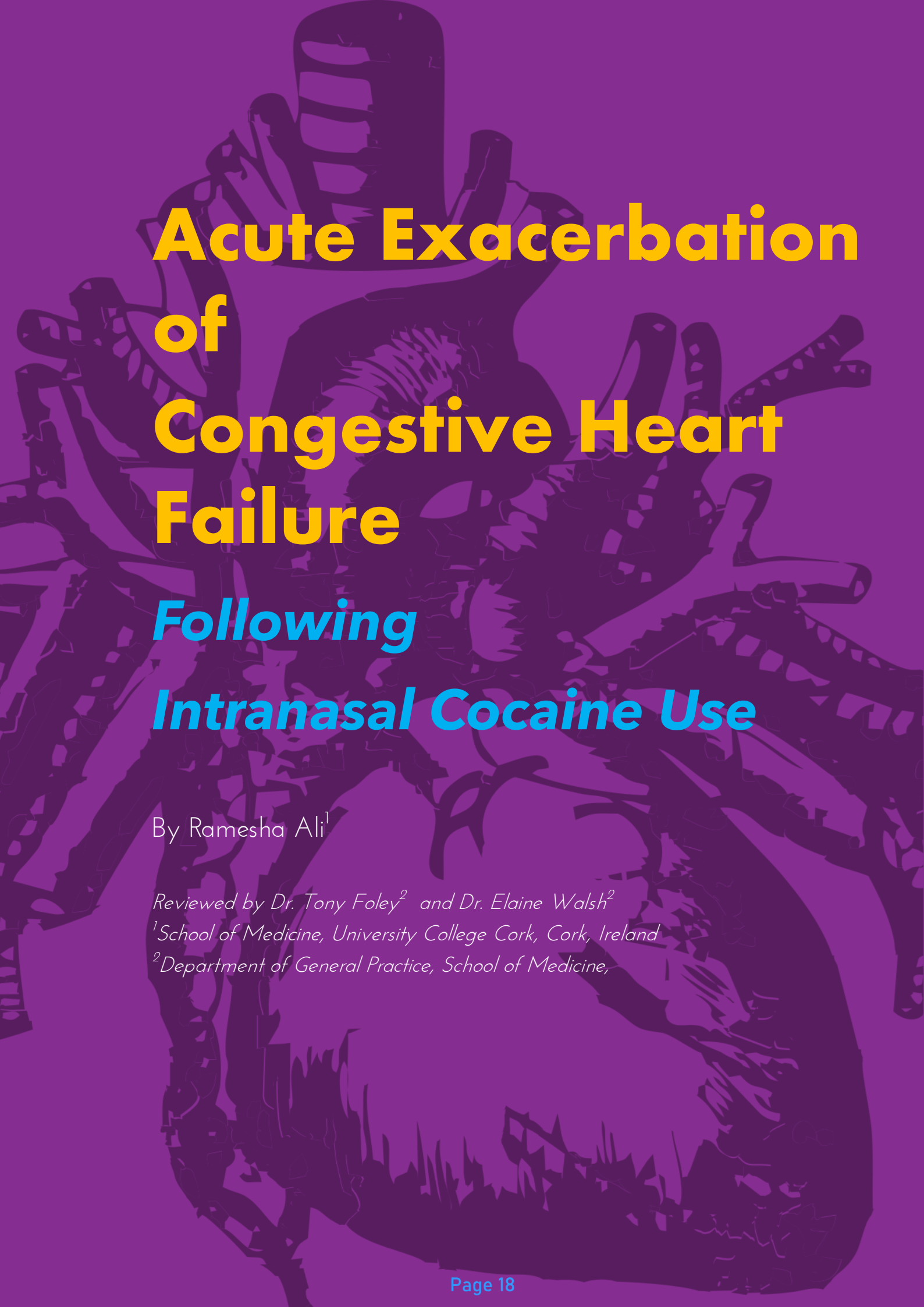

Acute Exacerbation of Congestive Heart Failure

Following Intranasal Cocaine Use

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Abstract

Background

Most presentations of decompensated congestive heart failure occur in patients diagnosed with pre-existing heart failure. Decompensation presents as progressive dyspnea, abdominal and peripheral congestion, as well as neurologic symptoms. It typically occurs following physiologic stressors such as infection, arrythmia, or medical non-adherence. Chronic heart failure may result from cardiovascular comorbid conditions, such as coronary artery disease, valvular disease, and long-standing hypertension.

Long term cocaine use also results in adverse cardiovascular health. Cocaine use can augment pre-existing risk factors for both chronic congestive heart failure and acute decompensations – namely, coronary artery disease, congestive heart failure, and peripheral vascular disease. It can also independently pose a cardiovascular risk by causing acute ischemia, vasoconstriction, tachycardia, systolic dysfunction, and cardiac remodelling.

The Case

The case of a 49-year-old Caucasian male who presented with worsening dyspnea on exertion and bilateral peripheral edema extending to his abdomen. His symptoms worsened over the preceding week and began after three consecutive days of intranasal cocaine use. He presented with a background history of congestive heart failure, coronary artery disease, peripheral vascular disease, a 30 pack-year smoking history, and weekly cocaine use for the past 12 years.

Conclusion

Cocaine use can lead to decompensation of congestive heart failure in patients with extensive cardiac and vascular disease. Cocaine use can also acutely worsen systolic function and cause demand ischemia, on a background of chronic remodelling and atherosclerotic changes.

Patient Consent Obtained

Yes.

Case Background

Decompensated heart failure may be the initial presentation of congestive heart failure (CHF) or a sign of worsening pre-existing and diagnosed heart failure. Development and severity of symptoms depend on the extent of underlying cardiac disease, as well as factors that may lead to acute worsening of symptoms. Nearly 70% of patients are admitted following an acute exacerbation and present with an acute-on-chronic heart failure episode[1]. Chronic heart failure may result from coronary artery disease, valvular disease, and poorly controlled hypertension. In the event of decompensated heart failure, precipitating factors such as infection, arrythmia, medical non-adherence, and uncontrolled hypertension, may be present [1]. Decompensation presents as progressive dyspnoea, abdominal and peripheral congestion, as well as neurologic symptoms.

Cocaine use is implicated in both chronic heart failure and decompensation with acute worsening of symptoms. Although it is not extensively identified as an acute precipitant of heart failure, it can worsen hypertension and lead to tachycardia, increasing myocardial oxygen demand while reducing oxygen supply through vasoconstriction [2]. It can also increase ventricular chamber size and cause systolic dysfunction, aiding in the pathogenesis of chronic cardiomyopathy. Cocaine use can induce fatal outcomes such as arrythmias and myocardial infarction [2]. By aiding in long-term cardiac remodelling and augmenting the risk factors for decompensation, it directly and indirectly worsens the prognoses of CHF and may contribute to an exacerbation. Additionally, cocaine can independently increase the risk of heart failure and systolic dysfunction, resulting in a poor cardiovascular health profile [2]. Previous literature has explored how long term cocaine use can lead to heart failure over time, however, an immediate and temporal association between cocaine use and decompensation has not been well documented.

Both cardiovascular mortality and all-cause mortality continue to be higher in cocaine users when compared to non-cocaine users. Cocaine users have two times the risk of sudden cardiac death compared to non-cocaine users alone [2]. The burden of cocaine on the healthcare system expands beyond its implications on heart disease, to further include the effects of chronic malnutrition often seen in cocaine users, due to its disruption of metabolic and neuroendocrine regulation [2].

This case report presents a 49-year-old Caucasian male who attended the emergency department with signs of decompensated CHF following intranasal cocaine use.

Case Details

A 49-year-old Caucasian male presented to the emergency department with worsening dyspnoea on exertion, lower extremity swelling extending to the abdomen, and lower extremity discomfort that started one week ago, following three consecutive days of intranasal cocaine use, approximately 80mg in total. The dyspnoea made him unable to carry out his activities of daily living and was relieved only by rest. Over the last week, his lower extremity swelling and abdominal distension gradually worsened, and he gained approximately three kilograms over the ten days preceding his admission.

He also experienced acute-on-chronic bilateral leg discomfort with erythema, eschar formation, and venous ulcers above the medial malleoli bilaterally. The lower extremity erythema and eschar formation, over both tibias, had been present for the past year. However, over the past week, he experienced increased skin breakdown with superficial wounds, areas of slough and serosanguinous discharge from both lower legs. He described his leg discomfort as diffuse, 10/10 in severity, rendering him unable to ambulate.

Systemically, he experienced generalized fatigue but no fever, chills, or sweats. He denied orthopnea, paroxysmal nocturnal dyspnoea, cough, recent illness, or any insult to his lower extremities.

He presented on a background history of congestive heart failure, coronary artery disease, peripheral vascular disease, 30 pack-year smoking history, and weekly cocaine use, approximately 15mg each week for the past 12 years. His past medical history was significant for dyslipidemia and three ST-elevation myocardial infarctions (STEMI MI), all requiring percutaneous coronary intervention (PCI). His medications included aspirin, metoprolol, ramipril, and spironolactone, to which he was adherent. Family history was significant for his father who passed away from a MI. Social history was significant for social housing in a low- income area associated with significant cocaine use among the residents, as well as the previously noted substance abuse. The patient also consumed approximately 10-12 units of alcohol per week, predominantly on the weekends.

On examination, the patient was unable to comfortably position his legs, which were elevated and exposed. His jugular venous pressure was elevated to 2cm above the angle of the jaw. Auscultation of the lungs revealed bilateral crackles in the lower lobes. The abdomen was distended but non-tender. The liver and spleen edge could not be ascertained due to the ascites and distension. Shifting dullness and fluid thrill were present. On peripheral examination, the patient had significant bilateral erythema and eschar formation, particularly inferior to the knee, and the right lower extremity was worse than the left. Extensive skin breakdown resulted in wounds that were draining serosanguinous fluid. There were ulcers above both medial malleoli, approximately 7cm in diameter each, with exudate. The temperature was warm and equal in both lower extremities, with tenderness to palpation bilaterally. Pitting oedema extended to the abdomen and was characterized as 4+. The dorsalis pedis and posterior tibialis pulses could not be palpated bilaterally. The patient's vital signs were within normal ranges. At this time, differential diagnoses included both decompensated liver failure as well as decompensated congestive heart failure.

Investigations

Laboratory investigation revealed hyponatremia (119 mmol/L), elevated troponin (0.039 mg/L), elevated gamma glutamyl transferase (GGT) (383 U/L) and an elevated C-reactive protein (8.4 mg/L). The patient also had lactic acidosis with lactate levels elevated at 2.6 mmol/L. The patient's GGT level was similar to that reported during his previous admission in 2017. However, remaining laboratory derangements were new or worsened.

An echocardiogram revealed a left ventricular (LV) ejection fraction of 18% with moderate right ventricular (RV) hypertrophy and moderate tricuspid regurgitation and dysfunction. A CT angiogram of the abdomen, pelvis, and lower extremities revealed significant burden of atherosclerotic disease, and a doppler ultrasound also revealed worsened peripheral vascular disease compared to a study performed in 2017. A chest x-ray revealed borderline enlargement of the pericardial silhouette with mild pulmonary oedema in the lower lobes and notable atelectasis. A wound culture confirmed colonization of venous ulcers with Pseudomonas aeruginosa. An ultrasound of the abdomen revelated an enlarged liver by 6-7cm and gallbladder wall thickening, secondary to ascites.

Following these investigations, the diagnoses was confirmed to be decompensated congestive heart failure, New York Heart Association (NYHA) Class III.

Management:

The patient was commenced on IV furosemide and Milrinone until he reached a euvolemic state, at which point he was transitioned to oral furosemide only. He was commenced on a low salt diet and fluid restriction with daily assessment of weight. A therapeutic ultrasound guided paracentesis was performed for ascites, and approximately four liters of fluid were removed. Troponin was slightly elevated secondary to demand ischaemia, as no ischemic changes were seen on ECG and necessitated no further workup. Wound care involved treatment with

both ceftazidime and vancomycin as well as adequate dressing and coverage.

Discussion

Heart failure continues to be a significant contributor to poor quality of life, healthcare burden, and admission times [1] Although many individuals do not experience an acute exacerbation following intranasal cocaine use, it can augment the risk posed by other risk factors and aid in an exacerbation. Persistent use of cocaine increases the risk of vascular disease as well as promotes cardiac remodelling, resulting in cardiomyopathy. Unfortunately, this patient's non-modifiable risk factors, namely age, sex, previous MI, and familial history place him at a higher risk of developing CHF, coronary artery disease (CAD), peripheral vascular disease (PVD), and associated complications [3]. In addition, modifiable risk factors such as smoking can independently double the risk of mortality and dyslipidema [3,4]. These risk factors can accelerate and increase the toxicity associated with cocaine use and cause increased deterioration of cardiovascular health.

This case demonstrates how a preceding poor cardiac function profile can accelerate progression to an acute CHF exacerbation. Specifically, an LV ejection fraction of 18% with moderate RV hypertrophy, and moderate tricuspid regurgitation and dysfunction, as seen in this patient, pose independent risks for CHF exacerbation [3]. Additionally, the use of cocaine can lead to the development of dilated cardiomyopathy due to deprived myocardial oxygen supply, ventricular hypertrophy, as well as decreased left ventricular contractility through blockage of sodium transport and norepinephrine uptake in the myocardium [3]. It can also promote platelet adherence and thrombosis, augmenting atherosclerotic disease burden. Therefore, cocaine use can be a toxic risk precipitant of both CHF exacerbations and vascular disease. Cocaine use is associated with complications such as MI and arrythmias and increases the risk of CHF by 5-7% [2,5]. Cocaine also promotes peripheral atherosclerosis, skin ischaemia , and deep vein thrombosis, which can further contribute to worsening ulcers, as seen in this patient [5]. Whereas much of the existing literature focuses on the long term effects of cocaine use, this case provides a unique example of an acute and immediate adverse effect of cocaine on a previously poor cardiovascular profile, as evidenced by the sudden decompensation experienced by this patient.

It is important to assess such risk factors to better manage long-term prognosis and mortality rate. Substance use alone poses significant risks and highlights the need for education as well as access to interventions and support. It is also important to understand how poor peripheral health may represent the cycle of ill health patients often experience, as oedema and skin breakdown can make it difficult to ambulate, which further increase the risk of developing venous ulcers, as in this patient. Attention to the psychological and social context to which this patient presents can aid in education and encouragement of cessation of substance abuse. Addressing many of this patient's comorbid conditions that are risk factors for decompensated CHF will help improve his wellbeing and prevent future exacerbations [6]. Practical recommendations may include coordination with social workers to help alleviate the social limitations experienced by this patient, such as late presentation due to accessibility concerns. Furthermore, motivational interviewing at bedside as well as frequent follow-up and support in both an inpatient and outpatient setting, respectively, can support this patient in cocaine cessation.

This patient had several comorbid conditions on presentation, and his progression throughout his admission also raised the concern of discontinuity of care – which independently increases the risk of prolonged and emergency admissions [7,8]. A CHF exacerbation may require care in both the cardiac care unit (CCU) and intensive care unit (ICU), as medications such as Milrinone require ICU supervision in some hos-

pitals. Patient transfers between the CCU to the ICU may lead to care provision by different physicians, as well as a change in allied health professional administered care, such as physiotherapists, occupational therapists, and social workers. Although these transitions are inevitable and performed in the best interest of the patient, each instance of discontinuity is associated with lower clinical condition scores and increased time to recovery, as assessed by the Rothman Index [7].

Conclusions

This case discusses a 49-year-old male who presented with decompensated CHF following intranasal cocaine, as well as PVD stigmata with superimposed bacterial infections. He had several modifiable and non-modifiable risk factors associated with worse cardiovascular health. Although a prior direct causal link has not been established between cocaine and acute CHF exacerbations, cocaine use can augment the deleterious effects of these risk factors in an acute setting. This case contributes to our understanding of how cocaine use can acutely lead to decompensated CHF on a background of poor cardiovascular health, secondary to chronic cocaine use.

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