ESC European Society of Cardiology
European Heart Journal - Case Reports
doi:10.1093/ehjcr/ytab031

CASE REPORT
Heart failure

Venoarterial extracorporeal membrane oxygenation as bridge to effective treatment in a 19-year-old woman with acute adrenal crisis: a case report

Enzo Lüsebrink[1,2†], Kathrin Krieg [1,2†], Steffen Massberg [1,2], and Martin Orban [1,2*]

[1]Intensive Care Unit, Medizinische Klinik und Poliklinik I, Klinikum der Universität München, Marchioninistraße 15, 81377 Munich, Germany; and [2]DZHK (German Center for Cardiovascular Research), partner site Munich Heart Alliance, Medizinische Klinik und Poliklinik I, Klinikum der Universität München, Marchioninistraße 15, 81377 Munich, Germany

Received 12 August 2020; first decision 28 September 2020; accepted 9 January 2021

Background Adrenal crisis is an acute life-threatening exacerbation of the Addison's disease or primary adrenal insufficiency (PAI) and is associated with a high mortality rate. It can be the first manifestation of adrenal insufficiency and is caused by a critical lack of glucocorticoids.

Case summary Here, we report the case of a 19-year-old woman presenting to the emergency room with unspecific symptoms, i.e. lethargy, fatigue, arthralgia, dyspnoea, and hypotension. The patient's examination showed major pericardial effusion resulting in cardiac tamponade requiring immediate pericardiocentesis. In the further course, acute right heart failure and progressive cardiogenic shock occurred. Due to recurrent bradycardia and finally asystole, the patient had to be resuscitated for 15 min in total until return of spontaneous circulation. However, non-invasive haemodynamic stabilization of the patient was not successful. Hence, venoarterial extracorporeal membrane oxygenation (VA-ECMO) was used as salvage intervention to provide temporary circulatory support. We diagnosed an Addison crisis as first manifestation of her previously unknown Addison's disease. An appropriate substitution therapy with hydrocortisone and fludrocortisone was immediately initiated and the patient's condition rapidly improved. After a total in-hospital stay of 4 weeks, she recovered completely and could be discharged from hospital.

Discussion An Addison crisis requires rapid diagnosis and immediate treatment to end a life-threatening condition caused by critical glucocorticoid deficiency. In patients with non-specific symptoms, such as fatigue, hypotension, weight loss, and hyponatraemia, adrenocortical insufficiency should be considered as differential diagnosis. If patients suffer from an Addison crisis, clinical suspicion requires immediate substitution of hydrocortisone as this is essential for patient's survival. Venoarterial extracorporeal membrane oxygenation therapy can serve as a bridge to diagnosis and effective treatment in patients requiring temporary cardiopulmonary support, especially as salvage intervention for patients in cardiogenic shock. To our knowledge, this is the first case of a young patient with acute Addison crisis and cardiogenic shock, who was successfully salvaged by VA-ECMO support.

Keywords Addison's diseases • Addison crisis • Venoarterial extracorporeal membrane oxygenation • Case report

* Corresponding author. Tel: +49 4400 75221, Email: MartinOrban@gmail.com;Martin.Orban@med.uni-muenchen.de
†These authors contributed equally to the study.
Handling Editor: Mohamed Hassan
Peer-reviewers: Elad Asher and Milenko Zoran Cankovic
Compliance Editor: Stefan Simovic
Supplementary Material Editor: Deepti Ranganathan

Learning points

- An adrenal crisis can be the first manifestation of adrenal insufficiency and can present with non-specific symptoms, such as fatigue, general weakness, hypotension, weight loss, gastrointestinal symptoms (nausea, vomiting, and abdominal pain), arthralgia, hyponatraemia, and dehydration.
- Pericardial effusion can be a serious complication in patients suffering from Addison crisis.
- Early and appropriate substitution of hydrocortisone, i.e. initial intravenous bolus dose of 100 mg hydrocortisone followed by a subsequent continuous intravenous infusion of hydrocortisone 200 mg/24 h, is essential for patient's survival in Addison crisis.
- In patients suffering from Addison crisis with accompanying progressive cardiogenic shock, venoarterial extracorporeal membrane oxygenation can serve as bridge to effective treatment.

Introduction

Primary and secondary adrenal insufficiency are rare diseases. The primary adrenal insufficiency (PAI) is caused by a malfunction of the adrenal gland itself, often triggered by an autoimmune-mediated adrenalitis.[1,2] Secondary adrenal insufficiency is the result of an impaired hypothalamic–pituitary–adrenal axis and a consecutive disruption of adrenocorticotropic hormone (ACTH) synthesis. Patients suffering from PAI are at higher risk of an adrenal crisis compared to patients with secondary adrenal insufficiency due to differences in the severity of glucocorticoid deficiency.[3] An Addison crisis can present with very unspecific symptoms, leading to a large number of possible differential diagnoses. The incidence is reported to be 5–8 per 100 patient-years.[4] Here, we report the case of a 19-year-old woman with acute life-threatening Addison crisis as first manifestation of PAI, who required venoarterial extracorporeal membrane oxygenation (VA-ECMO) as salvage intervention in progressive cardiogenic shock.

Case presentation

A 19-year-old woman was admitted to the emergency room of a general hospital with fatigue, lethargy, arthralgia, dyspnoea, and hypotension. The patient showed a positive shock index (blood pressure 82/55 mmHg, heart rate 132/b.p.m.) and strongly reduced overall condition. She had oral iron supplementation with iron(II)-glycine sulfate (ferro sanol duodenal 1 × 100 mg Fe/d) for iron deficiency anaemia and no other medication. Physical examination on admission showed reduced general condition, very pale skin, no pathologic murmurs, muffled breath sounds on both sides, diffuse abdominal pain with weakened peristalsis and without palpable resistance, no peripheral oedema, no focal neurological deficit, and full orientation. The electrocardiogram (ECG) revealed sinus tachycardia and low QRS voltage in all leads, but no signs of acute ischaemia, and no bundle branch block. A cardiomegaly was diagnosed by chest X-ray and subsequent transthoracic echocardiography showed pericardial effusion compressing right atrium and ventricle consistent with cardiac tamponade (*Figure 1A* and *B*). An immediate needle pericardiocentesis was performed, and a total of 800 mL serous pericardial effusion

Timeline

Initial presentation	Presentation with non-specific symptoms including fatigue, lethargy, arthralgia, dyspnoea, and hypotension Pericardial effusion compressing right atrium and ventricle consistent with cardiac tamponade Development of progressive cardiogenic shock with increasing need for catecholamine support Bradycardia and asystole	Emergency pericardiocentesis and relieve of total of 800 mL serous pericardial effusion Catecholamine therapy Resuscitation with return of spontaneous circulation (ROSC) after 15 min Implantation of portable venoarterial extracorporeal membrane oxygenation (VA-ECMO) system
Diagnosis phase and beginning of effective treatment	Blood biochemistry showed a low cortisol level and elevated adrenocorticotropic hormone levels Performance of a Synacthen test with inadequate increase in serum cortisol. Positive 21-Hydroxylase antibodies finally confirming diagnosis of Addison's disease	Suspected Addison crisis and immediate start of an appropriate substitution therapy with hydrocortisone and fludrocortisone
Clinical stabilization phase	The patient's condition start to improve under adequate substitution therapy Cardiac function completely recovered	Ongoing substitution therapy with gradual tapering of the hydrocortisone dosage according to clinical status of the patient Removal of VA-ECMO and extubation
Discharge	Patient has completely recovered and could be discharged	Extensive patient training in the handling of the disease Hand out of emergency medical kit and emergency pass

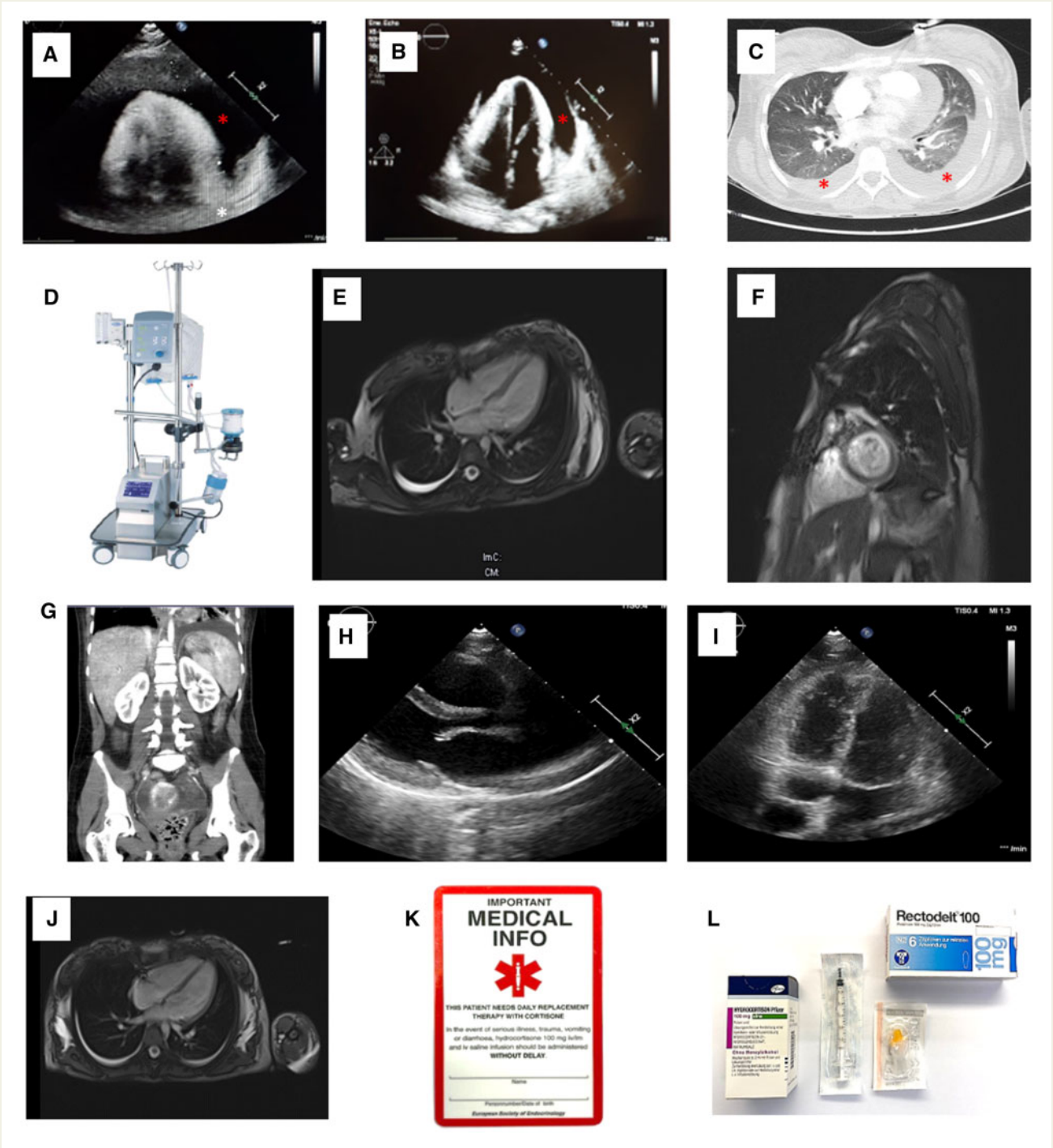

Figure 1 (*A* and *B*) Major circular pericardial effusion (*) with compression of right atrium and ventricle. (*C*) Bilateral pleural effusion (*) in acute Addison crisis. (*D*) Venoarterial extracorporeal membrane oxygenation system (SCPC System, Sorin, LivaNova Deutschland GmbH, München, Germany). (*E* and *F*) Cardiovascular magnetic resonance imaging without evidence of myocarditis, in particular, missing cardiac oedema and late enhancement. (*G*) Computed tomography of the abdomen showing inconspicuous kidneys and adrenal glands on both sides. (*H* and *I*) Echocardiographic control after needle pericardiocentesis and beginning of substitution therapy. (*J*) Regressing pleural effusion after initiation of substitution therapy. (*K*) Emergency card including personal data and emergency treatment instructions. (*L*) Exemplary emergency kit including hydrocortisone powder and a solution for preparing an intramuscular injection and cortisone suppositories.

was relieved without leaving the pericardial catheter in the pericardium. Blood biochemistry and haematology showed elevated inflammatory parameters (leucocytes 14.4 G/L, C-reactive protein 78.7 mg/L, and procalcitonin 2.97 µg/L), manifest hypothyroidism (thyroid stimulating hormone 15.54 mIU/L and free triiodothyronine 1.01 ng/L), and hyponatraemia (123 mmol/L) (*Table 1*). Computed

Table 1 Laboratory values and diagnostic tests

Parameters	Results	Normal range
Leucocytes	14.1 G/L	3.9–10.2 G/L
Procalcitonin	2.97 µg/L	<0.1 µg/L
C-reactive protein	78.7 mg/L	<0.5 mg/L
Interleukin 6	4913 pg/mL	<15.0 pg/mL
Sodium	123 mmol/L	135–146 mmol/L
Potassium	3.93 mmol/L	3.5–5.1 mmol/L
Thyroid stimulating hormone	15.54 mIU/L	0.51–4.30 mIU/L
Free trijodthyronin	1.01 ng/L	1.71–3.71 ng/L
Free thyroxine	0.94 ng/L	1.71–3.71 ng/L
Thyroid peroxidase antibodies	Negative	
Thyrotropin receptor antibodies	Negative	
Thyroglobulin autoantibodies	Negative	
21-Hydroxylase antibodies	Positive	
Cortisol	0.4 µg/dL	>18 µg/dL
Adrenocorticotropic hormone	1021 pq/mL	10–60 pq/mL
First Synacthen test 0'	2.8 µg/dL	
First Synacthen test 60'	3.2 µg/dL	>18 µg/dL
Rheumatoid factor	6 U/mL	<14 U/mL
Parvo B19-Virus-IgM	Negative	
Anti-EBV-VCA-IgG/M	Negative	
Anti-EBV-EA-IgG	Negative	
Anti-VZV-IgG	Positive	
Anti-VZV-IgM	Negative	
Anti-HHV6-IgG	Positive	
Anti-HHV6-IgM	Negative	
Anti-Influenza-A-IgA	Negative	
Anti-Influenza-A-IgG	Negative	
Anti-Influenza-B-IgA	Negative	
Anti-Influenza-B-IgG	Negative	
Anti-Mumps virus-IgG	Positive	
Anti-Mumps virus-IgM	Negative	
Anti-Rotavirus-IgG	Positive	
Anti-Rotavirus-IgM	Negative	
Human immunodeficiency virus	Negative	
Anti-Hantavirus-IgG	Negative	
Anti-Hantavirus-IgM	Negative	
QuantiFERON test	Negative	

tomography revealed bilateral pleural effusions (*Figure 1C*) and ascites, compatible with polyserositis.

The patient developed progressive shock with an increasing need for catecholamine support. Due to recurrent bradycardia and finally asystole, the patient had to be resuscitated for 15 min until we achieved return of spontaneous circulation. Since haemodynamic stabilization could not be achieved despite maximum non-invasive intensive care therapy, VA-ECMO) was used as salvage intervention to provide temporary circulatory support (*Figure 1D*). A portable VA-ECMO system was implanted by our mobile ECMO team directly on site and the patient was then transferred to the intensive care unit of our university hospital. However, the cause of circulatory failure initially remained unclear. After implantation of VA-ECMO, transthoracic echocardiography showed normal left ventricular function and normal left ventricular size. In contrast, right ventricle was dilated and showed reduced function. The cardiovascular magnetic resonance imaging did not detect any cardiomyopathy, in particular, we found no evidence suggesting myocarditis or an arrhythmogenic right ventricular cardiomyopathy (*Figure 1E and F*). Based on the suspected polyserositis, an extensive rheumatological/immunological diagnosis was performed. Diagnostic tests for antinuclear antibodies, anti-double-stranded DNA antibodies, rheumatic factor, cyclic citrulline, parvovirus B19 IgG and IgM, adeno-, cytomegalovirus and coxsackievirus IgG and IgM, and human immunodeficiency virus were negative as was the QuantiFERON test for tuberculosis (*Table 1*). Due to low cortisol level (0.4 µg/dL) adrenal insufficiency was proven. Adrenocorticotropic hormone levels were elevated (1021 pg/mL) which indicated PAI. In addition, an ACTH-stimulation test was performed and confirmed adrenal insufficiency as there was an insufficient increase in serum cortisol (*Table 1*). Serum cortisol levels <18.0 µg/dL (<500 nmol/L) at 30 or 60 min in an ACTH-stimulation test are sufficient to confirm adrenal insufficiency.[5] Furthermore, 21-Hydroxylase antibodies as markers of an autoimmune Addison's disease were positive and finally confirmed diagnosis. Computed tomography of the abdomen revealed no morphological alteration of kidneys and adrenal glands on both sides (*Figure 1G*). We immediately started an appropriate substitution therapy with hydrocortisone and fludrocortisone according to the substitution scheme illustrated in *Table 2* with initial intravenous bolus dose of 100 mg hydrocortisone followed by a subsequent continuous intravenous infusion of hydrocortisone 200 mg/24 h as long as the patient required catecholamines. This was followed by a gradual tapering of the hydrocortisone dosage within the next days according to clinical status of the patient. The patient's condition improved, VA-ECMO could be removed and the patient was extubated. The right ventricular function recovered completely, and pericardial effusion was no longer detectable (*Figure 1H and I*). In line with this, the initial low-voltage diminished on ECG. Likewise, pleural effusions (*Figure 1J*) and ascites almost completely regressed therefore no drainage

Table 2 Substitution scheme of hydrocortisone and fludrocortisone in this patient with Addison crisis

Timeline	Dose of hydrocortisone
Day 0	100 mg intravenous (bolus) and 200 mg/24 h intravenous[a]
Day 1	150 mg/24 h intravenous[b]
Day 2	100 mg/24 h intravenous[b]
Day 3–7	50 mg/day given 3 times daily per os 20–20–10 mg[b]
From Day 7	35 mg/day given three times daily per os 20–10–5 mg[b]
Timeline	Dose of fludrocortisone
From Day 0	0.1 mg/day given in the morning per os

[a]Continuous intravenous infusion of hydrocortisone 200 mg/24 h as long as the patient required catecholamines.
[b]Continuous tapering of hydrocortisone within the next days according to clinical status of the patient.

throughout the hospital stay was necessary. The transthoracic echocardiography at discharge confirmed normal biventricular function with normal left and right ventricular size. Initially, short-term levothyroxine substitution was necessary due to manifest hypothyroidism. The thyroid function, however, normalized in the course of the disease and finally no further substitution was necessary. In the absence of clinical evidence of a polyglandular autoimmune syndrome (no diabetes, no autoimmune hepatitis, no permanent hypothyroidism, no mucocutaneous candidosis, and no hypogonadism) no further antibody diagnosis was necessary. The intravenous hydrocortisone therapy was gradually reduced and then given three times daily per os (*Table 2*).

After a total stay of 4 weeks in the hospital, the patient has completely recovered and could be discharged. Prior discharge, she received an extensive patient training in the handling of the disease, in particular, adjustments to the cortisone dose in situations with an increased need for cortisol (fever, infection, vomiting, diarrhoea, and surgical interventions) and the timely detection and treatment of an Addison crisis. She was given an emergency pass including personal data and emergency treatment instructions (*Figure 1K*) as well as an emergency medical kit with hydrocortisone powder and a solution for preparing an intramuscular injection as well as cortisone suppositories (*Figure 1L*). In addition, the patient's twin sisters were also contacted and testing via the endocrinological outpatient clinic was recommended. The patient is doing very well to this day and symptom-free.

Discussion

Primary adrenocortical insufficiency is caused by a damage to the adrenal glands, often induced by an autoimmune adrenalitis (Morbus Addison)—either isolated or as part of a polyglandular autoimmune syndrome.[6,7] Patients suffering from PAI often present with unspecific symptoms, such as general fatigue and weakness, hypotension, weight loss, gastrointestinal symptoms (nausea, vomiting, and abdominal pain), arthralgia, and dehydration.[4,5] Other characteristic abnormalities are hyponatraemia, hyperkalaemia, hyperpigmentation, and hypothyroidism,[8] which also existed in the present case. In these constellations, adrenocortical insufficiency should at least be considered as differential diagnosis. Polyserositis with mostly pleural and also pericardial effusion has been less frequently described but can also be an unspecific symptom of PAI especially if the latter is part of a polyglandular autoimmune syndrome.[9] Unfortunately, in rare cases, pericardial effusion or even pericardial tamponade can be the first manifestation of PAI.[10,11] However, temporary heart failure caused by an Addison's disease has been very infrequently described in literature so far.[12] The pathophysiological mechanism underlying this temporary, reversible cardiac dysfunction remains insufficiently understood and such a patient condition must be considered as urgent emergency.[13] When patients suffer from an Addison crisis, immediate substitution of hydrocortisone due to clinical suspicion is essential for patient's survival[1,3,14,15] and cardiac abnormalities in patients with untreated Addison's disease have already been reported to regress after steroid therapy.[13]

Percutaneous circulatory support devices, in particular VA-ECMO, have emerged as an established salvage intervention for patients in cardiogenic shock and provide temporary circulatory support until other treatments are effective and enable recovery or serve as a bridge to ventricular assist devices, heart transplantation, or decision-making.[16] Here, VA-ECMO support was successfully used as a bridge to diagnosis, i.e. Addison crisis as first manifestation of a previously unknown Addison's disease, and effective treatment, i.e. appropriate substitution therapy with hydrocortisone and fludrocortisone. Kang *et al.*[17] reported a case of a 31-year-old man with hypothyroidism inducing adrenal crisis who was also successfully salvaged by VA-ECMO support and whose subsequent autoimmune work-up confirmed the diagnosis of autoimmune polyglandular syndrome type 2 with positive antiperoxidase antibodies, supporting the diagnosis of Hashimoto's thyroiditis. Additionally, Krishnamoorthy *et al.*[18] reported a case of a 21-year-old man suffering from acute Addison crisis who was successfully treated with a biventricular assist device, i.e. a CentriMag system (Levitronix LLC, Waltham, MA, USA). In comparison to such a system, VA-ECMO is less invasive and easier to implant and to remove, i.e. by using closure devices.[19] This is particularly advantageous if a temporary and reversible cardiac dysfunction due to an Addison crisis can be assumed.

Conclusion

Our case report shows that an Addison crisis requires immediate treatment to end a life-threatening condition caused by critical glucocorticoid deficiency. In patients with non-specific symptoms, such as fatigue, hypotension, weight loss, and hyponatraemia, adrenocortical insufficiency should be considered as differential diagnosis. If patients suffer from an Addison crisis, immediate substitution of hydrocortisone in adequate stress dosage is essential for patient's survival. Here, the VA-ECMO therapy can serve as a bridge to diagnosis and effective treatment in patients requiring temporary cardiopulmonary support, especially as salvage intervention for patients in cardiogenic shock.

Lead author biography

Enzo Lüsebrink graduated in economics from the University of Mannheim and received a doctor's degree at the University of Mannheim. He achieved his license to practice medicine at the University of Bonn with internships at the university hospitals of Bonn, Berlin, Hamburg, Heidelberg, and Munich. He finished his medical doctoral thesis at the University of Bonn. At present, he is a resident physician for cardiology and internal medicine at the university hospital of the Ludwig Maximilians-University Munich and is working in the cardiac intensive care unit.

Supplementary material

Supplementary material is available at *European Heart Journal - Case Reports* online.

Acknowledgements

We thank the nursing staff of our Intensive Care Unit for all their work. We would also like to thank Prof. Christoph Josef Auernhammer, Endocrinology, Medizinische Klinik und Poliklinik IV, Klinikum der Universität München, Munich, Germany for his support and expertise in this case.

Slide sets: A fully edited slide set detailing this case and suitable for local presentation is available online as Supplementary data.

Consent: The authors confirm that written consent for submission and publication of this case report including images and associated text has been obtained from the patient in line with COPE guidance.

Conflict of interest: M.O. received speaker honoraria from AstraZeneca, Bayer Vital, and SedanaMedical, outside the submitted work. All other authors declare no conflict of interest.

Ethical standards: All ethical standards were met in writing and submitting this correspondence.

Funding: none declared.

References

1. Husebye ES, Allolio B, Arlt W, Badenhoop K, Bensing S, Betterle C et al. Consensus statement on the diagnosis, treatment and follow-up of patients with primary adrenal insufficiency. *J Intern Med* 2014;**275**:104–115.
2. Simpson H, Tomlinson J, Wass J, Dean J, Arlt W. Guidance for the prevention and emergency management of adult patients with adrenal insufficiency. *Clin Med* 2020;**20**:371–378.
3. Dineen R, Thompson CJ, Sherlock M. Adrenal crisis: prevention and management in adult patients. *Ther Adv Endocrinol* 2019;**10**:204201881984821.
4. Betterle C, Presotto F, Furmaniak J. Epidemiology, pathogenesis, and diagnosis of Addison's disease in adults. *J Endocrinol Invest* 2019;**42**:1407–1433.
5. Yanase T, Tajima T, Katabami T, Iwasaki Y, Tanahashi Y, Sugawara A et al. Diagnosis and treatment of adrenal insufficiency including adrenal crisis: a Japan Endocrine Society clinical practice guideline [Opinion]. *Endocr J* 2016;**63**:765–784.
6. Manthri S, Bandaru S, Ibrahim A, Mamillapalli CK. Acute pericarditis as a presentation of adrenal insufficiency. *Cureus* 2018;**10**:e2474.
7. Neary N, Nieman L. Adrenal insufficiency: etiology, diagnosis and treatment. *Curr Opin Endocrinol Diabetes Obes* 2010;**17**:217–223.
8. Grinspoon SK, Biller BM. Clinical review 62: laboratory assessment of adrenal insufficiency. *J Clin Endocrinol Metab* 1994;**79**:923–931.
9. Algün E, Erkoç R, Kotan C, Güler N, Sahin I, Ayakta H et al. Polyserositis as a rare component of polyglandular autoimmune syndrome type II. *Int J Clin Pract* 2001;**55**:280–281.
10. Tucker WS Jr, Niblack GD, McLean RH, Alspaugh MA, Wyatt RJ, Jordan SC et al. Serositis with autoimmune endocrinopathy: clinical and immunogenetic features. *Medicine (Baltimore)* 1987;**66**:138–147.
11. Taxter AJ, Bellin MD, Binstadt BA. Pericarditis as the presenting feature of adrenoleukodystrophy. *Pediatrics* 2011;**127**:e777–e780.
12. Wolff B, Machill K, Schulzki I, Schumacher D, Werner D. Acute reversible cardiomyopathy with cardiogenic shock in a patient with Addisonian crisis: a case report. *Int J Cardiol* 2007;**116**:e71–e73.
13. Fallo F, Betterle C, Budano S, Lupia M, Boscaro M, Sonino N. Regression of cardiac abnormalities after replacement therapy in Addison's disease. *Eur J Endocrinol* 1999;**140**:425–428.
14. Bornstein SR, Allolio B, Arlt W, Barthel A, Don-Wauchope A, Hammer GD et al. Diagnosis and treatment of primary adrenal insufficiency: an endocrine society clinical practice guideline. *J Clin Endocrinol Metab* 2016;**101**:364–389.
15. Prete A, Taylor AE, Bancos I, Smith DJ, Foster MA, Kohler S et al. Prevention of adrenal crisis: cortisol responses to major stress compared to stress dose hydrocortisone delivery. *J Clin Endocrinol Metab* 2020;**105**:2262–2274.
16. Lüsebrink E, Orban M, Kupka D, Scherer C, Hagl C, Zimmer S et al. Prevention and treatment of pulmonary congestion in patients undergoing veno-arterial extracorporeal membrane oxygenation for cardiogenic shock. *Eur Heart J* 2020;**41**:3753–3761.
17. Kang MS, Sandhu CS, Singh N, Evans T. Initiation of levothyroxine in a patient with hypothyroidism inducing adrenal crisis requiring VA ECMO: a tale of preventable disaster. *BMJ Case Rep* 2019;**12**:e230601.
18. Krishnamoorthy A, Mentz RJ, Hyland KA, McMillan EB, Patel CB, Milano CA et al. A crisis of the heart: an acute reversible cardiomyopathy bridged to recovery in a patient with Addison's disease. *ASAIO J* 2013;**59**:668–670.
19. Lüsebrink E, Stremmel C, Stark K, Petzold T, Hein-Rothweiler R, Scherer C et al. Percutaneous decannulation instead of surgical removal for weaning after venoarterial extracorporeal membrane oxygenation-a crossed Perclose ProGlide closure device technique using a hemostasis valve Y connector. *Crit Care Explor* 2019;**1**:e0018.