CASE REPORT

Peritoneal catheter for massive cardiac ascites

Gabriel M Aisenberg

Department of Internal Medicine, The University of Texas Houston Health Science Center, Houston, Texas, USA

Correspondence to
Dr Gabriel M Aisenberg,
gaisenberg@yahoo.com

SUMMARY

Cardiac ascites represents 5% of all causes of ascites. Diuretics and salt restriction remain the cornerstone of management. Large volume paracentesis is needed among patients who do not respond to conservative management. The use of peritoneal catheters to continuously drain steady amounts of ascitic fluid has been generally used in malignant ascites. When the ascites of any other origin is massive and requires many consecutive days of large-volume paracentesis, the use of a catheter may represent a more convenient strategy. We present a patient with cardiac ascites that was successfully managed with a peritoneal catheter.

BACKGROUND

Liver cirrhosis is the cause of ascites in 80% of the cases in the USA.¹ Ascites is infrequently caused by congestive heart failure. It results from transudation from hepatic and peritoneal veins. Massive ascites in the setting of heart disease occurs most frequently in patients with tricuspid valve disease and constrictive pericarditis.² Those patients usually respond to sodium restriction and diuretics; however, if the ascites is massive that approach can cause intravascular volume depletion without significant loss of ascitic fluid. In such cases serial total large volume paracentesis (LVP) can control fluid overload.¹ The use of peritoneal catheters to continuously drain steady amounts of ascitic fluid has been generally used in malignant ascites.³ However, when the ascites of any other origin is massive and requires LVP for many consecutive days, the use of a catheter may represent a more convenient strategy.

CASE PRESENTATION

A 50-year-old white man with a history of hypertension presented to the hospital on 19 October 2006 with a 2-month long gradually increasing abdominal girth, limiting his mobility. He also complained of leg swelling, shortness of breath and orthopnoea. He did not have chest pain, palpitations, change in urine pattern or altered mental status. The patient was taking hydrochlorothiazide 25 mg/day. He admitted to drinking 3–5 glasses of wine a day on average, denying tobacco or recreational drugs consumption. His examination was restricted by his limited mobility from his massive ascites. His blood pressure was 140/103 mm Hg, temperature 36.6°C, pulse 130 bpm and had 22 breaths/min. The admission weight was 204 kg. His legs were swollen with no rash. He had a distended abdomen, non-tender with bulging flanks. Liver and spleen were not palpable. Heart sounds were distant, with a laterally displaced point of

maximum impulse. Lung volumes were low with no crackles or wheezing. No tremor or neurological deficit was observed.

INVESTIGATIONS

Remarkable laboratory data are shown in table 1.

A transthoracic echocardiogram showed dilated ventricles with bilaterally decreased function (ejection fraction less than 20%). Trace pericardial effusion was seen. A first abdominal CT scan showed significant ascites, with normal liver and spleen (figure 1A). Ascites fluid obtained through paracentesis showed proteins 4 g%, albumin 1.9 g%, glucose 108 mg%, lactate dehydrogenase 51, 3000 red cells/ μ l and 58 white cells/ μ l. With difficulty, 3 liters of ascites were drained in that occasion. Culture and cytopathology were negative.

TREATMENT

Therapy with salt restriction, spironolactone 100 mg/day, furosemide 120 mg/day, captopril, digoxin and carvedilol was started. Despite appropriate diuresis, weight and symptoms remained unchanged for 18 days. On 7 November, an 8 French pigtail was inserted in the peritoneal cavity allowing the daily drainage of 5 liters of ascites for 14 days (total 70 litres). For the last 6 days,

Table 1 Remarkable laboratory data

Variable	Value
Serum sodium	137 mEq/l
Serum potassium	4.5 mEq/l
Serum CO ₂	27 mEq/l
Serum creatinine	1.1 mg%
White blood cell count	8900/ μ l
Haemoglobin	13.7 g%
Platelet count	337 000/ μ l
Total proteins	7.5 g%
Serum albumin	3 g%
Bilirubin (total/conjugated)	5.7 mg%/2.5 mg%
Prothrombin time/INR	16.7 s/1.6
ALT	11
AST	27
Alkaline phosphatase	100
HIV serology	Negative
Hepatitis B and C serologies	Negative
Ferritin	Normal
Ammonia level	Normal
Ceruloplasmin	Normal
α -1-Antitrypsin	Normal
α -Fetoprotein	Normal

ALT, alanine transaminase; AST, aspartate transaminase; INR, international normalized ratio.

To cite: Aisenberg GM. *BMJ Case Rep* Published online: [please include Day Month Year] doi:10.1136/bcr-2013-008992

Figure 1 Abdominal CT scan showing significant amount of ascites over an otherwise non-focal, normal sized liver (A) and resolution of the ascites after therapy (B).

albumin was added owing to low blood pressure and a slight increase in creatinine level. Improvement of both the parameters was observed. The catheter was then removed. On 24 November a formal in-house rehabilitation programme was started. In figure 1B a new abdominal CT showed almost no ascites and a normal-sized liver. Liver biopsy showed chronic hepatitis, bridging fibrosis with no cirrhosis, Mallory bodies, viral inclusions or malignancy.

OUTCOME AND FOLLOW-UP

Patient was discharged asymptomatic, on the above medications on 1 December weighing 91 kg. He remained asymptomatic and with stable weight until our last contact 4 months later. Work-up of his underlying cardiopathy was deferred.

DISCUSSION

Congestive hepatopathy may be seen in several forms of cardiac dysfunction as rheumatic valvular disease, constrictive pericarditis, hypertensive and atherosclerotic heart disease being the two former more prevalent in older studies.⁴ However clinically significant cardiac ascites has an overall incidence of around 5% of all ascitic causes. Chemical and cytological study of the fluid supports this diagnosis in our patient.⁵

Literature on the use of catheters in the management of malignant ascites is abundant,³ but patients with cardiac ascites benefited by this procedure are under-represented in larger series⁶ and their clinical features are not fully characterised. Despite the fact that the ascites was not refractory according to its definition,^{1–7} its lack of response after 2 weeks of diuretic therapy was encouraging towards the use of this therapy. The use of transjugular intravenous portosystemic shunt, proven more beneficial than paracentesis, has not been validated in people with severely depressed ventricular function.⁸

In summary, placement of a peritoneal catheter is a valid instrument to rapidly improve symptoms and optimise management of massive cardiac ascites. We hypothesise that this approach may also be successful in patients with other causes of ascites presented as a transudate, such as nephrotic syndrome or liver cirrhosis. However the efficacy and long-term effect of this intervention remain to be supported by the appropriate level of evidence.

Learning points

- ▶ Massive ascites is an infrequent presentation among patients with congestive heart failure.
- ▶ Large volume paracentesis is indicated when there is no response to dietary and pharmacological treatment.
- ▶ Peritoneal catheters to continuously drain steady amounts of ascitic fluid have been generally used in malignant ascites, but not in cardiac ascites.
- ▶ This case shows the successful use of a peritoneal catheter to drain massive cardiac ascites.

Contributors GMA is the single author of this article responsible for patient's care, drafting the manuscript and subsequent editions until the current format.

Competing interests None.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

REFERENCES

- 1 Hou W, Sanyal AJ. Ascites: diagnosis and management. *Med Clin North Am* 2009;93:801–17.
- 2 Shimizu Y. Liver in systemic disease. *World J Gastroenterol* 2008;14:4111–19.
- 3 Lee A, Lau EN, Yeong KY. Indwelling catheters for the management of malignant ascites. *Support Care Cancer* 2000;8:493–99.
- 4 Dunn GD, Hayes P, Breen KJ, et al. The liver in congestive heart failure: a review. *Am J Med Sci* 1973;265:174–89.
- 5 Runyon BA. Cardiac ascites: a characterization. *J Clin Gastroenterol* 1988;10:410–12.
- 6 Po CL, Bloom E, Mischler L, et al. Home ascites drainage using a permanent Tenckhoff catheter. *Adv Perit Dial* 1996;12:235–6.
- 7 Arroyo V, Gines P, Gerbes AL, et al. Definition and diagnostic criteria of refractory ascites and hepatorenal syndrome in cirrhosis. International Ascites Club. *Hepatology* 1996;23:164–76.
- 8 Saab S, Nieto JM, Lewis SK, et al. TIPS versus paracentesis for cirrhotic patients with refractory ascites. *Cochrane database Syst Rev* 2006;4:CD004889.

Copyright 2013 BMJ Publishing Group. All rights reserved. For permission to reuse any of this content visit <http://group.bmj.com/group/rights-licensing/permissions>.
BMJ Case Report Fellows may re-use this article for personal use and teaching without any further permission.

Become a Fellow of BMJ Case Reports today and you can:

- ▶ Submit as many cases as you like
- ▶ Enjoy fast sympathetic peer review and rapid publication of accepted articles
- ▶ Access all the published articles
- ▶ Re-use any of the published material for personal use and teaching without further permission

For information on Institutional Fellowships contact consortiasales@bmjgroup.com

Visit casereports.bmj.com for more articles like this and to become a Fellow