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Subarachnoid Hemorrhage Presenting with Second-Degree Type I Sinoatrial Exit Block: A Case Report

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Abstract

The understanding of neural regulation of the cardiovascular function and the implications of a “Heart-Brain Axis” has been a topic of interest for clinicians for many years. Electrocardiographic (ECG) and structural cardiac changes, ranging from mild, asymptomatic, transient alteration in cardiovascular function to severe, irreversible, and potentially life-threatening injury, can actually be a manifestation of several neurological disorders. When managing cardiac disorders, a high index of clinical suspicion, detailed history-taking and physical examination skills, and an extensive workup that covers both cardiac and non-cardiac causes should be utilized. It is important to consider that cardiovascular dysfunction of an underlying neurological etiology may lead to difficulty in diagnosing and optimizing treatment of the latter. We report the case of a middle-aged female with the chief complaint of syncope preceded by a headache with no focal neurological deficits, originally diagnosed with- and whose syncope was attributed to sinus bradycardia and type I sinoatrial (SA) exit block on ECG. Subsequently, when the patient became altered, however, computer tomography (CT) angiography revealed subarachnoid hemorrhage (SAH) with middle cerebral artery aneurysm. This presentation emphasizes the importance of tabulating neurological injury as one of the differential diagnoses while managing ECG changes in cardiovascular disease (CVD), as missing and delaying the former can result in disastrous consequences.

Keywords

subarachnoid hemorrhage (SAH); electrocardiography (ECG); type 1 sinoatrial (SA) exit block; syncope; neurological injury; heart brain axis

1. Introduction

Syncope, defined as an abrupt and transient loss of consciousness due to cerebral hypoperfusion, accounts for 1–1.5% of emergency department (ED) visits [1]. It is categorized into neurally-mediated, cardiac, and orthostatic syncope according to the underlying cause. Neurally-mediated syncope is the most common cause of syncope in the general population and has a benign course, while cardiac syncope tends to correlate with an increased mortality and morbidity [1]. One trigger that can lead to electrocardiography (ECG) changes in patients is aneurysmal subarachnoid hemorrhage (SAH), a type of hemorrhagic stroke affecting approximately 30,000 persons per year in the United States [2,3]. ECG changes are the most studied and widely recognized abnormalities following SAH, seen in 25–90% of SAH patients [4,5,6,7,8]. In addition, rhythm and conduction abnormalities either can occur alone or in combination with other ECG changes. Several prospective and retrospective clinical studies on SAH have reported arrhythmias such as sinus bradycardia (15%), sinus tachycardia (13%), premature ventricular beats (13%), atrial fibrillation and ventricular tachycardia (2%), AV block (1.5%), and asystole (1%) [9,10,11,12,13,14]. Neurogenic heart disease is defined as the brain's effects on the heart, with underlying mechanisms including excessive catecholamine activity, acute stressors, autonomic disturbances, and reperfusion injury [15]. To the best of our knowledge, to date, there is no published literature revealing second degree type I sinoatrial (SA) exit block as a manifestation of aneurysmal SAH. Higher risk of rebleeding and related mortality is associated with a delay in the diagnosis and treatment of SAH [16]. Therefore, to avoid any catastrophic consequences, neurological injury must be considered a part of the list of potential etiological conditions causing cardiac conduction disorders.

2. Case Report

A 63-year-old African American female with a past medical history of hypertension, asthma and hyperlipidemia presented to the emergency department with a chief complaint of syncope preceded by headache. In the ED, the patient was noted to have regained consciousness shortly after her syncopal episode, but she complained of persistent headache and dizziness. On physical examination, no focal neurologic deficits were found. Her vitals at that time showed a blood pressure of 141/82 mmHg, heart rate of 48 beats per minute, temperature of 34.5°C (94.1°F), and respiratory rate of 19 cycles per minute. Her ECG showed sinus rhythm and pauses with junctional escape beats and atrial premature contractions with a ventricular rate of 46 beats per minute (Figure 1 and Figure 2). Laboratory investigations were within normal limits (Table 1). Cardiology was consulted for management of sinus bradycardia, sinoatrial block, and junctional escape. A transvenous pacemaker was placed with good capture (Figure 3).

The patient was initially admitted to the critical care unit for management of the bradyarrhythmia. Soon after admission to the critical care unit, she became altered, agitated, and unable to follow commands. Neurology recommended a head CT to rule out intracranial pathology. The imaging study revealed a diffuse SAH with hydrocephalus. An emergent extra-ventricular drain was placed by Neurosurgery. The bradyarrhythmia and the altered mental status then resolved. A follow up CT angiography of the brain showed a right middle cerebral artery (MCA) aneurysm (Figure 4). She was transferred to the surgical intensive care unit for further management of her SAH and right MCA aneurysm. The patient was started on intravenous tranexamic acid, levetiracetam, and nimodipine. She underwent cerebral angiography with Neuro-interventional Radiology. Cerebral angiography showed a ruptured posterior communicating artery aneurysm (Figure 5, left image) which was subsequently embolized with coil placement (Figure 5, middle image), resulting in near-complete aneurysm occlusion and preservation of the adjacent arteries (Figure 5, right image). The patient was transferred back to the surgical intensive care unit neurologically intact with mild functional limitations.

3. Discussion

Cardiac manifestations of SAH [17] are a well-known entity, ranging from mild ECG variability, reversible left ventricular dysfunction (Takotsubo), non-ST-elevation myocardial infarction (NSTEMI), ST-elevation myocardial infarction (STEMI), to cardiac arrest [18,19,20,21,22,23]. As per a systematic review analysis, the crude global incidence of SAH declined from 10.2 per 100000 person-years in 1980 to 6.1 in 2010, with large variation according to region, age, and sex [24]. In the United States, the annual incidence of aneurysmal SAH is 6–16 cases per 100,000 population, with approximately 30,000 episodes occurring each year [25]. Out of all strokes, 20% are hemorrhagic, with SAH and intracerebral hemorrhage each accounting for 10% [26]. 75–85% of non-traumatic SAH is caused by ruptured cerebral aneurysms [27]. The major cardiac manifestations resulting from SAH are arrhythmias [13] (35%), myocardial injury seen as troponin elevations in the first 24 hours [28] (28%), wall motion abnormalities [29] (28%), and classic deep septal T-wave inversion or QTc prolongation on ECG [30,31]. Most of the rhythm alterations are benign, including sinus bradycardia or tachycardia; atrial fibrillation, ventricular ectopics, and junctional rhythm have also been described in SAH patients [31]. To the best of our knowledge, there has been no prior report describing SAH as the underlying cause of a second-degree type 1 SA nodal exit block; such a scenario is being reported for the first time in this case report. The possible risk factors for arrhythmia occurrence include female gender, QTc prolongation, excessive sympathetic discharge, coronary vasospasm, electrolyte disturbances, and pre-existing hypertension [32]. Life-threatening arrhythmias occur in about 5% of cases [13]. Extensive myocardial injury following SAH can result in severely depressed global cardiac function, as well as reduction particularly in the left ventricular function. This condition is known as neurogenic stunned myocardium (NMS), recently restyled as stress-related cardiomyopathy syndrome [33].

New progress in neuroanatomic studies, pathological assessments, and imaging modalities have helped illuminate the neural regulation of cardiovascular functions [34], a design that involves a complex network of cortical and subcortical brain regions [35] along with the

intrinsic cardiac system. Figure 6 elucidates the role of the central nervous system (CNS) in regulating the cardiovascular system (CVS) functions.

The orbitofrontal cortex and dorsal cingulate cortex process afferent information and control efferent autonomic outflow to the CVS. The insular cortex is the main center regulating the 'Heart-Brain Axis' [36]. Some studies hypothesize that the right insula controls sympathetic actions, and when damaged, can effect ECG changes and lead to increased mortality [37,38]. Others postulate the same for the left insula [39]. The amygdala receives inhibitory inputs from the prefrontal and orbitofrontal cortices and processes them with the hypothalamus and the brain stem nuclei [40,41]. In this way, it modulates the effect of emotional stimuli on the heart [42]. The nucleus tractus solitarius (NTS), located in the posterior medulla, receives hemodynamic inputs and sends efferents to the rostral ventrolateral medulla (RVLM) and dorsal motor nucleus (DMN) of the vagus, which control the sympathetic and parasympathetic outflow, respectively, to the heart [34]. The CNS regulates the CV function through cardiomotor sympathetic and parasympathetic outflow of the autonomic nervous system (ANS) to the myocardium and cardiac conduction system [43]. In addition, there is an intrinsic cardiac nervous system, composed of interconnected ganglionated plexi in epicardial fat tissue, innervating the SA node, AV node, and pulmonary veins [44]. Oxidative stress, nitric oxide levels, and abnormal baroreceptor and chemoreceptor signals are conveyed to NTS, paraventricular nucleus, and RVLM through cardiac afferent fibers, which results in fatal arrhythmias and ischemia [45,46].

The neuronal control of the CVS undergoes major alterations in different circumstances and may contribute to progression of the underlying heart disease. Immediately after SAH, there is intense activity in the hypothalamus, insula, and brain stem, causing significant activation of sympathetic nerve endings, with release of norepinephrine [32]. In patients with SAH, repolarization abnormalities, caused by an increase in sympathetic activity in SAH, are the most common ECG finding [47]. The SA node acts as the normal pacemaker of the heart, generating action potentials that travel through the conduction system to cause myocardial contraction [48]. SA nodal exit block is a condition in which the impulse fired by the SA node is unable to reach the neighboring atrial tissue [49]. It is divided into three degrees of block, similar to AV blocks. On ECG, SA nodal exit block can be inferred from the P wave activity, but only a second degree SA block can be detected in the ED using a 12-lead ECG [50]. Second degree SA nodal exit block is of two types: type I Wenckebach and type II. Type I has a P-P interval that progressively shortens in duration until there is a dropped P wave [49].

At times, it can be difficult to establish neurological injury as the cause or the consequence of cardiovascular dysfunction due to the high prevalence of coexistent cardiac disorders and shared risk factors in these patients. Electrocardiographic alterations and conduction defects, both benign and fatal, are commonly seen after brain injury even without a preexisting heart disease. However, when the cardiovascular presentation of the symptoms are not correlated with their neurological cause, it can result in delayed or missed diagnosis of neurological diseases.

4. Conclusion

Between 50 to 100% of patients experience cardiac rhythm disturbances during the acute phase of SAH, but only 1–4% of patients experience a clinically significant arrhythmia. SAH is a serious condition that frequently leads to neurological impairment and death; therefore, its timely diagnosis and management is of utmost significance. The current article is an effort at consolidating the information available in an attempt to avoid errors in the diagnosis of neurological disease presenting in an atypical manner. Recently, the ‘Heart-Brain Axis’ concept, which acknowledges a complex network of neural regulation of cardiovascular functions, has gained much popularity. More collaborative studies and research are likely to further advance our understanding of the neural control of the cardiac tissue.

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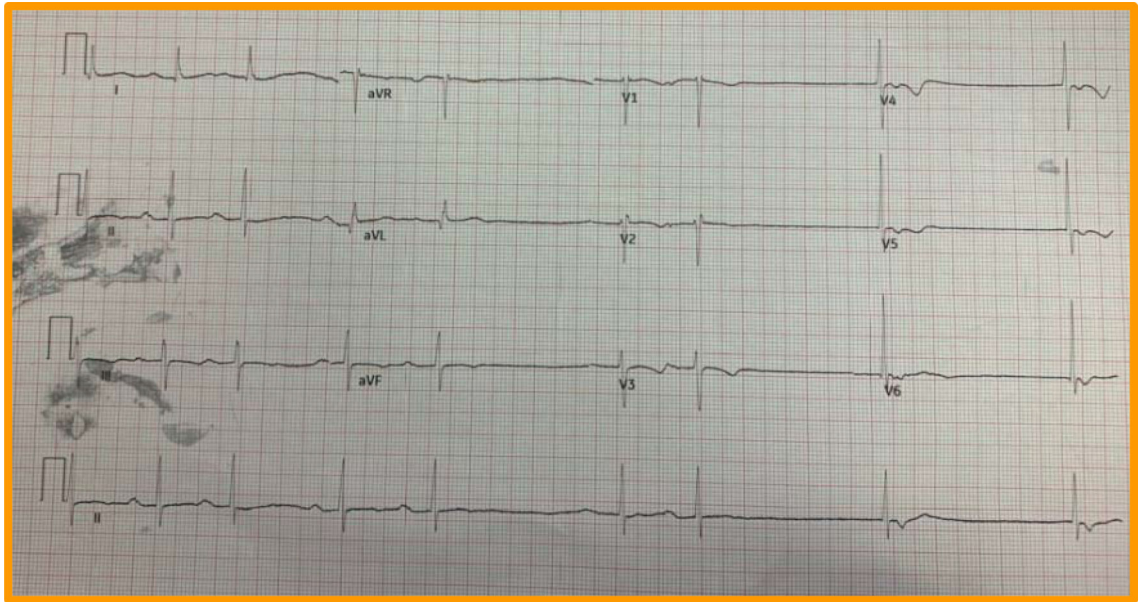


Figure 1.
Electrocardiogram showing Mobitz type I sinus atrial node exit block with progression to junctional rhythm

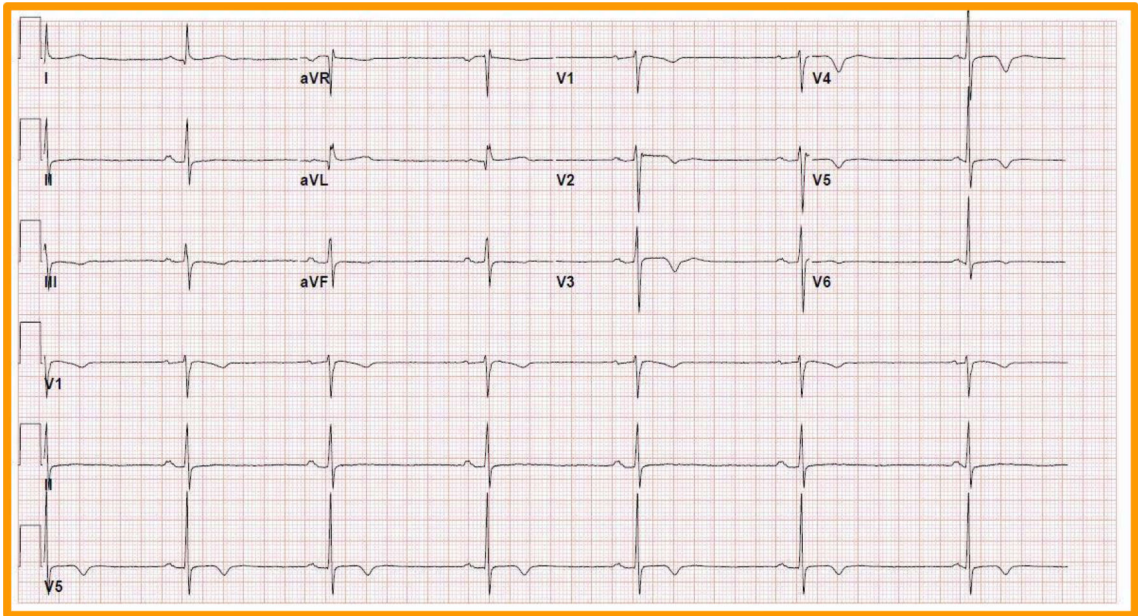


Figure 2.
EKG showing sinus bradycardia

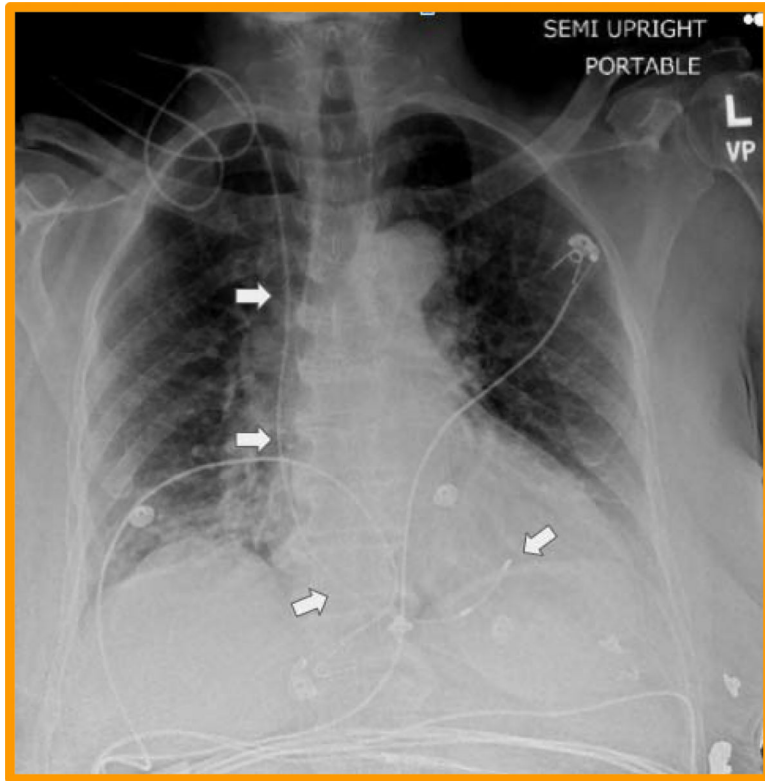


Figure 3. Portable chest X-ray showing transvenous pacer (indicated by arrows)



Figure 4.
Computed tomography scan of the head showing right middle cerebral artery aneurysm



Figure 5. Cerebral angiogram showing an aneurysm in the right posterior communicating artery (left), during endovascular coil placement (middle), and complete regression of aneurysm following endovascular coil placement (right)

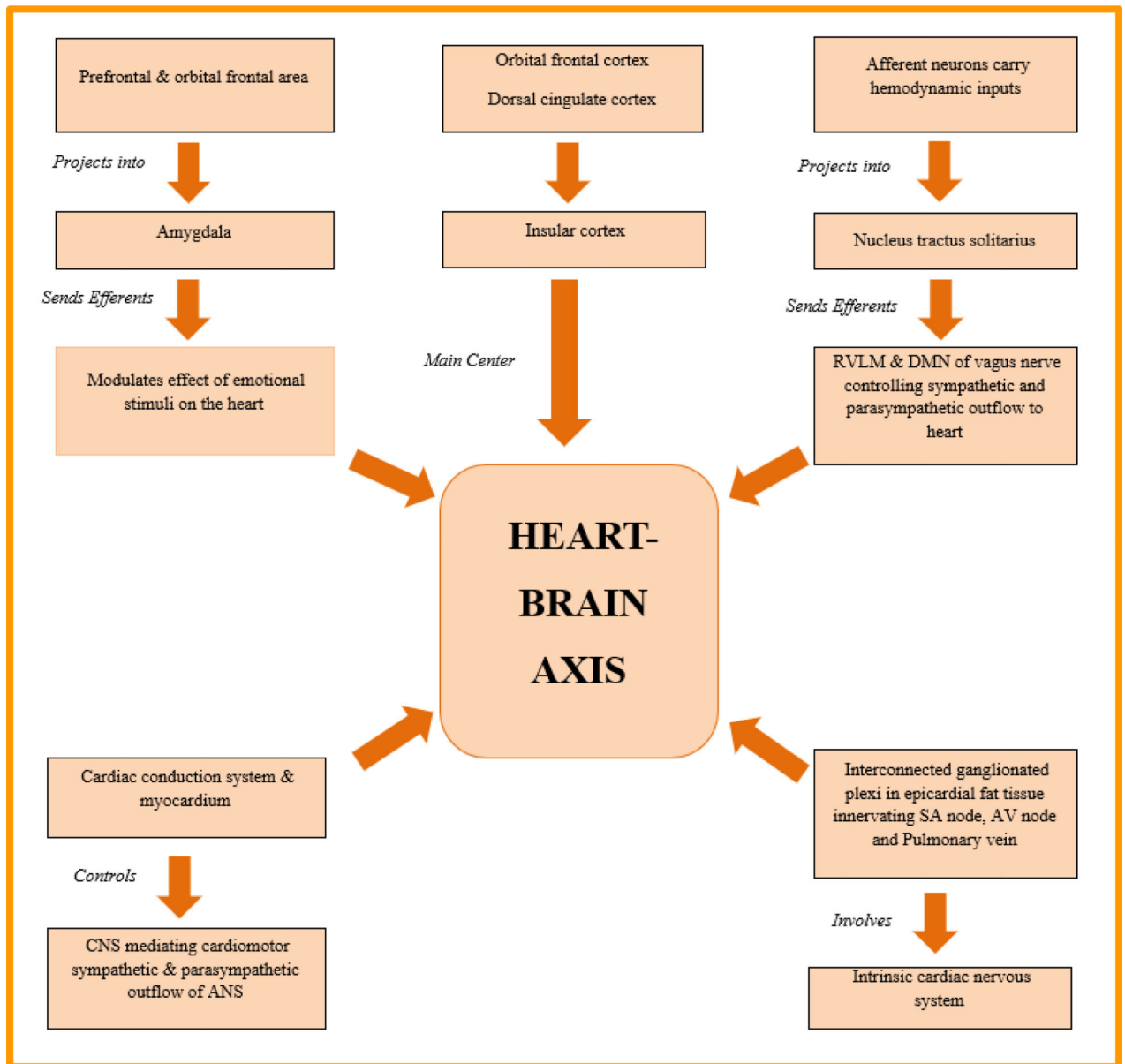


Figure 6. This flow diagram outlines the Heart-Brain Axis (Legend: **RVLN**- rostral ventrolateral medulla, **DMN**- dorsal motor nucleus, **ANS**-autonomic nervous system, **CNS**- central nervous system, **SA**- sinus atrial, **AV**- atrioventricular)

Table 1.

Complete Blood Count

Lab Data	Reference Range	On Admission
COMPLETE BLOOD COUNT		
White blood cell count (10 ³ /uL)	4.10 – 10.10	12
Neutrophils (%)	44.5 – 73.4	70.9
Lymphocytes (%)	17.8 – 42.0	19.8
Monocytes (%)	5.7 – 11.2	8.8
Eosinophils (%)	0.2 – 6.0	0.1
Basophils (%)	0.3 – 1.1	0.4
Neutrophils absolute (10 ³ /uL)	1.40 – 6.80	8.47
Lymphocytes absolute (10 ³ /uL)	1.10 – 2.90	2.37
Monocytes absolute (10 ³ /uL)	0.20 – 1.00	1.05
Eosinophils absolute (10 ³ /uL)	0.00 – 0.40	0
Basophils absolute (10 ³ /uL)	0.00 – 0.10	0
Red blood cells (10 ⁶ /uL)	4.33 – 5.43	4.74
Hemoglobin (g/dL)	12.9 – 16.7	13.6
Hematocrit (%)	40.0 – 47.0	40.8
Mean corpuscular volume (fL)	80.8 – 94.1	85.9
Mean corpuscular hemoglobin (pg)	27.1 – 31.2	28.7
Mean corpuscular hemoglobin conc (g/dl)	31.0 – 34.4	33.4
Red cell distribution width (%)	12.3 – 14.6	13.8
Mean platelet volume (fL)	7.9 – 11.0	8.7
Platelets (10 ³ /uL)	153 – 328	223
Troponin I (ng/mL)	0.012 – 0.034	0.4
P-Natriuretic Peptide (pg/mL)	11.1 – 125.0	80.7
Ferritin (ng/mL)	17.90 – 464.00	95.4
CHEMISTRY		
Glucose (mg/dL)	70 – 99	140
Blood urea nitrogen (mg/dL)	9.0 – 20.0	10
Creatine (mg/dL)	0.66 – 1.25	0.90
Sodium (mEq/L)	133 – 145	136
Potassium (mEq/L)	3.5 – 5.1	4.5
Chloride (mEq/L)	98 – 107	103
Calcium (mg/dL)	8.4 – 10.5	9.7
Anion gap (mEq/L)		13
Total Protein (g/dL)	6.3 – 8.2	8.2
Albumin (g/dL)	3.5 – 5.0	4.1
Total Bilirubin (mg/dL)	0.2 – 1.3	0.7
Aspartate transaminase (U/L)	21 – 72	37

Lab Data	Reference Range	On Admission
Alanine aminotransferase (U/L)	17 – 59	40
Alkaline Phosphatase (U/L)	38.0 – 126.0	74
Serum Bicarbonate, Co2 (mEq/L)	22 – 30	23
D-dimer (ng/mL)	0 – 230	50
Troponin I	0.00–0.2	0.01

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