor, and spontaneous bleeding.  
125 Radiologists examined all 57 patients using standard angiographic techniques as  
126 shown later in detail. Those radiologists were well trained, board-certified, and had  
127 more than 8 years of SAE experience in the emergency department. The decision to  
128 perform embolization was ultimately made by those radiologists. Indications usually

129 included the presence of extravasation or pseudoaneurysm. Even if there was no  
130 evidence of extravasation, patients proceeded to SAE if they had evidence of  
131 disruption of terminal arteries or avascularity and irregularity in the accumulation of  
132 contrast medium. A good clinical outcome was defined as the ability to control  
133 bleeding successfully without the use of ancillary methods. A poor clinical outcome  
134 of patients was defined as inadequate hemostasis as documented by ultrasound (an  
135 expanding collection), intraoperative observations (visual bleeding), clinical scenario  
136 (hemodynamic instability despite of continuous blood transfusion) (n=6), and death  
137 within 6 hours of SAE (n=4).

138

### 139 **Splenic artery embolization**

140 After initial stabilization in the emergency room, patients with suspected active intra-  
141 abdominal bleeding that did not require immediate surgery underwent angiographic  
142 investigations. Additionally, hemodynamically stable patients with apparent splenic  
143 bleeding on CT proceeded to the procedure. Splenic artery angiography was  
144 obtained using intra-arterial administration of 61.24% iopamidol (Iopamiron 300,  
145 Nihon Schering, Osaka, Japan), 64.71% iohexol (Omnipaque 300, Daiichi, Tokyo,  
146 Japan), or 61.24% iomeprol (Iomeron 300, Eisai, Tokyo, Japan) at a rate of 3-4  
147 ml/sec (for a total of 10-15 ml) using a digital subtraction angiographic device  
148 (POLYSTAR T.O.P, SIEMENS, Munchen, Germany or Ultimax-1, Toshiba Medical  
149 Systems, Tochigi, Japan). We used 5 Fr catheters such as MP-YT5.0F, MP-YT5.0F  
150 (1)-805-S, RM3, and shepherd hook (Cathex, Tokyo, Japan), a 4 Fr cobra-head  
151 catheter (TERUMO, Tokyo, Japan) and 2.0 -3.0 Fr microcatheters, such as SP  
152 catheter, Sniper 2 (TERUMO, Tokyo, Japan), FASTRACKER 325 (Boston Scientific,  
153 Cork, Ireland), or Bobsled (Kaneka medical, Osaka, Japan). Coil embolization was  
154 performed with coils ranging from 3-10 mm in diameter. Gelatin sponges used in  
155 SAE were SPONGEL (Yamanouchi, Tokyo, JAPAN) or GELFORM (Pharmacia and  
156 Upjohn, Tokyo, Japan). N-butyl cyanoacrylate (NBCA) (Histoacryl B, Braun,  
157 Melsungen, Germany) was also used for distal branches in case rapid embolization  
158 was needed with disrupted coagulation system. NBCA was mixed with iodized oil  
159 Lipiodol (Terumo Co.) at a ratio of 20%-25% and infused through a microcatheter.  
160 SAE was performed by placing coils in the main trunk of the splenic artery between

161 the origin of the dorsal pancreatic branch and the next most distal pancreatic branch,  
162 or the intraparenchymal branch of the spleen if there was prominent bleeding that  
163 was not difficult to approach with the catheter. Gelatin sponge particles or NBCA  
164 were injected into the distal branches of the splenic artery with outstanding  
165 extravasation. The choice of coils or microcoils depended on the size of the catheter  
166 used, and choice of coil size (3 mm, 5 mm, 6 mm, 8 mm or 10 mm diameter) was  
167 based on visual interpretation of the arterial diameter on angiogram. Stasis of  
168 contrast material at the proximal point of the splenic artery was demonstrated via  
169 fluoroscopy, and coil delivery was completed to ensure hemostasis. Perfusion to the  
170 spleen was maintained by the left gastric – short gastric route or the dorsal  
171 pancreatic artery. These routes were not embolized to maintain splenic perfusion.  
172 Finally, a celiac arteriogram was obtained to confirm occlusion of main trunk of the  
173 splenic artery. Follow up CT scans were obtained in 35 of the 47 patients. The good  
174 clinical outcome group all had a partial low-density area in the splenic parenchyma  
175 on contrast-enhanced CT, which was associated with the injury itself or a small  
176 amount of splenic infarction, while the remaining splenic parenchyma maintained  
177 good perfusion. The following factors were assessed to determine their relationship  
178 to procedure outcomes: age, Shock Index (SI), hemoglobin (Hb), hematocrit (Ht),  
179 prothrombin time (PT), systolic blood pressure (BP), BP changes during SAE, and  
180 blood transfused by the end of SAE. For blunt splenic injuries, the American  
181 Association for the Surgery of Trauma (AAST) grade and Injury Severity Score (ISS)  
182 were also assessed. Traumatic patients were classified using the AAST Organ Injury  
183 Scale-Spleen, based on CT findings or intraoperative observation. Statistical  
184 analyses were performed using the Excel add-on software, Xlstat (addinsoft,  
185 Cologne, Germany). Statistical analysis was performed using the Mann-Whitney U-  
186 test, and statistical significance was set at  $P < 0.05$ .

187

## 188 RESULTS

189 Of the 45 patients with blunt splenic injury that underwent TAE, 43 qualified for this  
190 study. Of these 43 patients, we confirmed good clinical outcome in 34 patients  
191 (79.1%). Grade of injury was not a significant predictor of clinical outcome (Table 1).  
192 There was no significant association between patient age and clinical outcome or



193 between SI (0.44 to 2.50), ISS (4.0 to 50.0) and clinical outcome (Table 2). Lower  
194 values of Hb and Ht were significantly associated with poor clinical outcome (Table  
195 2), but differences in PT were not (Table 2). BP range was 50-161 mm Hg just  
196 before SAE and 40-170 mm Hg after SAE. BP elevation during SAE ranged from -50  
197 to 70 mm Hg. Lower values of each of these parameters were significantly  
198 associated with poor clinical outcome (Table 2). Blood transfusion requirements  
199 before SAE (range, 0-40 units) were significantly associated with clinical outcome of  
200 the patients (Table 2), with lower blood transfusion requirements associated with a  
201 favorable clinical outcome. In the non-traumatic group, mean age was significantly  
202 higher than that of the traumatic group, SI, Hb, and Ht were lower, and post-SAE BP  
203 was higher (Table 3). Inciting events for non-traumatic splenic injury included rupture  
204 of splenic artery aneurysm (n=11), vascular malformation (n=1), tumor (n=1), and  
205 spontaneous bleeding (n=1) (Table 4). One case with non-traumatic splenic injury in  
206 which SAE failed showed a trend for lower Hb (4.9 vs 8.6±1.6: average), lower Ht  
207 (14.2 vs 26.2±5.0), prolonged PT (2.1 vs 1.2±0.3), and lower BP (76 vs 125.6±30.6),  
208 but no statistical analysis was performed because this was the only case with failure  
209 of SAE in non-traumatic splenic injury.

210

## 211 DISCUSSION

212 Several decades ago, splenectomy was the sole treatment of choice for the patients  
213 with blunt splenic injuries. Over the last decade, non-operative management has  
214 become the preferable treatment for hemodynamically stable patients with blunt  
215 splenic injuries and the failure rate of the non-operative management has  
216 considerably decreased, possibly attributed to the introduction of SAE. A number of  
217 studies have previously assessed the success rate of SAE for the management of  
218 bleeding in blunt splenic injury. In this series, we confirmed good clinical outcome in  
219 79.1% of patients, which is slightly lower than previously reported success rates of  
220 over 80% [4-6]. Considering the wide indications for SAE (such as including patients  
221 with multiorgan injuries) and strict definition of good clinical outcome in the present  
222 study, these factors might contribute to the reduced success rate, and we were able  
223 to attain a considerably favorable outcome. Mean ISS in our study (22.8±11.7) were  
224 slightly higher than those in previous reports [4-6]. For example, Sclafani et al.



225 reported a success rate of 95%, but patients in their study had a mean ISS of 18,  
226 and the rate of associated complications such as pelvic injury was lower than that in  
227 the present study (4/60<0.1% vs. 12/43=27.9%) [4]. A large analysis that included  
228 23,532 patients with blunt splenic injuries showed that the frequency of non-  
229 operative management failure was proportional to a higher AAST grade and ISS  
230 [18]. Another study of 6,308 patients with blunt splenic injuries showed the frequency  
231 of unsuccessful non-operative management were associated with the AAST grade  
232 and the amount of intraperitoneal hematoma [19]. Brasel et al. reported that injury  
233 grade was the only factor related to the success rate of non-operative management  
234 [7]. In the present study, splenic injury was distributed between all AAST grades (I-  
235 V), but injury grade did not emerge as a significant factor influencing the clinical  
236 outcome of SAE. Additionally, ISS did not affect the outcome. These results are  
237 contrary to previous reports supporting the influence of higher AAST grade and  
238 higher ISS on the higher failure rate of non-operative management. Reasons for this  
239 discrepancy may include the backgrounds of the patients or design of the present  
240 study. Indeed, Moore et al. noted that the AAST splenic injury scale was not  
241 developed to assign a prognostic value [20]. In grading splenic injury, we used the  
242 established practice of computed tomography (CT). However, there were some  
243 reports indicating CT findings often show no correlation to the severity of the splenic  
244 injury [21] [22] and were notoriously poor in identifying vascular injuries [4-5]. For  
245 example, Sutyak et al. reported that CT findings were a poor predictor of operative  
246 findings of the degree of adult splenic injury [21]. However, currently CT  
247 examinations have been thought essential for the choice of treatment in patients with  
248 splenic injuries [6] [14,15] [23]. The results presented by the National Trauma  
249 Registry of the American College of Surgeons showed contrast CT blush was one of  
250 the factors in non-operative management failure [14]. The results of other studies  
251 also have shown that the presence of contrast blush on CT was correlated with  
252 extravasation on angiography, and was correlated with a definitely higher risk of non-  
253 operative management failure [6] [23]. Bhullar et al. found a strong correlation  
254 between the presence of contrast blush found on CT and active bleeding found on  
255 angiography [15]. They also emphasized that CT contrast blush indicated the  
256 necessity of applying embolization in patients with blunt splenic injuries who qualified

257 for non-operative management. Good clinical outcome was independent of patient  
258 age. Smith et al. suggested that patients over 55 years of age require surgical  
259 management [2], while Brasel et al. concluded that there was no correlation between  
260 age and the success rate of nonsurgical management [7]. The concept that patient  
261 age does not affect the success rate of non-operative management seems to have  
262 prevailed recently [7], but some studies indicate that aging is one of the predictors for  
263 the failure of non-operative management [24]. Previous studies have demonstrated  
264 that operative management of patients with blunt splenic injury was employed for  
265 patients with significantly higher Injury Severity Scores than for those managed  
266 nonoperatively [6] [21]. Velmahos et al. compared the Injury Severity Scores of  
267 patients with positive angiograms to those with negative angiograms and found no  
268 significant difference [8]. Davis et al. reported that a higher ISS does not  
269 automatically predict failure of non-operative management [6]. Olthof et al. undertook  
270 a systematic review of studies to identify prognostic factors for non-operative  
271 management failure in patients with blunt splenic injuries. The severity of injury  
272 according to the ISS, with ISS>25, is one of the factors predicting non-operative  
273 management failure [24]. In the present study, mean SI and mean ISS had no  
274 significant influence on the clinical outcome of the patients with blunt splenic injury.  
275 Although it is reasonable to expect that low Hb, low Ht, impairment of the coagulation  
276 system and low blood pressure would contribute to poor clinical outcome, there is no  
277 definite data to confirm this supposition. In the present study, we demonstrated that  
278 low Hb, low Ht, and low blood pressure were associated with poor clinical outcome  
279 of the patients with blunt splenic injury who underwent SAE. However, impairments  
280 in coagulation (PT) showed no association with clinical outcome. As to non-traumatic  
281 splenic injuries, most of the cases involve rupture of splenic artery  
282 pseudoaneurysms, which were associated with postoperative inflammatory changes  
283 around the surgical site, such as inflammation of the pancreas, stomach, colon and  
284 other causes of pancreatitis. A tumor could also be the cause of atraumatic splenic  
285 rupture. In the present study, a 61-year-old male presented to our institution for a  
286 splenic rupture of initially unknown origin, and SAE was performed using coils to stop  
287 bleeding (Figure 1). After his hemodynamic condition was improved, splenectomy  
288 was performed. Pathological examination showed large cell endocrine carcinoma in

289 the ruptured spleen. Splenectomy is the first choice for treatment of atraumatic  
290 splenic rupture. A study of atraumatic splenic rupture found that even if all  
291 preconditions for non-operative management are met, the failure rate is high [25]. It  
292 also states that even in hemodynamically stable patients, there are three reasons  
293 splenectomy is chosen as follows: 1. Histological examination of the spleen will  
294 establish the etiology of the atraumatic splenic rupture as well as any underlying  
295 systemic diseases. 2. A significant number of malignant diseases may cause  
296 atraumatic splenic rupture, so any organ-preserving approach should be prohibited.  
297 3. The splenic function might already be compromised by a pathological alteration or  
298 infiltration of the splenic parenchyma, and under such a hyposplenic condition,  
299 removal of the non-functioning spleen is justified and will not increase the risk of an  
300 overwhelming postsplenectomy infection. We performed SAE first even for non-  
301 traumatic patients if they were included in the category of non-operative  
302 management. Instead of splenectomy, SAE can be an alternative option to improve  
303 hemodynamic condition in either trauma or non-trauma patients with splenic  
304 hemorrhage. However, this approach should be used if the interventional radiology  
305 team has the appropriate knowledge and experience to perform SAE. Matsumura et  
306 al. reported two cases of atraumatic splenic rupture that received splenic artery  
307 occlusion before splenectomy as well [26]. Schnüriger et al. found no significant  
308 difference in major complications, such as requiring splenectomy after SAE between  
309 proximal and distal embolization, but minor complications, such as minor infarctions,  
310 were significantly more frequent after distal embolization [27]. Concerns exist  
311 regarding the remaining splenic function after embolization. A small study comparing  
312 15 previously embolized patients, 14 splenectomy patients, and 30 control subjects  
313 showed both embolized and splenectomy patients had higher leukocyte and platelet  
314 counts compared to controls. It also showed that there was no significant difference  
315 in the size of the spleen or immunoglobulin titers between embolized patients and  
316 controls [28]. A Japanese study reported on immunologic alterations after splenic  
317 preservation such as embolization or splenorrhaphy compared to those who  
318 underwent splenectomy, and it showed no discernible advantage to preservation  
319 over splenectomy [29]. These results quite engage our interest, and the immunologic  
320 effects after SAE still remains unclear and needs to be discussed further.

**321 CONCLUSION**

322 We retrospectively reviewed the medical records of 84 patients with blunt splenic  
323 injury and assessed 43 patients in regards to SAE clinical outcome. Low Hb, low Ht,  
324 low blood pressure before and after SAE, decreases in blood pressure during the  
325 procedure, and increased transfusion requirements before SAE were all associated  
326 with poor clinical outcome. Injury grade, patient age, SI, ISS, PT did not significantly  
327 affect the clinical outcome of the patients who underwent SAE in blunt splenic injury.  
328 We also performed SAE in 14 cases for non-traumatic patients. Non-traumatic  
329 splenic hemorrhage occurs mostly in patients with rupture of splenic artery  
330 pseudoaneurysm; however, other rare cases such as malignant tumors should be  
331 taken into account. Patients with non-traumatic splenic injury tend to be older in age  
332 and have lower Hb and lower Ht, but the result of SAE was considerably favorable.  
333 These results may indicate the treatment of choice in patients with traumatic and  
334 non-traumatic splenic injuries. More prospective, randomized studies are still  
335 required.

336

**337 CONFLICT OF INTEREST**

338 Authors declare no conflict of interest.

339

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478 **TABLES**

479

480 Table 1: Distribution of AAST Grades with respect to clinical outcome of the 43  
481 trauma patients performed SAE

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AAST Grade system (No. of patients)	Good outcome (No. of patients)	Poor outcome (No. of patients)
I (1)	0	1
II (9)	6	3
III (18)	15	3
IV (13)	11	2
V (2)	2	0
Total (%)	34 (79.1)	9 (20.9)
Mean (sd) Grade	3.3 (0.8)	2.7 (1.0)

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484 Abbreviations:

485 AAST, American Association for the Surgery of Trauma

486 SAE, splenic artery embolization

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500 Table 2: Comparison of factors with respect to clinical outcome of SAE in the 43  
 501 patients with traumatic splenic injury

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	Good outcome (Mean ± sd)	Poor outcome (Mean ± sd)	Significant difference
Age (years)	36.1 ± 8.8	43.1 ± 20.7	No.
Shock Index	1.0 ± 0.4	1.2 ± 0.7	No.
Injury Severity Score	22.4 ± 12.1	24.3 ± 10.4	No.
Hb <sup>a</sup> (mg/dl)	11.4 ± 2.4	8.5 ± 2.4	p < 0.01
Ht <sup>b</sup> (%)	33.6 ± 6.7	25.3 ± 7.5	p < 0.01
PT <sup>c</sup> (INR)	1.2 ± 0.4	1.2 ± 0.1	No.
BP <sup>e</sup> before TAE (mmHg)	107.2 ± 23.7	86.3 ± 21.1	p < 0.03
BP after SAE (mmHg)	118.0 ± 19.7	83.0 ± 36.3	p < 0.01
Elevation of BP after SAE (mmHg)	11.5 ± 8.3	-10.8 ± 18.5	p < 0.01
Blood transfused before SAE (unit)	5.5 ± 8.3	13.0 ± 10.4	p < 0.03

503

504 Abbreviations:

505 a. Hb, hemoglobin

506 b. Ht, hematocrit

507 c. PT, prothrombin time

508 d. APTT, activated partial thromboplastin time

509 e. BP, systolic blood pressure

510 sd: standard deviation

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516 Table 3: Comparisons of various factors in the traumatic and non-traumatic patients  
 517 who underwent SAE

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	Patients with traumatic injury (n= 43)	Patients without traumatic injury (n = 14)	Significant difference
Age (years)	37.6 ± 19.1	67.4 ± 9.4	p < 0.01
Shock Index	1.08 ± 0.4	0.78 ± 0.3	p < 0.03
Hb <sup>a</sup> (mg/dl)	10.7 ± 2.6	8.6 ± 1.6	p < 0.03
Ht <sup>b</sup> (%)	31.8 ± 7.5	26.2 ± 5.0	p < 0.03
PT <sup>c</sup> (INR)	1.2 ± 0.3	1.2 ± 0.3	No.
BP <sup>e</sup> before SAE (mmHg)	105.7 ± 25.9	125.6 ± 30.6	No.
BP after TAE (mmHg)	109.2 ± 30.7	135.0 ± 32.5	p < 0.03
Elevation of BP after SAE (mmHg)	4.2 ± 21.0	9.8 ± 22.1	No.
Blood transfused before SAE (unit)	7.0 ± 9.1	5.1 ± 5.7	No.

519

520 Abbreviations:

521 a. Hb, hemoglobin

522 b. Ht, hematocrit

523 c. PT, prothrombin time

524 d. APTT, activated partial thromboplastin time

525 e. BP, systolic blood pressure

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531 Table 4: Distribution of the cause of hemorrhage in the 14 patients with non-  
532 traumatic splenic injury

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Cause of splenic hemorrhage	No of patients
Aneurysm	11
Post-surgery	4
Pancreatitis	5
Unknown	2
Vascular malformation	1
Neoplasm	1
Spontaneous	1
Total	14

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553 **FIGURE LEGEND**

554 Figure 1: 61-year-old male with atraumatic splenic rupture. (A) – Contrast-enhanced  
555 CT at presentation. A large amount of hematoma was seen in the parenchyma of the  
556 spleen and the surrounding peritoneal cavity. (B) – Celiac arteriogram before SAE.  
557 The avascular area had spread around the splenic hilum (arrows). No extravasation  
558 was observed. (C) – Celiac arteriogram after SAE. The main trunk of the splenic  
559 artery was occluded with coils (arrows). Perfusion to the spleen was decreased but  
560 maintained via the short gastric artery and the pancreatic artery (not shown). The  
561 cause of the hemorrhage was determined to be from a neoplasm. Pathological  
562 examination confirmed large cell endocrine carcinoma.

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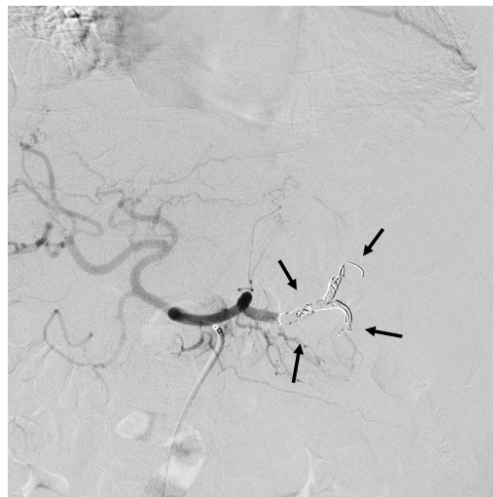
585 **FIGURE**

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(A)

(B)



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(C)

590 Figure 1: 61-year-old male with atraumatic splenic rupture. (A) – Contrast-enhanced  
591 CT at presentation. A large amount of hematoma was seen in the parenchyma of the  
592 spleen and the surrounding peritoneal cavity. (B) – Celiac arteriogram before SAE.  
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