Ruptured Abdominal Aortic Aneurysm

Categorization of Sonographic Findings and Report of 3 New Signs

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Objective. The purpose of this study was to categorize the sonographic findings seen in patients with a ruptured abdominal aortic aneurysm (AAA) and to describe 3 previously undescribed sonographic findings. ***Methods.*** From January 1997 to December 2003, we evaluated 388 consecutive patients with an AAA (transverse aortic diameter >30 mm). Among these patients, 29 had surgical or computed tomographic demonstration of aneurysm rupture. The remaining 359 were asymptomatic and had no evidence of AAA rupture at follow-up. ***Results.*** Findings recognized among 29 positive cases included AAA deformation (n = 12), luminal thrombus inhomogeneity (n = 20), clear interruption of a luminal thrombus (n = 5), retroperitoneal hematoma (n = 22), and hemoperitoneum (n = 11). In addition, 3 previously unreported findings were noted: an intraluminal floating thrombus layer (n = 8), a parietal hypoechoic focus due to aneurysm wall interruption (n = 3), and a para-aortic hypoechoic area adjacent to the bleeding side (n = 4). Aside from AAA deformation and thrombus heterogeneity, no other signs were recognized among subjects with a nonruptured aneurysm. ***Conclusions.*** In the past, sonography has been used mainly to rapidly confirm aneurysm presence in the clinical setting of a patient with a suspected rupture. Instead, this retrospective series shows how this imaging technique can frequently identify several direct and indirect signs of aneurysm rupture itself. Moreover, 3 new indicators of AAA rupture have been observed. ***Key words:*** abdominal aorta; abdominal aortic aneurysm; rupture; sonography.

Abbreviations
AAA, abdominal aortic aneurysm; CT, computed tomography

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Rupture of an abdominal aortic aneurysm (AAA) is a medical catastrophe requiring prompt diagnosis and treatment. Diagnostic delay is known to be one of the main causes of death in these patients.[1,2] Consequently, patients with typical symptoms should not undergo prolonged preoperative investigation.

In the past, rupture of an AAA was mainly diagnosed with computed tomography (CT).[3–6] This technique has high accuracy in detecting both an AAA and retroperitoneal hemorrhage; in addition, eventual contrast medium extravasation may directly confirm the rupture presence and site. In recent years, with worldwide growing use of emergency department screening sonography, there has been an increasing number of AAAs detected sonographically.[7–11] Sonography is now largely used in emergency detection of aneurysms in patients with sus-

pected rupture. Nevertheless, to our knowledge, surprisingly, no series to date has yet categorized the specific imaging patterns encountered. Moreover, we have observed 3 previously undescribed sonographic signs of AAA rupture.

The purpose of this study was to categorize the sonographic features of AAA rupture, to define sensitivity and specificity of each sign, and to illustrate 3 new findings.

Materials and Methods

From January 1997 to December 2003, we evaluated 388 consecutive patients with an AAA. Original sonographic examinations were carried out by 11 different radiologists, using AU5, EsaTune, and Technos units (Esaote SpA, Genoa, Italy) and curved phased array transducers (3–6 MHz). Diagnosis of an AAA was based on demonstration of a transverse diameter of larger than 30 mm (aside from patient body habitus).

Among the 388 patients, there were 29 (7.5%) who had surgical (27 of 29) or CT (2 of 29) demonstration of AAA rupture. These 29 patients (18 men and 11 women; age range, 67–84 years; mean age, 77 years) were evaluated on an emergency basis. We rapidly explored the right upper quadrant, left upper quadrant, paracolic gutters, and pelvis to search for free peritoneal fluid. Subsequently, we moved the transducer to the umbilical area, and we evaluated the aorta and the periaortic area to assess the aneurysm with longitudinal, transverse, and oblique scans and to detect potential contiguous hematoma. Last, we moved the transducer anterolaterally to identify possible pararenal and perirenal fat blurring and renal displacement. Sonographic studies were personally performed by radiologists with at least 5 years of experience in emergency sonography applications. Emergent examinations lasted from 1 to 5 minutes and were carried out 10 to 120 minutes after patient arrival (mean, 20 minutes). The time windows between occurrence of symptoms and patient arrival to the emergency department were known in 19 of 29 patients and ranged from 30 minutes to 8 hours (mean, 1 hour). A CT correlation was available in 18 of 29 cases. Two patients (who underwent CT confirmation of rupture) died before surgery; 5 died during surgery for AAA rupture; and 7 died within 2 weeks after surgery for AAA rupture; the remaining 15 patients survived after surgical AAA repair. Survival was similar between the 11 patients undergoing surgery immediately after sonography and the 18 patients also undergoing CT examination in their diagnostic workup: survival rates were, respectively, 46% and 50%. These 29 patients were considered to have had ruptured aneurysms for reviewing purposes.

The remaining 359 patients (92.5% of 388) were asymptomatic at the time of sonographic examination. This group of patients (229 male and 130 female; age range, 30–89 years; mean age, 68 years) was examined for different clinical scenarios, and an AAA was an already known finding in some of them, whereas it was an incidental finding in others. For patients undergoing serial sonographic studies, only the first examination was considered retrospectively. These 359 patients were considered to have had nonruptured aneurysms for our retrospective analysis purpose. Follow-up data were available for 211 of 359 patients (range, 1 month to 6 years; mean, 14 months). None of these patients had aneurysm rupture within the first 3 months of follow-up, but the diagnosis of a ruptured AAA was made in 2 patients at 4 months, in 6 at 1 year, in 2 at 2 years, in 5 at 3 years, in 1 at 4 years, and in 1 at 5 years. Given the prolonged intervals between the sonograms and the ruptures in these 17 patients, we concluded that the AAA was not ruptured at the time of our sonographic examination.

Retrospectively we assessed the stored single scans (first 157 patients, from January 1997 to December 2000) or the stored video clips (next 231 patients, from January 2001 to December 2003) to identify eventual sonographic signs of rupture. Images were reviewed in a nonblinded manner by consensus of 2 authors. In both groups, with and without aneurysm rupture, the following findings were categorized: AAA deformation (defined as clear irregularity of aneurysm shape), luminal thrombus inhomogeneity (defined as heterogeneous echogenicity of eventual peripheral thrombosis), luminal thrombus interruption (defined as clear focal discontinuity of eventual peripheral thrombosis at the lumen-to-thrombus interface), a thrombus floating layer (defined as a thrombus layer attached on 1 side and freely floating within the lumen on the other side), focal parietal interruption (defined as clear focal discontinuity of the outer aneurysm wall), a para-aortic hypoechoic focus (defined as a small hypoechoic area adjacent to the aneurysm wall and surrounded on

the opposite side by echoic, heterogeneous hematoma), retroperitoneal hematoma (defined as fluidlike or masslike obliteration of retroperitoneal spaces adjacent to the AAA), and hemoperitoneum (defined as free fluid filling peritoneal spaces).

Moreover, the following aspects were noted in both groups of patients: aneurysm size (larger transverse diameter), aneurysm location (suprarenal if the proximal margin extended above the superior mesenteric artery level or infrarenal if the proximal margin was seen below the superior mesenteric artery level), and aneurysm luminal thrombosis (absent, present and concentric, or present and eccentric).

Results

Among the 29 patients with a ruptured AAA, the following findings were recognized: AAA deformation in 12 cases, luminal thrombus inhomogeneity in 20 cases (Figure 1), clear interruption of the lumen thrombus in 5 cases (Figure 2), an intraluminal floating thrombus layer in 8 cases (Figure 3), aneurysm wall interruption in 3 cases (Figure 4), a para-aortic hypoechoic area adjacent to the bleeding site in 4 cases (Figure 5), retroperitoneal hematoma in 21 cases (Figure 6), and hemoperitoneum in 10 cases. The distribution of the sonographic signs of rupture is reported in Table 1. Retroperitoneal hematoma was right sided in 8 cases, left sided in 12, and bilateral in 1, and it frequently caused renal dislocation. Hemoperitoneum was limited to 1 peritoneal space in 8 cases (Morison pouch in 6 and Douglas pouch in 2), to 2 spaces in 1 case, and to 3 spaces in 1 case. Ruptured aneurysms ranged in size from 41 to 136 mm (mean, 67 mm). Twenty-seven were considered infrarenal and 2 were considered suprarenal. Lumen thrombosis was present in all 29 cases, being concentric in 3 and eccentric in 26.

Among 359 patients with a nonruptured aneurysm, the following findings were retrospectively identified: AAA deformation in 27 cases and luminal thrombus inhomogeneity in 89. No other sonographic sign of rupture was identified. The sizes of these nonruptured aneurysms ranged from 31 to 99 mm (mean, 46 mm). Three hundred eight were infrarenal and 51 were suprarenal. A lumen peripheral thrombus was seen in 298 cases and was considered concentric in 44 cases and eccentric in 254.

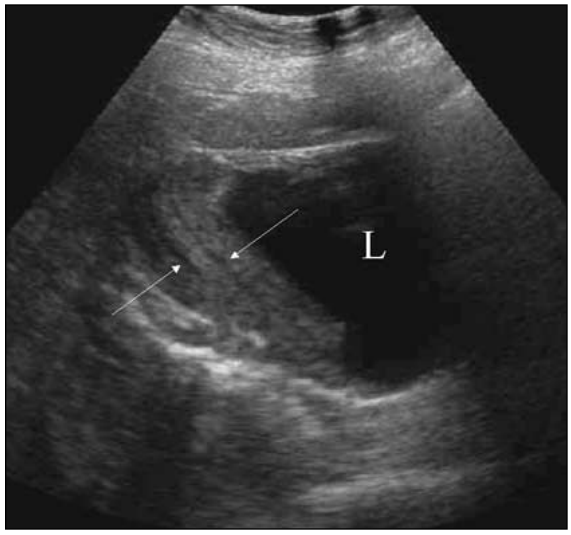


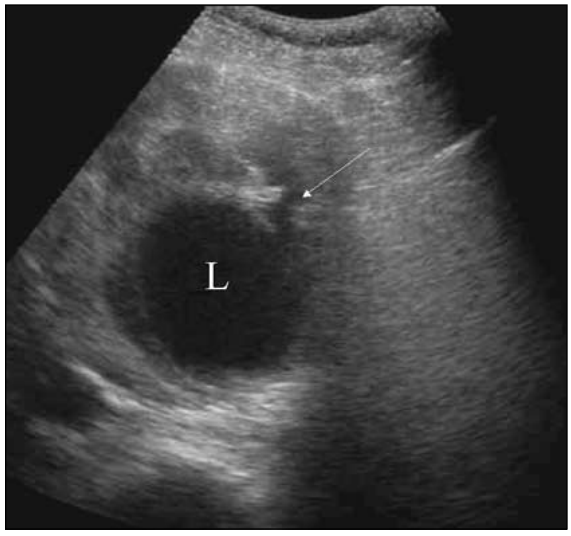
Figure 1. Inhomogeneous layered luminal thrombus. Multiple hypoechoic bands (arrows) are recognizable with peripheral eccentric thrombosis. L indicates aortic lumen.

Sensitivity and specificity of categorized sonographic signs of rupture are given in Table 2.

Discussion

Most sonography reviews and textbooks on the topic of AAA rupture, even recently, have focused on emergent aneurysm detection in patients with the clinical suggestion of rupture.[12–14]

Figure 2. Internal interruption of a luminal thrombus. A clear discontinuity is shown extending deeply within the inhomogeneous mural thrombus (arrow). L indicates aortic lumen.



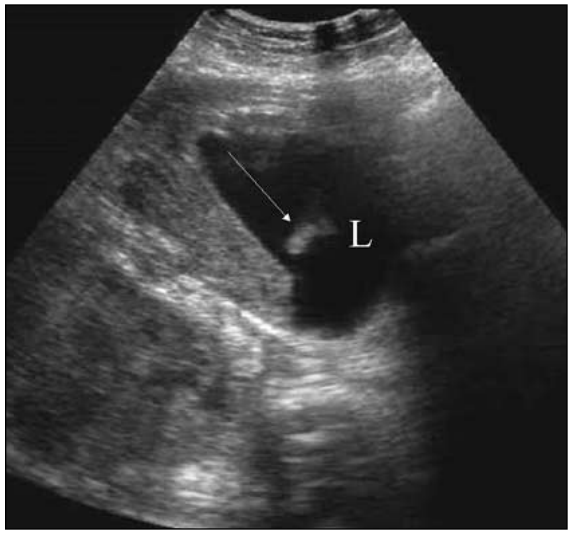


Figure 3. Floating thrombus layer (same patient as in Figure 1). A thin layer (arrow) detached from the mural thrombus is visible floating within the aneurysm lumen (L).

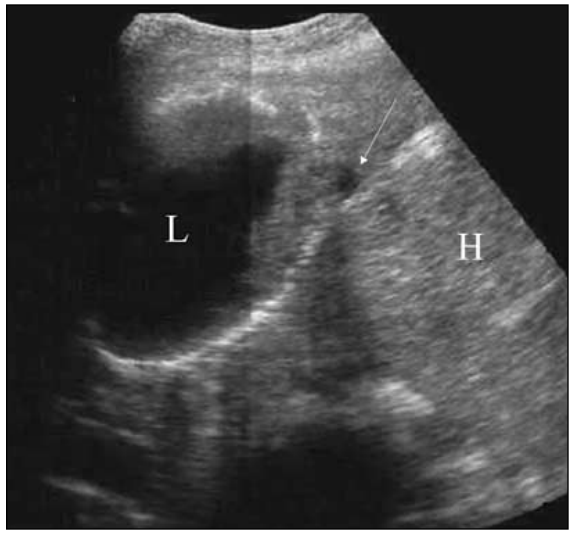


Figure 5. Para-aortic hypoechoic focus (same patient as in Figure 4). A small hypoechoic area (arrow) is seen adjacent to the aorta, on the retroperitoneal hematoma (H) side, in close relationship with the parietal discontinuity shown in Figure 4. L indicates aortic lumen.

Sonography is only considered as a rapid screening tool for AAA presence, but no effort is done to analyze the possibility of sonographically confirming effective aneurysm rupture. Barkin and Rosen[12] stated that "US is insensitive for retroperitoneal bleeding, so it should not be used to detect the presence of rupture."

We agree that, in the proper clinical setting, sonographic detection of an AAA should prompt further imaging (CT examination) or immediate surgery. However, we also think that current state-of-the-art sonographic equipment allows recognition of many signs of rupture itself.

Figure 4. Focal parietal interruption. A small hypoechoic area of discontinuity (arrow) is evident within the calcified wall. L indicates aortic lumen.

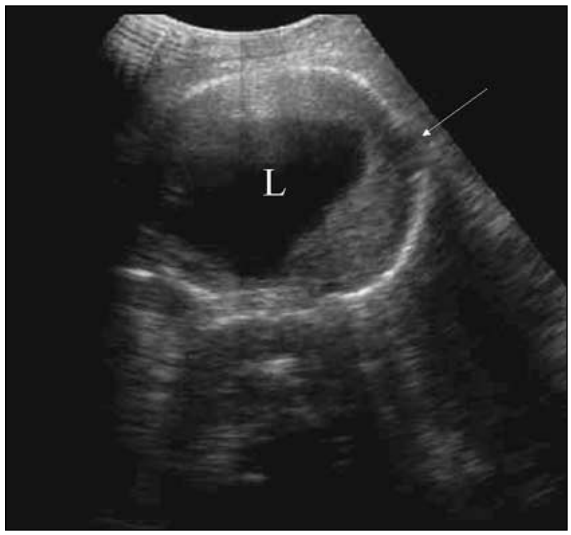


Figure 6. Retroperitoneal hematoma (same patient as in Figures 4 and 5). A large, inhomogeneously hyperechoic, left-sided hematoma (H) is visible adjacent to the aorta. L indicates aortic lumen; and P, psoas muscle.

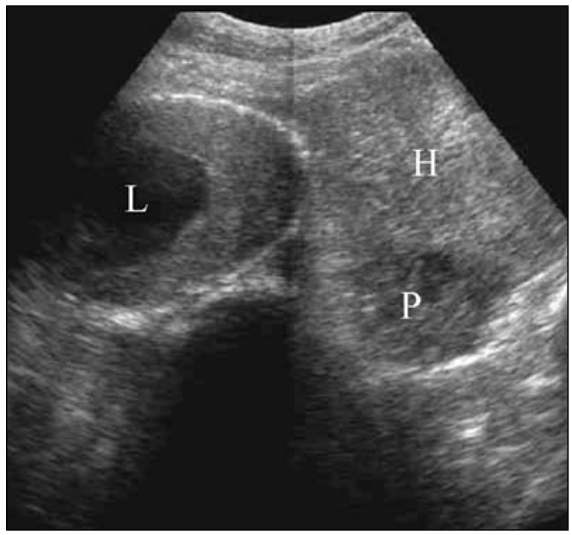


Table 1. Sonographic Findings in 29 Patients With AAA Rupture

Sonographic Finding	Patient																												
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29
AAA deformation		+			+				+		+	+	+		+						+	+	+			+			+
Thrombus inhomogeneity	+	+	+	+			+	+	+		+	+	+	+	+		+	+		+	+			+		+	+	+	
Thrombus interruption		+										+						+										+	+
Floating thrombus layer		+						+				+	+					+				+			+	+			
AAA wall interruption								+				+						+											
Para-aortic hypoechoic area		+						+				+						+											
Retroperitoneal hematoma		+	+	+		+		+		+	+	+	+		+	+	+	+	+	+	+		+	+	+		+	+	
Hemoperitoneum		+	+	+				+	+			+			+	+					+	+							

Many patients do not have typical clinical signs, and just recognizing an AAA in these patients may not be enough to diagnose rupture. An AAA is not uncommon in the elderly. In clinical practice, it may also happen that a patient already known to have an AAA arrives at the emergency department with some vague symptoms: in this case, the referring clinician wants to know whether the aneurysm is actually bleeding.

In the 1980s, with older-generation sonographic scanners, Shuman and colleagues[10] tested the possibilities of 1-minute emergency department sonography. In their experience, there were 60 patients arriving at the emergency department because of suspected AAA rupture. An AAA was found on sonography in 22 patients, but surgery confirmed rupture in 21 (ie, the surgical decision was not correct in 1 patient with an AAA). Extraluminal blood was seen in only 1 patient with rupture. Our experience shows instead that, whenever sonography is used for triage of patients with suspected AAA rupture, a dedicated search for rupture signs should be performed.

Most of the sonographic signs considered in our retrospective analysis were specific, with the major exception of an inhomogeneous lumen thrombus, which is frequently recognized in patients without AAA rupture. It is well known that a stable, nonruptured aneurysm frequently may show an inhomogeneous thrombus with hypoechoic layers interspersed by more echoic layers.[13–15] It should be noted that some of our cases with a ruptured AAA showed marked heterogeneity of the lumen thrombus with irregular hypoechoic areas instead of regularly spaced layers of different echogenicity.

Some signs in our series, such as the floating layer, interrupted thrombus, interrupted aneurysm wall, and para-aortic hypoechoic area, were insensitive, being detected in a minority of ruptured AAAs and being usually recognized in combination in a small subset of patients. To our knowledge, 3 of the signs categorized in the present study have not been reported yet: a floating thrombus layer, focal interruption of the aneurysm wall, and a para-aortic hypoechoic focus at the rupture site. The floating thrombus layer should not be confused with dissection. The latter is characterized by an extended echoic intimal flap, attached on both ends to the aortic wall.[16] Instead, we describe the floating layer as an irregular flap, detached focally from the aneurysm thrombus on 1 side and freely floating within the aneurysm lumen. Thrombus interruption was seen as a defined tear of the lumen-to-thrombus interface, usually spreading within the thrombus itself in combination with inhomogeneously hypoechoic intrathrombotic foci. An AAA outer wall tear was seen as a focal hypoechoic area on the side of retroperitoneal hemorrhage, with interruption of the aneurysm's calcified rim. Finally, the para-aortic hypoechoic area was seen as a small anechoic focus immediately adjacent to the AAA wall, surrounded by retroperitoneal hematoma and indicating the location of the freshest area of bleeding. The importance of this latter sign was confirmed at surgery and, in 2 cases, at CT imaging.

Table 2. Sensitivity and Specificity of Sonographic Findings of Rupture in 388 Patients With AAA

Sonographic Finding	Sensitivity, %	Specificity, %
AAA deformation	41	92
Thrombus inhomogeneity	69	75
Thrombus interruption	17	100
Floating thrombus layer	28	100
AAA wall interruption	10	100
Para-aortic hypoechoic area	14	100
Retroperitoneal hematoma	72	100
Hemoperitoneum	34	100

Aside from these 3 newly reported signs, there were some sonographic findings in our series, such as retroperitoneal hematoma and hemoperitoneum, that were recognizable in most patients with AAA rupture. Retroperitoneal hematoma was seen as a pseudomass of variable echogenicity on 1 side of a ruptured aneurysm. It was usually hyperechoic, was slightly inhomogeneous, and displaced the ipsilateral kidney anteriorly. Hemoperitoneum was found in all but 1 case in combination with retroperitoneal hematoma. Typically, we found a limited amount of hemoperitoneum, confined to a single peritoneal space in most cases. This is probably because patients with more dramatic peritoneal hemorrhage do not survive long enough to undergo sonographic exploration.

We think that sonographic demonstration of rupture signs is important to prove that an identified aneurysm is the true cause of symptoms. It should be noted that many patients do not have typical symptoms; consequently, simply detecting an AAA in these patients may not be sufficient to decide on immediate surgery. The classic clinical triad of hypotension, low back pain, and a pulsatile abdominal mass is present in only a minority of subjects.[1,2] Patients with less specific clinical characteristics may have alternative causes of symptoms and may require adequate, even if rapid, diagnostic workup.

Computed tomography is clearly superior to sonography in assessing all features of AAA rupture. Nevertheless, it is more time-consuming than sonography, also requiring patient transfer to the radiology department. Additionally, adequate CT studies usually require contrast medium administration. Stable patients should undergo CT, but it is believed that surgery should be performed immediately whenever a patient is unstable, has typical symptoms plus sonographic detection of an AAA, or has atypical symptoms plus sonographic demonstration of rupture signs.[2,3] It should be mentioned, however, that survival rates in patients undergoing CT examination after sonography and those who did not in our series were similar, and CT did not seem to cause a relevant time loss in this small group of patients. In addition, many institutions now have a CT scanner in the emergency department, and many have adequate assessment of AAA rupture with unenhanced scans only.

Our study has many limitations to be discussed. First, aneurysms in patients with and without rupture were different, being larger and showing several changes possibly related to aneurysm "aging" in the group with rupture. Nevertheless, it is known that ruptured aneurysms are usually of large dimensions, and comparing the sonographic signs in 2 groups of patients with similar aneurysms is difficult in clinical practice. Second, the sonographic signs considered had different specific relevance because detection of retroperitoneal hematoma clearly has greater importance than detection of aneurysm deformation. In addition, it may be hypothesized that patients in our series had end-stage AAA rupture (fewer than half survived), and our demonstration of several sonographic signs of rupture may mainly reflect the very advanced phase of bleeding. Also, the control group in our series included asymptomatic patients with stable aneurysms, but, from a clinical point of view, it would have been better to enroll patients with suspected AAA rupture (ie, patients with suspected aneurysm rupture but with proved stable aneurysms). Moreover, the overall number of ruptured aneurysms included was clearly limited to allow optimal statistical analysis. Finally, we were clearly aware of ruptured cases, and blinded review of the sonographic studies was impossible. Consequently, no effort was done to determine the interobserver variability and reproducibility of the sonographic findings.

In conclusion, although in the past it was thought that sonography could only show the "presence" of an AAA in a patient with suspected rupture, our series shows that state-of-the-art sonography can identify several signs of AAA rupture. If our findings are confirmed in prospective studies, it may be possible to expedite surgical treatment of hemodynamically unstable patients when these sonographic findings are identified.

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