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ORIGINAL ARTICLE

Development of Acute Decompensated Heart Failure Among Hospital Inpatients: Incidence, Causes and Outcomes

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Background We aimed to investigate the incidence, precipitants, and outcomes of acute decompensated heart failure (ADHF) that develops during the inpatient stay.

Methods We undertook a case-control study in the medical, oncology, surgical, and orthopaedic wards of a tertiary referral hospital (February–May, 2016). Patients aged $\geq$18 years who developed ADHF during their inpatient stay were enrolled as cases. One control patient was matched to each case by age, gender, presenting complaint/surgery performed and co-morbidities. Multivariate regression was employed to determine variables associated with ADHF.

Results The incidence of ADHF was 1.0% of patients. Eighty cases were well-matched to 80 controls ($p > 0.05$). ADHF precipitants comprised infection (30%), inappropriate intravenous (IV) fluid and medication management (23.8% and 8.8%, respectively), tachyarrhythmia (12.5%), ischaemic heart disease (8.8%), renal failure (1.3%), and other/unclear causes (15%). Three variables were associated with ADHF: not having English as the preferred language (OR 3.5, 95%CI 1.2–9.8), a history of ischaemic heart disease (OR 3.3, 95% CI 1.2–9.1), and the administration of >2000 ml of IV fluid on the day before the ADHF (OR 8.3, 95%CI 1.5–48.0). The day before the ADHF, cases were administered significantly more IV fluids than controls (median 2,757.5 versus 975 ml, $p = 0.001$). Medication errors mostly related to failure to restart regular diuretics. Cases had significantly greater length of stay (median 15 versus 6 days, $p < 0.001$) and mortality (12.5% versus 1.3%, $p = 0.01$).

Conclusions New onset ADHF is common and a substantial proportion of cases are iatrogenic. Cases experience significantly increased length of hospital stay, morbidity, and mortality.

Keywords Acute decompensated heart failure • Intravenous fluids • Inpatients • Iatrogenic

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Introduction

Acute decompensated heart failure (ADHF) is a syndrome of dyspnoea associated with the rapid accumulation of fluid within the pulmonary interstitial and alveolar spaces as a consequence of acutely raised cardiac filling pressures [1]. Patients often require multi-drug treatment regimens and may experience decreasing function, long hospital and intensive care admissions, and high rates of rehospitalisation and mortality [2]. Its increasing global incidence has resultant individual and health care costs and leads to lost productivity [3–6].

While ADHF is a common reason for presentation to hospital, it can also develop during the inpatient stay where it is associated with higher mortality [7,8]. The precipitants of ADHF include myocardial ischaemia, infection, arrhythmias, and inappropriate medication, fluid or salt administration [9]. Hospital inpatients are subject to all of these factors, some of which are iatrogenic and potentially preventable. Intravenous (IV) fluid administration is one iatrogenic precipitant specific to the hospital setting.

Few studies have investigated the causes of ADHF when it develops in the inpatient setting. This is despite associated poorer outcomes and increases in both length of stay and mortality [7,10,11–14]. We aimed to investigate the incidence, precipitants, and outcomes of ADHF that develops during the inpatient stay. We hypothesised that iatrogenic causes, including inappropriate IV fluid and medication management, comprise a substantial proportion of overall causes. The findings will identify inpatients at risk of ADHF and inform improvements in clinical practice that aim to mitigate this risk and improve patient care.

Material and Methods

We undertook a case-control study in the medical, surgical and orthopaedic inpatient wards of the Austin Hospital between February 1 and May 31, 2016. The Austin Hospital is a metropolitan, university affiliated, tertiary referral centre in Melbourne, Australia. The study was approved by the Austin Health Human Research Ethics Committee. As data were collected from the medical record and the treating staff, informed patient consent was not required.

Patients were enrolled as cases if they were medical (including oncology) or surgical (including orthopaedic) inpatients, aged 18 years or more, and were treated for ADHF that occurred during their inpatient stay. A history of heart failure was not required. Patients were excluded if they had an admission diagnosis inclusive of ADHF, right-sided heart failure only or acute respiratory distress syndrome.

In this study, a diagnosis of ADHF required both diagnostic criteria (shortness of breath/paroxysmal nocturnal dyspnoea/orthopnoea/fatigue as well as inspiratory crackles and/or chest X-ray changes) and treatment criteria (drug treatment with diuretics/nitrates/morphine and/or respiratory pressure support with positive pressure ventilation/intubation) [1].

Each case was matched with one control patient. The control entrance criteria were the same as the case criteria except that controls did not experience ADHF during their inpatient stay. Controls were matched by age (±10 years), gender, reason for admission (same admission presentation/diagnosis or same operation/procedure) and important co-morbidities (e.g. known cardiac disease/risk factors, diabetes, anaemia). Other important co-morbidities likely to contribute to the risk of developing ADHF, increase length of stay, or require patients to receive IV fluids were matched as closely as possible. This included conditions such as end-stage kidney or liver disease, alcoholism, and cancer.

Both cases and controls were enrolled retrospectively. A consecutive sample of all patients meeting the case entrance criteria during the study period was enrolled. Cases within the previous 24–48 hours were identified by attendance of investigators (LP, TW and AP) at medical handover and by enquiry of ward nurses and treating medical teams. At other times, staff were encouraged to contact the investigators should new cases arise. The Austin Hospital's Medical Emergency Team (MET) call database was also examined for potential cases that may have otherwise been missed.

Control patients were identified by retrospective screening of electronic inpatient lists. The medical records of potentially suitable controls were examined and the patient was enrolled if they met the study entrance criteria and were a suitable match.

A data collection document was designed by the investigators specifically for the study. Data collection was largely undertaken by the first author (LP, assisted by TW and AP) and involved explicit extraction of a range of data items from the patients' electronic medical records. Patient characteristic data included demographics, medical history, medications on admission, smoking status and the reason for hospitalisation and/or operations/procedures performed. Clinical data included vital signs, medication changes, weight and fluid balance, IV fluid administration and laboratory investigations. Control IV fluid administration data were taken at the same number of days post admission/surgery as their respective case. Outcome data included length of hospital stay and discharge status (death, home, rehabilitation/subacute care).

For ADHF cases, additional data included clinical signs and symptoms of ADHF and details of the ADHF management. The inpatient medical and nursing staff were asked to determine the causes of ADHF. This was a clinical decision and based upon a critical review of the way in which the patient had been managed, with particular interest in recent drug and IV fluid administration. In cases of uncertainty, a panel of investigators (LP, DT, DJ and LB) reviewed each case together and made a final determination. If a precipitant could not be clearly determined, none was assigned.

The primary study endpoints were the incidence and precipitants of ADHF. The secondary endpoints were the clinical outcomes of treatment required for ADHF cases, mortality, and length of acute hospital stay.

The sample size calculation was based upon an exposure rate of 50% among the controls (e.g. proportion with a potential risk factor for ADHF) and a clinically significant relative risk of 3.0 among the cases. Given these values, at least 74 cases and 74 matched controls were required (level of significance 0.05, two-sided, power 0.85). To account for some assumptions in this calculation, we planned to enrol 80 patients in each group.

The data are reported descriptively (percentages, means, medians). The case and control groups were compared statistically using Chi square, Student's t-test and the Mann-Whitney U test, where appropriate. These calculations employed SPSS for Windows statistical software (version 22.0, SPSS Inc., Armonk, NY, USA).

Multivariate logistic regression was undertaken to determine patient and management variables associated with the development of ADHF. Only variables with a p value of <0.1 in the univariate analyses were included in the regression model. The regression analyses employed Intercooled Stata 6.0 for Windows 98/95/NT (Stata Corporation, College Station, TX, USA). Cox regression analysis was employed to examine the cumulative hazard for death (SPSS for Windows statistical software. Version 22.0, SPSS Inc., Chicago, Armonk, NY, USA). The level of significance was 0.05.

Results

During the 4-month study period, 80 (1.04%) of 7,678 patients admitted to the hospital's medical, oncology, surgical and orthopaedic wards met the study criteria for the development of ADHF as an inpatient. Half of the patients were male, most were elderly and there were similar numbers of medical and surgical patients. The case and control groups were well matched: both groups had a mean age of 76.2 years and included 40 males and 43 medical (non-surgical) patients. The group mean blood pressure and heart rate on admission did not differ ($p > 0.05$).

The precipitants of ADHF included infection, inappropriate IV fluid and medication management, tachyarrhythmia, myocardial ischaemia, renal failure, and other or unclear causes (Table 1). The precipitant was iatrogenic in 26 (32.6%) cases.

For each of the 19 cases (10 medical, 9 surgical patients) deemed to have inappropriate IV fluid management, it was clear that this was the actual precipitant. For most of these cases, the treating staff nominated fluid management as the clear precipitant. For the remainder no other precipitant was evident and justification for the large volumes of fluid administered could not be determined by careful scrutiny of the medical records. Indeed, when compared to their matched controls, these 19 cases were administered approximately three times the IV fluid volumes (Table 2). Importantly, none of these cases was caused by equipment malfunction or administration at a rate faster than ordered. The ADHF patients considered to have/not have inappropriate fluid balance did not differ significantly ($p > 0.05$) in any of the clinical variables examined (i.e. all the variables listed in Table 4).

Table 1 Precipitants of ADHF among the cases (n = 80).

Precipitant	n (%)
Infection	24 (30)
Inappropriate fluid management	19 (23.8)
Tachyarrhythmia	10 (12.5)
Ischaemic heart disease	7 (8.8)
Inappropriate medication management	7 (8.8)
Unclear	5 (6.3)
Renal failure	1 (1.3)
other[a]	7 (8.8)

[a]other: pancreatitis, liver failure, heart block, non-compliance with fluid restriction, multiple possible precipitants.

Seven cases (three medical, four surgical patients) had their ADHF precipitated by inappropriate medication management (Table 3). In all cases, the patients' usual medications (usually diuretics) were appropriately withheld on admission due to hypovolaemia or hypotension. However, after resolution of their presenting condition, these medications were not restarted or were restarted too late.

The univariate and multivariate analyses of demographic and clinical variables potentially associated with ADHF are reported in Table 4. In the univariate analyses, most variables had relatively small effect sizes (OR < 2.5, Table 4). The main exceptions were the IV fluid volumes administered on Days 1 and 2 before, and on the day of, the ADHF. The multivariate regression indicated that three variables were significantly and independently associated with ADHF: not having English as the preferred language, a history of ischaemic heart disease and the administration of >2000 ml of IV fluid on the Day 1 before the ADHF. Some other variables had considerable effect sizes (digoxin, high potassium, very low albumin) but had small patient numbers and were not statistically significant.

Table 2 Intravenous fluid volume administered to cases perceived to have had inappropriate IV fluid management.

Days preceding ADHF	cases (n = 19)	controls (n = 19)	p
Day 2 before ADHF ml, median (IQR)	2,000 (2128)	751 (2400)	0.03
Day 1 before ADHF ml, median (IQR)	2,757.5 (2338)	975 (1126)	0.001
Day of ADHF ml, median (IQR)	1,165 (1056)	480 (1113)	0.04

Abbreviations: ADHF, acute decompensated heart failure.

Table 3 Details of the seven cases attributed to inappropriate medication management.

Clinical condition	Comorbidities	Medication change	ADHF cause
Small bowel obstruction	IHD, HTN	frusemide* ceased	not restarted
PR bleeding, abdominal pain	HTN, VD	frusemide* ceased	not restarted
Instability post-banding oesophageal varices	HTN, DM	spironolactone ceased on admission	not restarted
Bradycardia, lethargy, dizziness	IHD, PVD, HTN	metoprolol ceased	not restarted
Hypotension on admission	IHD, HTN	frusemide* and spironolactone ceased	not restarted
Hypotension on admission	IHD, HTN	spironolactone ceased	too slow to restart
Acute renal injury, diarrhoea, hypotension on admission	IHD, HTN, HF, VD	frusemide* and spironolactone ceased	too slow to restart

Abreviations: ADHF, acute decompensated heart failure, IHD, ischaemic heart disease, HTN, hypertension, VD, valvular disease, DM, diabetes mellitus, AF, atrial fibrillation, HF, heart failure.
*furosemide.

Table 4 Univariate and multivariate analyses of demographic and clinical variables potentially associated with acute decompensated heart failure (ADHF).

variable	variable category	ADHF status		univariate analyses			multivariate analyses		
		control (n)	case (n)	OR	(95% CI)	p	OR	(95% CI)	p
English preferred language	yes	71	61	1			1		
	no	9	19	2.5	(1.0-5.5)	0.04	3.5	(1.2-9.8)	0.02
Smoker	no	57	52	1					
	yes	23	28	1.3	(0.7-2.6)	0.40	–	–	–
Ischaemic heart disease	no	64	48	1			1		
	yes	16	32	2.7	(1.3-5.4)	0.01	3.3	(1.2-9.1)	0.02
Peripheral vascular disease	no	70	75	1					
	yes	10	5	0.5	(0.2-1.4)	0.18	–	–	–
Hypertension	no	25	19	1					
	yes	55	61	1.5	(0.7-2.9)	0.29	–	–	–
Heart failure	no	69	59	1			1		
	yes	11	21	2.2	(1.0-5.0)	0.051	1.7	(0.6-5.0)	0.30
Valve dysfunction	no	74	67	1			1		
	yes	6	13	2.4	(0.9-6.7)	0.09	1.3	(0.3-4.7)	0.73
Diabetes	no	56	55	1					
	yes	24	25	1.1	(0.5-2.1)	0.86	–	–	–
Beta blocker	no	55	46	1					
	yes	25	34	1.6	(0.9-3.1)	0.14	–	–	–
Calcium channel blocker	no	61	64	1					
	yes	19	16	0.8	(0.4-1.7)	0.57	–	–	–
ACEI/ARB	no	39	44	1					
	yes	41	36	0.8	(0.4-1.5)	0.43	–	–	–
Diuretics	no	48	44	1					
	yes	32	36	1.2	(0.7-2.3)	0.52	–	–	–
Spironolactone	no	76	69	1			1		
	yes	4	11	3.0	(0.9-10.0)	0.07	2.0	(0.5-8.5)	0.34
Digoxin	no	72	78	1			1		
	yes	8	2	0.2	(0.1-1.1)	0.07	0.2	(0.0-1.2)	0.07

Table 4. (continued).

variable	variable category	ADHF status		univariate analyses			multivariate analyses		
		control (n)	case (n)	OR	(95% CI)	p	OR	(95% CI)	p
Nitrates	no	74	66	1			1		
	yes	6	14	2.6	(1.0-7.2)	0.06	1.6	(0.4-6.8)	0.50
Haemoglobin[a], g/L	very low	13	18	1.8	(0.8-4.4)	0.17	–	–	–
	low	30	34	1.5	(0.8-3.0)	0.25	–	–	–
	normal	37	28	1					
Sodium, mmol/L	<130	2	5	2.8	(0.5-14.8)	0.24	–	–	–
	130-135	15	18	1.3	(0.6-2.9)	0.47	–	–	–
	≥136[b]	63	57	1					
Potassium, mmol/L	<3.5	4	4	1.2	(0.3-4.9)	0.83	0.5	(0.1-3.2)	0.44
	3.5-5.1[b]	75	64	1			1		
	>5.1	1	12	14.1	(1.8-111.1)	0.01	8.8	(0.9-88.0)	0.06
Albumin, g/L	<26	8	19	3.4	(1.3-8.9)	0.01	3.0	(1.0-9.6)	0.06
	26-34	35	35	1.4	(0.7-2.8)	0.31	1.0	(0.4-2.5)	0.95
	35-52[b]	37	26	1			1		
Urea[c], mmol/L	normal	40	34	1			1		
	high	29	20	0.8	(0.4-1.7)	0.58	0.9	(0.4-2.4)	0.88
	very high	11	26	2.8	(1.2-6.5)	0.02	1.4	(0.5-4.2)	0.58
Creatinine[d], μmol/L	normal	41	35	1					
	high	24	23	1.1	(0.5-2.3)	0.76	–	–	–
	very high	15	22	1.7	(0.8-3.8)	0.18	–	–	–
IV fluid received on Day 2 before ADHF (ml)	<500	64	53	1			1		
	500-1000	7	6	1.0	(0.3-3.3)	0.95	0.8	(0.2-3.6)	0.72
	1001-2000	3	10	4.0	(1.1-15.4)	0.04	3.9	(0.7-22.1)	0.13
	>2000	6	11	2.2	(0.8-6.4)	0.14	1.1	(0.2-5.0)	0.93
IV fluid received on Day 1 before ADHF (ml)	<500	54	35	1			1		
	500-1000	12	11	1.4	(0.6-3.6)	0.46	1.7	(0.5-5.7)	0.37
	1001-2000	11	16	2.2	(0.3-5.4)	0.07	1.8	(0.5-7.1)	0.38
	>2000	3	18	9.3	(2.5-33.8)	0.001	8.3	(1.5-48.0)	0.02
IV fluid received on day of ADHF (ml)	<500	62	47	1			1		
	500-1000	11	16	1.9	(0.8-4.5)	0.14	1.4	(0.5-4.6)	0.54
	1001-2000	7	17	3.2	(1.2-8.4)	0.02	2.3	(0.6-9.5)	0.26
	>2000	0	0	–	–	–	–	–	–

Abbreviation: ACEI/ARB, angiotensin converting enzyme inhibitor/angiotensin receptor blocker.
[a]Haemoglobin (g/L0: Males – very low <100, low 100-129, normal 130-180. Females – very low <90, low 90-114, normal 115-165.
[b]normal range.
[c]Urea (mmol/L): Males – normal 3.0-9.2, high 9.3-14, very high >14. Females – normal 3.5-7.2, high 7.3-12, very high >12.
[d]Creatinine (μmol/L): Males – normal 62-106, high 107-140, very high >140. Females – normal 44-80, high 81-130, very high >130.

The drug treatment of the cases' ADHF included diuretics (frusemide [furosemide] 78 cases, ethacrynic acid 1 case), morphine (1 case) and nitroglycerine (11 cases). Twelve cases required respiratory support including continuous positive airway pressure (cPAP) (six cases) and endotracheal intubation (six cases).

The outcomes of the case and control groups differed significantly (Table 5). The cases had a length of stay more than twice that of the controls and a much greater mortality. Slightly more than one half of cases were discharged to home, significantly fewer than the control patients. The unadjusted Cox regression analysis for cumulative hazard of death indicated that the groups

Table 5 Outcomes subsequent to ADHF.

Outcome	Cases (n = 80)	Controls (n = 80)	p
Length of stay, days, median (interquartile range (IQR))	15 (18)	6 (7)	<0.001
Died in hospital, n (%)	10 (12.5)	1 (1.3)	0.01
Sub-acute care post admission, n (%)	20 (25)	11 (13.8)	0.07
Discharged to home, n (%)	46 (57.5)	65 (81.3)	<0.01

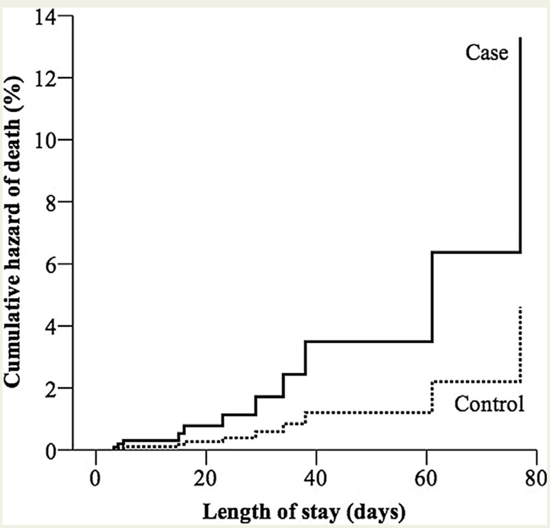

Figure 1 Unadjusted Cox regression analysis curve for the cumulative hazard of death during the entire hospital inpatient stay (patient follow-up period).

did not differ (Figure 1). The Hazard Ratio for death in cases compared to controls was 2.9 (95% CIs 0.3–25.1), p = 0.34.

Discussion

This case-control study identified an ADHF incidence of 1%. Although this incidence represents a small proportion of patients, it would amount to a considerable number over time. This is particularly relevant given that one third of cases were identified as having an iatrogenic precipitant. The ADHF precipitants are very similar to an earlier report of inpatients, with infection being the most common [13]. Pulmonary infections are particularly prone to precipitate ADHF in the community setting [14].

Taylor et al. [13] reported inappropriate IV fluid management as the second most common precipitant of ADHF. Their proportion of cases attributed to IV fluid (25.5%) was almost identical to that of this study (23.8%). These findings are concerning, particularly given the significantly increased mortality from ADHF among inpatients [8,12,13,15].

A range of complications associated with IV fluid administration have been reported [10,12,15]. The important association between IV fluid and ADHF was highlighted by Bikdeli et al. [10]. They reported that, among patients with a primary discharge diagnosis of heart failure, those who received at least 500 ml of IV fluids experienced significantly higher rates of critical care admission, intubation, renal replacement therapy and death [10]. In a clinical update of IV fluid problems, Hilton et al. [11] reported that inappropriate management is a significant cause of patient morbidity and mortality and that either too much, too little, or the incorrect type of fluid may be the cause. Patients with pre-existing cardiorespiratory disease and those with sepsis and other severe illness are particularly at risk [11,16].

Medications and medication mismanagement, especially medication reduction, have been reported as important iatrogenic precipitants [8,12,14]. In this study, almost all cases were due to the cessation of the patient's usual diuretic. It is not possible to determine, retrospectively, exactly how appropriate these cessations were. However, it is more likely that the failure to restart the diuretics was the most likely cause.

Tachyarrhythmia and myocardial ischaemia were less often the precipitant compared to the report by Taylor et al. [13]. The small proportion of cases precipitated by myocardial ischaemia was unexpected as substantially more cases had a history of ischaemic heart disease and this was a variable significantly associated with ADHF. Although speculation, ischaemia may have contributed to the ADHF in combination with other variables or may have been unrecognised by the treating staff.

The regression results are important. The association between ADHF and a preferred language other than English was not expected and is difficult to explain. Although speculation, it may reflect either underlying communication difficulties that impact adversely upon optimal patient care or a differing health status among this patient subgroup.

The association between ADHF and ischaemic heart disease is not surprising and consistent with other reports [8,13,14,17]. In this condition, the myocardium has less reserve which often becomes manifest when challenged by intercurrent illness, surgery or increases in cardiac pre-load. Excessive IV fluid volumes may increase pre-load to a level that cannot be accommodated by the heart, with the precipitation of ADHF. While increased pre-load may have contributed to the association between ADHF and IV fluid administration, the precipitant in most patients is likely to be multi-factorial. The large IV fluid volumes administered likely reflect the presence of medical conditions that may also predispose the patient to ADHF e.g. infection, myocardial ischaemia with cardiovascular instability. Although significantly associated with AHDF, the finding that digoxin tended to be a protective factor against ADHF is consistent with its inotropic effect.

Renal failure caused only a single case of ADHF in this study. Poor patient outcomes are more likely among those with higher urea and creatinine levels [15,18]. It is notable, therefore, that neither of these variables was associated with ADHF. Whilst hyperkalaemia had a large effect size (OR 8.8), the lack of an association with ADHF is likely attributable to the small number of patients with this condition.

The mortality among our cases was substantial, significantly higher than the controls and consistent with other reports [13]. Both Taylor et al. [13] and Patel et al. [8] have reported that mortality associated with ADHF that develops in hospital is almost three times that when it develops out of hospital. The finding that our cases had a significantly longer median length of stay and smaller proportion than those who were discharged to home is consistent with one other report [8]. While these associations are important, the contributions of the ADHF event itself and a range of other variables cannot be clearly determined. Indeed, the cases were a sicker group: more had ischaemic heart disease, elevated potassium and urea, very low albumin levels and greater IV fluid volumes. It is likely, however, that the ADHF event did contribute substantially to the poorer outcomes and these have been reported previously among similar patients [13].

A number of recommendations in regard to the iatrogenic precipitations can be made. Firstly, meticulous IV fluid management is required including frequent and ongoing review of the patient's fluid balance and the indications for fluid, its nature and administration rate. Diligent documentation of fluid balance is also recommended including daily weighing of patients at increased risk. It has been recommended that, in the uncomplicated perioperative patient, a restrictive IV fluid replacement regimen should be used [11]. Fixed IV fluid regimens (e.g. 3 L/day) are used frequently in this setting [19]. However, these should be considered as guides only and adjusted to clinical response and serial heart rate, blood pressure and urine output measurements [11]. Secondly, greater vigilance is required when a patient's usual medication regimen is adjusted to account for intercurrent illness. Mechanisms to remind clinicians that re-adjustment of the medications may be needed upon resolution of the illness. These mechanisms may involve the ward pharmacy service or automatic flagging embedded within the electronic medical record.

This study has important limitations. Although a range of techniques was employed to identify all potential cases, some may have been missed. Also, the strict definition of ADHF may have precluded the enrolment of mild cases resulting in an under-estimation of the ADHF incidence and selection bias. Despite extensive matching of cases with controls, the cases and controls differed in a number of baseline variables. The retrospective identification of some cases and the controls may have been affected by the accuracy of the electronic medical record. Also, details of important potential confounding variables may not have been collected and accounted for in the regression analyses.

As the treating staff were aware of the study, the Hawthorne Effect [20] (a change in behaviour as a result of being observed) may have been introduced with staff being more attentive to fluid balance and medication management. Although some cases were identified several days after the ADHF event, recall bias among the treating staff was not apparent. Some treating staff could not be contacted and the precipitant had to be determined retrospectively by the panel of investigators. Only a single precipitant was assigned to each case although the cause may have been multi-factorial. As the precipitant could not be identified in a small proportion of cases, the nature of all precipitants is not fully known. A prospective study carefully examining a range of potential precipitants is recommended. It would also allow detailed information on the cause of death to be ascertained.

The study only examined IV fluid volumes and was not designed to examine fluid management in detail e.g. indications, nature of the fluids, rates and quality of patient monitoring. Before the ADHF event, patient weight was measured in only 27 cases and was rarely measured on a daily basis. Also, fluid balance charts tended to be largely incomplete and could not be used with any reliability. Finally, as a single centre study, the generalisability of the findings is questionable.

Conclusions

The incidence of ADHF developing among hospital inpatients is considerable and associated with significantly increased length of hospital stay, morbidity and mortality. Acute decompensated heart failure is precipitated by a range of conditions or events with infection the most common cause. Importantly, approximately one third of cases are iatrogenic and precipitated by inappropriate IV fluid and medication management. A preferred language other than English and a history of ischaemic heart disease were associated with the onset of ADHF. Mechanisms to better monitor IV fluid administration and flags to prompt the recommencement of suspended usual medications are recommended.

Author Contribution

Study design and protocol development were done by LP, DT, DJ and LB. Ethics approval was obtained LP and DT. Patients were identified by LP, TW and AP. Data collection was done by LP, assisted TW and AP. Patient reviews were performed by LP, DT, DJ and LB. Data interpretation and analysis were undertaken by LP, DT, SP and AU. All authors contributed to the manuscript preparation and revision and have approved the final version.

Conflicts of Interest

None.

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