ATYPICAL CHEST PAIN IN THE CONTEXT OF SUBSTANCE USE AND CHRONIC PAIN

Omee Upadhyaya, DO[1], Adam Voelckers, MD[1]
1. Family Medicine, University of Pittsburgh Medical Center, Lititz, PA

UPMC
LIFE CHANGING MEDICINE

Introduction

Cardiac symptomatology in the context of acute intoxication with cocaine necessitates consideration of substance-induced vasoconstriction and prothrombotic effects, leading to manifestations such as chest pain.

The vasoconstrictive property of cocaine has been well-established via mechanisms such as arterial alpha-adrenergic receptor stimulation, endothelin-1 amplification, and nitric oxide decrease [1]. Similarly, cocaine predisposes one to coronary arterial thrombus development, both acutely by direct effects on platelets and increases in von Willebrand factor & plasminogen-activator inhibitor, as well as chronically by progression of atherosclerosis [1-3, 5, 6].

While myocardial ischemia/infarction must in part constitute the differential diagnosis in a patient with such a history or presentation, the inclusion of other cardiopulmonary etiologies and broad factors that may complicate perception and management of pain is paramount. The clinical utility of this holistic care approach can be noteworthy among patients with preexisting complex, or otherwise substantial, pain management regimens, given the multifactorial neuropsychologic experience of chronic pain (Figure 1).

Neuroadaptations within persons afflicted with addiction may align with those present in persons who suffer from chronic pain, with respect to hedonic homeostatic dysregulation (Figure 1). From the level of more fundamental nociceptive processing to the elaborate premeditative aspects of prefrontal cortical activity, pain-perceptive neurocircuitry is connective. Although the consideration of such mechanisms and their nuanced variability in certain patient populations may appear to deviate from the traditional pace and content of chest pain evaluations, comorbidities do not reside within contexts so distinct as to preclude their interplay. Such is the value of multidisciplinary, multiple-context health care.

Chronic Pain Syndrome Progression

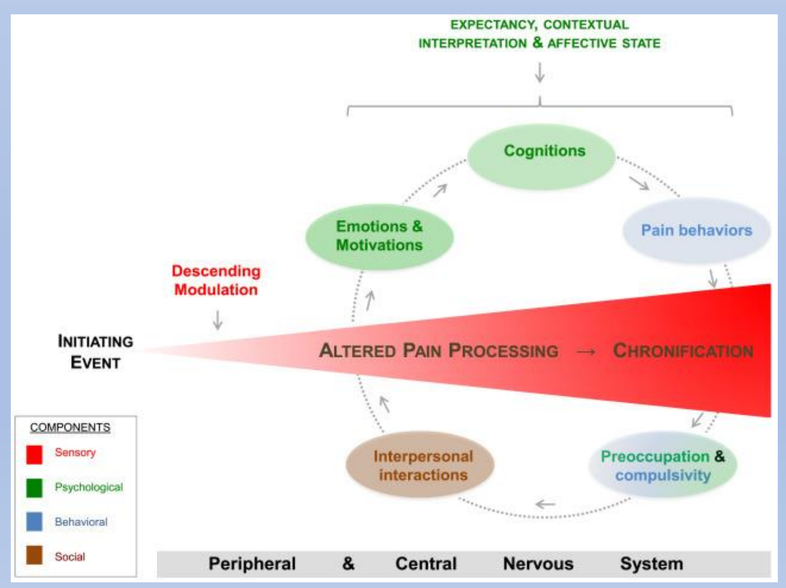

Figure 1. Nociceptive detection of injury to nerves/tissues, leading to reward process disruption and compromising sensory, psychological, behavioral, and social aspects. [4]

Atypical Chest Pain

Table 1. Symptoms of Angina.*

Classic (Typical)	Atypical, Noncardiac
Sensations in chest of squeezing, heaviness, pressure, weight, vise-like aching, burning, tightness	Pain that is pleuritic, sharp, pricking, knife-like, pulsating, lancinating, choking
Radiation to shoulder, neck, jaw, inner arm, epigastrium (can occur without chest component); band-like discomfort	Involves chest wall; is positional, tender to palpation; can be inframammary; radiation patterns highly variable
Relatively predictable	Random onset
Lasts 3–15 min	Lasts seconds, minutes, hours, or all day
Abates when stressor is gone or nitroglycerin is taken	Variable response to nitroglycerin

* Data are from Sangareddi et al.[7]

Figure 2. Symptomatic differentiation of chest pain typicality [7].

Case Presentation

In this piece, we discuss a 52-year-old male patient with a past medical history notable for cocaine use, schizophrenia, bipolar disorder, anxiety, and baseline back pain. In clinic, this patient reported on presentation three weeks of intermittent, episodic, sharp, substernal chest/left axillary pain, worse upon exertion, that had caused left hemiparesis/flaccidity and hours of feeling drained. Associated symptoms included irregular heartbeat, palpitations, dyspnea, dizziness, and painful active extension of the upper extremities after an episode. Several episodes occurred while exiting a car. He endorsed last cocaine use one year prior, heroin overdose (with cardiopulmonary arrest) four months ago, and a positive familial cardiac history.

POC UDS was positive for buprenorphine and marijuana, with the former finding consistent with ongoing medication-assisted treatment (MAT). POC EKG revealed first-degree block, with possible age-undetermined anterior infarct (Figure 3).

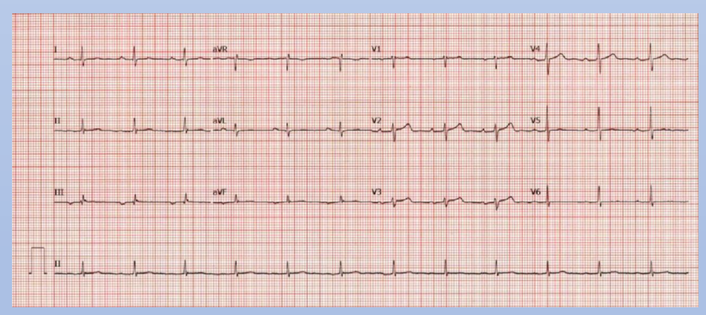

Figure 3. POC EKG with sinus rhythm with first-degree AV block, with possible age-undetermined anterior infarct.

The patient was advised to visit the ED for further cardiac evaluation. In the ED, EKG showed NSR without indication of acute ischemia, troponins satisfied rule-out criteria, and CXR was negative for apparent acute process. With overall condition improved as well, the patient was diagnosed with unspecified chest pain and discharged with reassurance and follow-up instructions.

He remained stable after these encounters, with persistent chest symptoms and subsequently pursued cardiac workup alongside recommendation of an over-the-counter pain management regimen.

Discussion

The atypical symptomatology of this patient within the context of his history warranted a holistic consideration of musculoskeletal and psychiatric etiologies, such as thoracic outlet syndrome and somatic symptom disorder, respectively, alongside apparent cardiac conditions (Figure 2). Recommendations for immediate cardiac evaluation were necessary, given the nature of the history provided and point-of-care considerations; however, his comorbid longstanding pain and substance use histories, respectively, indicated comprehensive patient care, per the primary osteopathic tenet. This tenet maintains the constitution of person by body, mind, and spirit, an especially critical approach when presentation and concise point-of-care workup deviate from pathologic typicality in those with altered pain perception – a likely reasonable characterization of this patient. Consideration of potential confounders in reward processing is significant, as is the acknowledgement of biopsychosocial model-aligned patient efforts amid such variables. This clinic patient was committed to his Suboxone treatment, counseling, psychopharmacologic regimen, and self-advocacy.

As in the case of this patient with such an acutely concerning history, however, the resource allocation necessary at times for diagnostic rapidity can impede such comprehensive considerations, underscoring the value of continuity-based primary and specialty care. Despite an ultimately negative cardiopulmonary workup in the ED, completed at one point in time per his acute indications, his case exemplifies the indubitable need for expansive diagnostics and care for those with perceptual specifications, as influenced by behavioral and physical comorbidities.

Conclusions

1. Application of the primary osteopathic tenet, with a consideration of both acute and chronic health afflictions, is vital to workup and patient care provision.
2. Evaluation of chest pain, while often appropriately expedited, should, in nonacute care contexts, encompass etiologies including, but not limited to, substance use, musculoskeletal distress, chronic pain-related reward processing variations, personality, and cardiopulmonary concerns.

References

1. Bachi, K., Mani, V., Jeyachandran, D., Fayad, Z. A., Goldstein, R. Z., & Alia-Klein, N. (2017). Vascular disease in cocaine addiction. *Atherosclerosis*, *262*, 154–162. https://doi.org/10.1016/j.atherosclerosis.2017.03.019
2. Bozkurt, B., Colvin, M., Cook, J., Cooper, L. T., Deswal, A., Fonarow, G. C., Francis, G. S., Lenihan, D., Lewis, E. F., McNamara, D. M., Pahl, E., Vasan, R. S., Ramasubbu, K., Rasmusson, K., Towbin, J. A., Yancy, C., & American Heart Association Committee on Heart Failure and Transplantation of the Council on Clinical Cardiology; Council on Cardiovascular Disease in the Young; Council on Cardiovascular and Stroke Nursing; Council on Epidemiology and Prevention; and Council on Quality of Care and Outcomes Research (2016). Current Diagnostic and Treatment Strategies for Specific Dilated Cardiomyopathies: A Scientific Statement From the American Heart Association. *Circulation*, *134*(23), e579–e646. https://doi.org/10.1161/CIR.0000000000000455
3. Hobbs, W. E., Moore, E. E., Penkala, R. A., Bolgiano, D. D., & López, J. A. (2013). Cocaine and specific cocaine metabolites induce von Willebrand factor release from endothelial cells in a tissue-specific manner. *Arteriosclerosis, thrombosis, and vascular biology*, *33*(6), 1230–1237. https://doi.org/10.1161/ATVBAHA.113.301436
4. Igor Elman, David Borsook, Common Brain Mechanisms of Chronic Pain and Addiction, Neuron, Volume 89, Issue 1, 2016, Pages 11-36, ISSN 0896-6273, https://doi.org/10.1016/j.neuron.2015.11.027.(https://www.sciencedirect.com/science/article/pii/S0896627315010338)
5. McCord, J., Jneid, H., Hollander, J. E., de Lemos, J. A., Cercek, B., Hsue, P., Gibler, W. B., Ohman, E. M., Drew, B., Philippides, G., Newby, L. K., & American Heart Association Acute Cardiac Care Committee of the Council on Clinical Cardiology (2008). Management of cocaine-associated chest pain and myocardial infarction: a scientific statement from the American Heart Association Acute Cardiac Care Committee of the Council on Clinical Cardiology. *Circulation*, *117*(14), 1897–1907. https://doi.org/10.1161/CIRCULATIONAHA.107.188950
6. Sáez, C. G., Pereira-Flores, K., Ebensperger, R., Panes, O., Massardo, T., Hidalgo, P., Mezzano, D., & Pereira, J. (2014). Atorvastatin reduces the proadhesive and prothrombotic endothelial cell phenotype induced by cocaine and plasma from cocaine consumers in vitro. *Arteriosclerosis, thrombosis, and vascular biology*, *34*(11), 2439–2448. https://doi.org/10.1161/ATVBAHA.114.304535
7. *What do we mean by atypical chest pain ?*. Dr.S.Venkatesan MD. (2009, July 4). https://drsvenkatesan.com/2008/09/14/what-do-we-mean-by-atypical-chest-pain/