CASE REPORT

Long-term peritoneal port-catheter in a patient with cardiac ascites

Albulena Mecinaj,¹ Frode Reier-Nilsen,² Tri Minh Le,¹ Kjetil Steine¹¹Department of Cardiology, Akershus Universitetssykehus HF, Lorenskog, Norway²Department of Vascular and Thoracic Surgery, Akershus Universitetssykehus HF, Lorenskog, Norway

Correspondence to

Dr Kjetil Steine, kjetil.steine@medisin.uio.no

Accepted 26 April 2017

SUMMARY

A peritoneal port-catheter was inserted in a 70-year-old man because of repeated paracentesis due to cardiac ascites. Instead of frequent hospital admissions, the patient could drain his ascites at home, which dramatically improved his quality of life and enabled him to perform his daily activities.

BACKGROUND

To the best of our knowledge, long-term peritoneal port-catheter (PPC) insertion on patients with heart failure and ascites has not been reported, which will be the substance of this case presentation.

CASE PRESENTATION

A 70-year-old man with a history of smoking, but otherwise fit and well, presented in 2008 with a history of chest pain and was admitted to the local hospital. ECG showed sinus rhythm, but frequent ventricular and supraventricular premature beats. He was thoroughly examined, which included a normal coronary angiogram. The echocardiographic examination, however, showed a hypertrophic and dilated left ventricle (LV) with end-diastolic diameter of 67 mm, LV ejection fraction of 49% and normal-sized left atrial area of 16 cm². The right ventricle (RV) was not described, but assumed to be normal, as echocardiograms later demonstrated normal right-sided function. Blood pressure was 170/95 mm Hg. The echocardiographic findings were attributed to either hypertensive or dilated cardiomyopathy. Treatment with beta-blocker and A2-blocker was initiated.

He was readmitted in 2010 to the same hospital with rapid atrial fibrillation, frequent non-sustained ventricular tachycardia, peripheral oedema and bilateral pleural effusions. A repeated echocardiogram showed a dilated LV with an end-diastolic diameter of 72 mm, a significantly reduced left ventricular ejection fraction (LVEF) of 25%, left atrial area of 35 cm² and normal RV function. Inferior vena cava was dilated with reduced respiratory variation, with estimated systolic pulmonary artery pressure of 55 mm Hg by echo. Right heart catheterisation was therefore performed and showed a postcapillary pulmonary hypertension with pulmonary vascular resistance of 2.5 WU, and mean pulmonary artery pressure and pulmonary capillary wedge pressure of 42 and 29 mm Hg, respectively. A repeated angiogram was normal. Electrical and chemical cardioversion with amiodarone failed. The treatment of his heart failure was medically optimised.

In view of non-sustained ventricular tachycardia, left bundle branch block with a QRS duration of 165 ms, New York Heart Association class III and LVEF <35%, a cardiac resynchronisation therapy device with defibrillator was implanted. Because of poor frequency control of his atrial fibrillation despite amiodarone, an atrioventricular node ablation was attempted three times without success.

Two years later, in 2012, he was readmitted with decompensated heart failure, including tense ascites, dyspnoea at rest and cardiorenal syndrome (creatinine 400 µmol/L). LVEF was estimated at 20%. There was neither biochemical nor ultrasonographic evidence of liver cirrhosis. The clinical findings were managed with abdominal paracentesis and diuretics. From then on he had increasingly frequent hospital admissions, with ascites requiring further paracentesis. At last, he was drained to around 10 L every third week. He reported that after paracentesis, his symptoms improved and lasted for 2–3 days. Then it was followed by a gradual deterioration, with increasing abdominal distension and dyspnoea. He was progressively limited in his daily living.

TREATMENT

In May 2015, a PPC was inserted by a surgeon (FRN) from the surgical department at the same hospital (see online supplementary file for description of the procedure).

OUTCOME AND FOLLOW-UP

With help from his wife, the patient was able to drain 1.2–1.5 L at home every second day. As a consequence, his quality of life improved markedly, and he was able to perform his daily activities. He was no longer confined to his house and has not been admitted to the hospital for 6 months. On follow-up after 6 months, we observed no significant change in his LVEF, but N-terminal pro b-type natriuretic peptide was reduced from 900 to 300 pg/L.

Unfortunately, 7 months after insertion, he acutely deteriorated and died from fulminant streptococcal septicaemia. His wife described a wound on his leg 1 day before admission. There was no clinical evidence of focal infection at the catheter site or peritonitis. This was, therefore, not considered to be a probable cause of his death.

DISCUSSION

Bursi *et al*¹ have shown in the Olmsted study that 79% of patients with preserved and reduced LVEF

CrossMark

To cite: Mecinaj A, Reier-Nilsen F, Le TM, *et al*. *BMJ Case Rep* Published Online First: [please include Day Month Year]. doi:10.1136/bcr-2017-219258

had systolic pulmonary artery pressure above 35 mm Hg, and that these patients had increased morbidity and mortality. In a study by Kim *et al*,² predictors for cardiac ascites formation were tried and identified. One hundred and ninety-six patients, being evaluated for heart transplantation and/or ventricular assist device, were examined, and 29 (15%) had significant ascites.² Elevated right-sided pressures (right atrial pressure and RV end-diastolic pressure) and severe tricuspid regurgitation were directly correlated with the presence of significant ascites. They could not identify a threshold minimum of right atrial pressure elevation to develop significant ascites. In addition, renal function and sodium and albumin levels were also predictors for ascites development. The presence of ascites was associated with significantly higher mortality.²

Chronic exposure of the RV to increased afterload leads to RV systolic dysfunction. This facilitates elevation of pressures in the right atrium and oedema formation. The elevated pressures in the inferior vena cava and hepatic veins will be transmitted to the liver, causing portal hypertension and ascites. It is anticipated that ascites and thus increased intra-abdominal pressure affects the intrathoracic pressure through the diaphragm, preventing venous return.³ Improved cardiac function during paracentesis appears to be caused by an augmented filling of the heart due to a larger venous return.³ Moreover, decompression of the descending aorta decreases the afterload imposed on the LV.⁴ Frequent fluid removal by a PPC may, thus, be better than intermittent paracentesis, as it provides a more continuous and favourable effect on the intrathoracic pressure. Furthermore,⁵ Acosta and coworkers have also demonstrated increased RV end-diastolic volume, RV stroke volume index and RV end-diastolic compliance after paracentesis of cirrhotic patients with ascites. In a study including 27 patients with ascites due to malignancy and liver cirrhosis undergoing PPC, one patient had a major complication with bacterial peritonitis. The clinical success rate, however, was 96%, and the long-term patency rate was 100%.⁶

In summary, we have presented a case with severe cardiac ascites undergoing insertion by PPC. Our patient experienced a marked improvement in quality of life and significant

Learning points

- Peritoneal port-catheter seems to be safe and effective.
- It enables patients to drain ascites at home, reducing hospital admissions.
- It improves quality of life and should be considered in all patients with refractory cardiac ascites.

reduction of hospital admissions. Although we do not consider the patient's death to be caused by PPC, this cannot entirely be excluded. However, the method seems to be safe and should also be considered in patients with refractory cardiac ascites.

Contributors FR-N has inserted the peritoneal port-catheter and has done the follow-ups regarding the procedure and the draining. He also wrote the description of the procedure. TML has made an attribution to acquisition of data and clinical follow-ups. AM initiated the treatment, performed the clinical follow-ups and drafted the manuscript. KS has drafted and revised the manuscript, and has given final approval of the version to be published.

Competing interests None declared.

Patient consent Consent obtained from next of kin.

Provenance and peer review Not commissioned; externally peer reviewed.

© BMJ Publishing Group Ltd (unless otherwise stated in the text of the article) 2017. All rights reserved. No commercial use is permitted unless otherwise expressly granted.

REFERENCES

- 1 Bursi F, McNallan SM, Redfield MM, *et al*. Pulmonary pressures and death in heart failure: a community study. *J Am Coll Cardiol* 2012;59:222–31.
- 2 Kim B, Tan A, Limketkai BN, *et al*. Comparison of outcome in patients with versus without ascites referred for either cardiac transplantation or ventricular assist device placement. *Am J Cardiol* 2015;116:1596–600.
- 3 Guazzi M, Polese A, Magrini F, *et al*. Negative influences of ascites on the cardiac function of cirrhotic patients. *Am J Med* 1975;59:165–70.
- 4 Acosta F, Sansano T, Palenciano CG, *et al*. Hemodynamic effects of rapid paracentesis on liver transplantation. *Transplant Proc* 2000;32:2661.
- 5 Acosta F, Sansano T, Palenciano CG, *et al*. Effects of rapid paracentesis on right ventricular-arterial coupling in liver transplantation. *Transplant Proc* 2005;37:3867–8.
- 6 Savin MA, Kirsch MJ, Romano WJ, *et al*. Peritoneal ports for treatment of intractable ascites. *J Vasc Interv Radiol* 2005;16:363–8.

Copyright 2017 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