Ongoing caffeine anaphylaxis: a differential for mental illness

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Abstract

Caffeine, a monoamine oxidase inhibitor (MAOI), sensitizes, causes masked allergic response, anticholinergic effects and other biochemical abnormalities. When the disorder is wrongly diagnosed and an allergic patient continues ingesting caffeine, the patient suffers ongoing caffeine anaphylaxis fight or flight toxicity, chronic allergic response. Ongoing caffeine anaphylaxis alters homeostasis by increasing neurotransmitter and hormone output, causes cerebral vasculitis, breaks down cell walls, generates cerebral toxicity, toxic dementia, rhabdomyolysis, hyperglycemia, ataxia, adrenal exhaustion and other physical disorders. This article discusses the majority of biochemical abnormalities accompanying ongoing caffeine anaphylaxis and relating to mental illness. The author suggests that physicians look for signs of caffeine anaphylaxis before diagnosing and stigmatizing patients with symptoms of mental illness with a mental disorder. Ongoing caffeine anaphylaxis is a physical condition confused with and diagnosed as mental illness.

Keywords: caffeine, allergic response, anaphylaxis, biochemical abnormalities, mental illness

1. Introduction

Controversy surrounds the benefits and dangers of caffeine. Yet, according to chemical manufacturers, caffeine is harmful when swallowed and if inhaled [1,2].

Controversy does not surround drug allergies. Every drug, including caffeine, can sensitize and cause an allergic reaction.

An allergic reaction to caffeine presents as the "fight or flight response" and anaphylaxis [3-6]. Chronic allergic response, *ongoing caffeine anaphylaxis fight or flight toxicity*, causes cerebral vasculitis, breaks down the blood-brain barrier, and generates toxic dementia, resulting in symptoms of all mental disorders, including ADHD, bipolar disorder and schizophrenia [3,5,6].

An allergic caffeine consumer ingesting caffeine on a chronic basis and the doctor treating the patient remain unaware of the individual's caffeine allergy. Caffeine strips insight [3, 5-10], and masks allergic response by generating adrenaline and cortisol release [3,5-7]. And caffeine converts to theophylline, maintaining open airways [3,5-7,11]. Doctor and patient believe the allergic patient suffers from a treatable but incurable mental disorder. A wrong diagnosis contributes to continued caffeine intake, resulting in brain inflammation and progressive toxicity.

Psychiatric patients chronically consume absurd amounts of caffeine [12-14], putting them at risk for developing an allergy to caffeine and theophylline. According to one study, the daily caffeine intake was >750 mg in 13% of 98 psychiatric patients. The average intake was 405 mg/day before hospitalization. During hospitalization, the average intake was 332 mg/day [12].

The abnormal chemical imbalances of ongoing caffeine anaphylaxis match the imbalances of ADHD and the mental disorders [5,6]. Unable to pinpoint the cause of mental illness, doctors disregard indicators of allergic toxicity, and refer patients for psychiatric counseling.

Abnormal ATP, cyclic AMP, catecholamine, serotonin, CPK MM, y-Aminobutyric acid (GABA), oxygen, lactic acid, aldosterone, antidiuretic hormone (ADH), cortisol and prostaglandin levels, along with cholinergic insufficiency, indicative of chronic caffeine anaphylaxis, are commonly erroneously used as correlates of mental illness. We examine these biochemical changes and more.

2. Adenosine triphosphate and cyclic AMP

Adenosine triphosphate (ATP), the fuel of all cells, made primarily in the mitochondria, is a three-phosphate adenosine nucleotide. ATP must be continuously replenished. Providing long-term energy, *oxidative phosphorylation* occurs in the mitochondria and makes most of our ATP. During dephosphorylation, free energy is released when ATP drops a phosphate molecule, thus converting to adenosine diphosphate (ADP) and further to AMP. Supplying energy for metabolic reactions, ATP is a participant in almost every biochemical reaction, probably all. It is a key player in the cyclic AMP cycle.

When a first messenger binds to a receptor and activates a G-protein, the G-protein stimulates the enzyme adenylate cyclase, converting ATP to cyclic AMP. Cyclic AMP triggers a specific kinase or enzyme, generating a biochemical response.

3. Caffeine anaphylaxis, catecholamine release, ATP and cyclic AMP

Caffeine, a monoamine oxidase inhibitor (MAOI) [15], stimulates catecholamine release, delays catecholamine breakdown, and disrupts normal cell signaling [4-6]. Caffeine inhibits *phosphodiesterase* breakdown of cyclic AMP [16-18]. Phosphodiesterase inhibition increases cyclic AMP [17], and enables ongoing cyclic AMP-mediated reactions [6].

During anaphylaxis, initially cyclic AMP rises, followed by a sharp fall [19]. However, during ongoing caffeine anaphylaxis, continued caffeine intake and allergic response mistaken for mania causes a catecholamine and cyclic AMP excess and detrimental homeostatic changes. With caffeine cessation or withdrawal, a depressed recovery state mistaken for depression [3,6,7], elevated catecholamine and cyclic AMP levels decrease [6].

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Caffeine anaphylaxis causes brain edema [3-7,11]. Cyclic AMP, involved with cell wall breakdown, contributes to brain edema [20] and is involved with cerebral injury. On an experiment with dogs, cerebral spinal fluid (CSF) cyclic AMP rose drastically forty-eight hours after acute cerebral trauma, thereafter declining to sub-maximal levels [21].

Along with endogenous adrenaline, endogenous cortisol and theophylline, cyclic AMP is helpful in quelling anaphylaxis. Cyclic AMP inhibits histamine release, masking allergic response [3,5-7,22,23].

Despite caffeine anaphylaxis' ability to minimize allergic response, it further upsets homeostasis. With toxicity progressing, caffeine anaphylaxis is capable of decreasing cyclic AMP. Ongoing caffeine anaphylaxis results in a constant energy demand, diminishing ATP and poisoning muscles. Allergic toxicity manifests with rhabdomyolysis [6,24], injury to skeletal muscle presenting with leakage of internal chemicals, which may include CPK, magnesium, calcium and other substances, and an ATP reduction. Drugs generating a constant energy demand are capable of reducing ATP, inducing rhabdomyolysis [25]. A substance coating cell membranes can reduce cell function by decreasing ATP [26]. Caffeine toxicity can decrease ATP and cause rhabdomyolysis [6,27,28]. And anaphylaxis utilizes ATP [6,29,30]. Progressive ATP deficiency can lead to a progressive cyclic AMP reduction and other biochemical abnormalities.

4. ATP, cyclic AMP and mental illness

ATP is a factor of mental illness [31-35]. Researchers suggest that ATP is decreased in the frontal lobe of schizophrenic patients [31]. Another report indicates that the ATP level is low with schizophrenia [32]. According to a study, ATP rose with increased activity and dropped with decreased activity in bipolar patients [33].

The cyclic AMP level is also altered with psychosis [36-42]. Plasma cyclic AMP has been marked increased with childhood-onset psychosis [36], and the CSF cyclic AMP level is elevated in schizophrenic patients [37-39]. Cyclic AMP has been low with the depressed state of bipolar and elevated with mania [40-42].

These changes are indicative of caffeine anaphylaxis. During allergic crisis, cyclic AMP rises. During recovery periods, or periods of expended energy, cyclic AMP can decrease [6].

5. Caffeine anaphylaxis and creatine phosphokinase

Under normal conditions, skeletal muscles contain a large amount of ATP and creatine. The enzyme creatine phosphokinase of skeletal type (CPK MM, CPK) catalyzes the reversible reaction: creatine phosphate + ADP ↔ creatine + ATP. An ATP lag results in excess CPK [43].

By attempting to correct for altered homeostasis, a stimulated system utilizes ATP faster than nonstimulated organs. Drugs that disrupt ATP production or increase energy requirements can raise the CPK level [25,26].

Ongoing caffeine anaphylaxis elevates CPK [6, 24]. Caffeine can elevate CPK [27,28], and CPK can rise with allergic response [44, 45], and sepsis [46,47].

6. CPK and mental illness

Studies confirm that persons diagnosed with mental disorders, including patients diagnosed with personality disorder, mania, bipolar disorder, depression, schizophrenia and catatonia, exhibit an elevated CPK level [48-56]. In fact, CPK is erroneously being used as a biological correlate of bipolar disorder and schizophrenia.

7. Caffeine anaphylaxis and Na+/K+-ATPase

The forcing of ions or small molecules through cell membranes against their concentration gradient involves normal activity of the sodium-potassium pump (Na^+/K^+-ATPase). Located in every cell, the Na^+/K^+-ATPase is regulated by calcium and ATP.

Caffeine anaphylaxis disrupts Na^+/K^+-ATPase [6]. An ATP deficit leads to decreased Na^+/K^+-ATPase function. Along with increased cyclic AMP and cell mediator secretion, reduced Na^+/K^+-ATPase contributes to cell wall breakdown, leading to edema [6].

8. Na^+/K^+-ATPase, edema and mental illness

Alterations in Na^+/K^+-ATPase function affect depressed, manic, bipolar and schizophrenic patients [57-60]. Edema is also common with psychiatric illness [61,62]. Breakdown of the blood-brain barrier is known to accompany schizophrenia [63, 64]. Out of 15 patients newly diagnosed with schizophrenia, eight showed signs of blood-brain barrier permeability [63].

9. Caffeine anaphylaxis and serotonin

Serotonin, 5-HT, is a neurotransmitter and mast cell mediator released during anaphylaxis. Serotonin depends on monoamine oxidase (MAO) for breakdown.

Caffeine anaphylaxis increases 5-HT [3,5,6]. Caffeine inhibits MAO, platelets are 5-HT carriers and increase with damage to vascular beds, and serotonin is commonly released during allergic response [6]. Excess serotonin contributes to cell wall breakdown.

10. Serotonin and mental illness

It is proposed that serotonergic dysfunction may be common with all mood disorders [65], and that a psychoactive substance may be acting on 5-HT receptors [66]. According to studies, 5-HT metabolism is decreased with depression, bipolar disorder, schizophrenia, and suiciders [65,67-69].

Considering caffeine's psychoactive effects, the amounts of caffeine ingested by psychiatric patients, and that psychiatric patients are often stressed and malnourished, doctors should consider caffeine as the chemical altering serotonergic activity.

11. Caffeine anaphylaxis causes anticholinergic effects

During the fight or flight response, the sympathetic nervous system accelerates. *Homeostasis* returns after danger has

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passed. However, for a caffeine allergic person, chronic caffeine ingestion results in ongoing fight or flight with continuous sympathetic and parasympathetic acceleration [6,11].

Caffeine anaphylaxis stimulates acetylcholine release [6,11]. During allergic response, the parasympathetic nervous system releases acetylcholine in an attempt to relax muscles and correct for the over stimulated system. An experiment with rats proved caffeine's dose-dependent ability to antagonize adenosine (A1) receptors and stimulate ACh release in the hippocampus [70]. Anaphylaxis also stimulates the cholinergic system [6, 19].

ATP is required for muscle contraction. An ATP reduction contributes to anticholinergic symptoms, which include a clouding of consciousness, paranoia, memory deficits, muscle rigidity, including the inability of ocular muscles to efficiently track, temporomandibular joint dysfunction (TMJ), paralysis, ataxia, ataxic gait and other mental and physical conditions that doctors mistake for mental and physical disorders [6].

Pesticides work by inhibiting *acetylcholinesterase* (AChE), causing anticholinergic effects. Caffeine is a pesticide [71]. And adrenochrome, an adrenaline breakdown product found in schizophrenic patients, inhibits AChE [15]. When it over stimulates and causes the adrenalin-adrenochrome conversion, caffeine is capable of inhibiting AChE [6].

12. Anticholinergic effects and mental illness

Psychotic patients suffer from ataxia, TMJ, the inability of ocular muscles to properly track and other anticholinergic symptoms [6]. But before doctors diagnose a mental disorder they should consider ongoing caffeine anaphylaxis' anticholinergic effects. Mania and the psychoses present with altered cholinergic function [72-75]. The general belief seems to be that a cholinergic lag is a factor of psychosis [6].

13. Caffeine anaphylaxis and y-Aminobutyric acid

y-Aminobutyric acid (GABA), the primary inhibition neurotransmitter, acts as a natural inhibitor. GABA helps prevent mania.

Caffeine anaphylaxis antagonizes GABA function. Caffeine intolerance decreases GABA activity in several areas of the brain, including the cerebral cortex, hypothalamus and pons-medulla [76]. Experiment results on rats suggest caffeine (10-40 mg/k) increases motor activity by decreasing GABA [77]. And adrenochrome decreases GABA [15].

In the early stage of ongoing caffeine anaphylaxis, GABA probably decreases allergic symptoms [6], because experiment results suggest that GABA inhibits anaphylactic histamine release [78,79]. Thus, depending on the degree of allergic toxicity, GABA may seem to protect caffeine allergic persons from increased symptoms of allergic toxicity, including children and teenagers suffering symptoms of ADHD [6]. Yet, as chronic allergic response progresses, GABA diminishes and endogenous toxicity progresses. Increased brain poisoning is mistaken for anxiety, hysteria, panic disorder, obsessive-compulsive disorder (OCD), mania, bipolar psychosis and schizophrenia [6].

14. GABA and mental illness

According to studies, GABA deficiency plays a part in anxiety, panic disorder, OCD, bipolar disorder and schizophrenia [80-83]. Doctors should consider chronic caffeine anaphylaxis. An allergic reaction is capable of generating symptoms of anxiety [6, 84], bipolar disorder and schizophrenia [3,5,6]

15. Caffeine anaphylaxis reduces oxygen

Caffeine anaphylaxis decreases oxygen, increasing carbon dioxide (CO_2) [6]. Caffeine [85], excess adrenaline [86], exogenous or endogenous, and sepsis are capable of reducing blood flow. A reduction in blood flow is accompanied by an oxygen deficit and a CO_2 increase.

Excess CO_2 diminishes brain function, inviting hallucinations and delusions into the conscious mind [87]. Excess CO_2 also presents the risk of carbon dioxide sensitivity. An allergic reaction to CO_2 likely heightens the effects of ongoing caffeine anaphylaxis [6].

16. Decreased oxygen and mental illness

A reduction in cerebral blood flow may be common with mental illness. According to studies, there is evidence of decreased prefrontal cortex blood flow with major depression, schizophrenia and bipolar disorder [88-92].

17. Caffeine anaphylaxis increases lactic acid

When oxygen decreases, *anaerobic glycolysis* occurs, producing a small amount of ATP from glucose. Pyruvate, a glycolysis end product is converted to lactic acid (lactate) [6].

Caffeine anaphylaxis causes lactic acid production [6]. Anaphylaxis can cause lactic acid production [93] and caffeine increases lactic acid [94,95].

18. Lactic acid excess and mental illness

An elevated lactic acid level is indicative of aerobic dysfunction. And excess lactic acid, noted with schizophrenia [96], affects panic disorder patients [97]. However, because excess lactic acid often accompanies diabetes, doctors likely consider diabetes as the cause of elevated lactic acid levels in mentally ill patients.

19. Caffeine anaphylaxis, glucose and mental illness

Ongoing caffeine anaphylaxis fight or flight toxicity elevates glucose [6]. It is well known that fight or flight and sepsis raise the blood glucose level.

Because caffeine [98] and theophylline toxicity can elevate glucose [99], it is proposed that ongoing caffeine anaphylaxis fight or flight toxicity causes diabetes.

Doctors should consider a patient's caffeine intake and the symptoms of allergic toxicity before diagnosing diabetes or mental disorder. Diabetes is a prevalent condition of persons diagnosed with a major mental disorder [100,101].

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20. Caffeine Anaphylaxis and Aldosterone

Aldosterone helps maintain sodium and potassium balance and assists with the reabsorption of sodium into the blood. Aldosterone works to protect the body against hyperkalemia, an elevated potassium level.

During the early stage of ongoing caffeine anaphylaxis, because masked symptoms are minimal and go undetected, a doctor may not request an aldosterone level, an infrequently ordered laboratory test. Aldosterone goes untested until symptoms of psychosis increase.

It is supposed that aldosterone increases with an exacerbated allergic response to caffeine [6], because aldosterone increases with trauma and shock [102], is used as a marker of acute cerebral trauma [103], and is regulated by cyclic AMP. However, several substances inhibit aldosterone. Dopamine [104,105] and *natriuretic peptides (ANPs)* inhibit aldosterone [106]. Fluctuating aldosterone levels, proposed to be codependent on caffeine intake and the severity of toxicity, are expected during a course of allergic toxicity.

21. Aldosterone and mental illness

Aldosterone rises with mania and schizoaffective disorder [107, 108], and when introduced into rat cortical cells synthesizing aldosterone, serum of bipolar patients inhibits aldosterone [109]. This suggests an aldosterone inhibitor in bipolar patients, probably dopamine [6].

In caffeine allergic caffeine consumers, an aldosterone increase is suggestive of exacerbation of anaphylactic shock [6]. This rise is probably followed by a dopamine-increased aldosterone reduction [6]. Studies are needed to confirm.

22. Ongoing caffeine anaphylaxis, the dopamine hypothesis and psychosis

Researchers believe that caffeine in extreme excess increases dopamine. Contrary to this, by inhibiting MAO, ongoing caffeine anaphylaxis increases dopamine [6]. A study concluded that administration of caffeine to rats in amounts sufficient to cause behavioral changes stimulated a dopamine release in the shell of the nucleus accumbens [110]. Thus, the Dopamine Hypothesis, which contends that because amphetamine raises dopamine and dopamine is increased with schizophrenic psychosis, can be pinpointed to allergic toxicity in caffeine allergic persons [6].

23. Caffeine anaphylaxis and antidiuretic hormone

Antidiuretic hormone (ADH, vasopressin) assists in regulating water balance. When the sodium level rises, ADH is released to help the body conserve water. When the sodium concentration returns to normal, ADH secretion stops. A decrease in blood volume (hypovolemia) with an increase in blood solutes triggers ADH release [6].

Chronic caffeine anaphylaxis is capable of increasing ADH release [6]. ADH increases at the onset of septic shock, and later decreases [111]. With caffeine acting as a powerful diuretic and with toxins in the bloodstream, toxic fluid is forced through cell membranes, lowering blood volume [6]. Releasing ADH, the pituitary attempts to rectify faulty homeostasis [6].

The hematocrit level, a marker of the amount of packed cells in the body, is often interpreted as a marker of blood volume. This is a dangerous practice, enabling doctors to overlook dehydration and volume loss in patients.

24. ADH, syndrome of inappropriate antidiuretic hormone and mental illness

According to studies, ADH is elevated with mania and schizophrenia [112-114]. However, a report reveals increased ADH output with intracranial disorders [114], an indication that cerebral trauma raises ADH. The same report notes that ADH has risen with mania and in persons recovering from depression [114].

Results are suggestive of caffeine anaphylaxis. Chronic allergic response may elevate ADH, whereas recovery lowers ADH. Studies should be performed.

The hematocrit level is another factor of mental illness. Some psychotic patients have an elevated hematocrit level [115]. Likely the hematocrit level is falsely elevated in psychiatric patients due to dehydration and sepsis. This would help explain the exhaustion and loss of motivation of psychiatric patients.

Syndrome of inappropriate antidiuretic hormone (SIAH), a prevalent disorder of the psychiatric community, entails excess ADH secretion without an apparent cause. Given that SIADH can be generated by cerebral injury [114,116-118], doctors should consider ongoing caffeine anaphylaxis and its ability to cause cerebral damage.

25. Caffeine anaphylaxis, hyponatremia and polydipsia

Ongoing caffeine anaphylaxis can generate hyponatremia. Hyponatremia is a known symptom of brain swelling [119,120]. Increased ADH output causes sodium to move into brain cells.

Chronic caffeine anaphylaxis causes polydipsia, abnormal thirst [6]. Anticholinergic toxicity dries mucous membranes, and continuous fight or flight increases blood glucose and calcium, conditions linked with polydipsia.

26. Hyponatremia, polydipsia and mental illness

Hyponatremia and polydipsia are prevalent conditions of psychiatric patients. Clozapine is given to restore water balance in psychotic patients. However, before administering clozapine or another psychiatric medication, doctors should consider caffeine's ability to alter electrolyte balance and cause edema.

27. Caffeine anaphylaxis, cortisol and adrenal exhaustion

Cortisol, an extraneuronal uptake inhibitor [121], decreases catechol-O-methyltransferase (COMT) activity [121,122]. COMT is an enzyme required for catecholamine breakdown.

Cortisol is a key player in allergic toxicity. Ongoing caffeine anaphylaxis generates cortisol release [3,5-7]. Caffeine stimulates cortisol release [123,124] and cortisol is released during

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fight or flight and an inflammatory state. Cortisol quells allergic-induced inflammation, assisting in allergic masking [3, 5-7].

During the early stage of ongoing caffeine anaphylaxis, a reduction in COMT may contribute to catecholamine excess, endogenous toxicity and symptoms of psychosis [6]. But during late-stage ongoing caffeine anaphylaxis, cortisol decreases [5,6]. Hypocortisolism is connected with weakness and adrenal exhaustion, symptoms of adrenal insufficiency, which can be mistaken for chronic fatigue and/or depression.

28. Cortisol, mental illness, adrenal exhaustion and the residual stage of schizophrenia

Because cortisol is elevated with psychosis, cortisol is theorized to be an indicator of a developing psychosis [125]. Yet, doctors should consider chronic caffeine anaphylaxis as the cause of excess cortisol in psychiatric patients. The stress cascade, theorized to affect schizophrenics [126], indicates that stress or a substance is stimulating the hypothalamic-pituitary-adrenal (HPA) axis.

Generally doctors do not order a cortisol level for symptoms of adrenal exhaustion. But because psychotic persons exhibit symptoms of adrenal exhaustion [127,128], before a patient reaches a state of advanced allergic toxicity, a cortisol level should be drawn.

Doctors may be mistaking the mental changes related to *hypocortisolism* for the residual stage of schizophrenia. When cortisol decreases, COMT activity likely increases, contributing to catecholamine breakdown, reducing psychotic symptoms [6].

29. Caffeine anaphylaxis reduces prostaglandin production and increases the pain threshold

For decades, researchers have been trying to find the cause of prostaglandin deficiency as it pertains to mental illness. Ongoing caffeine anaphylaxis is a cause.

At high dose, caffeine [129] and theophylline inhibit prostaglandin production [130]. By generating the release of excess *glucocorticoids*, caffeine and theophylline may inhibit prostaglandin synthesis. Glucocorticoids suppress prostaglandin production, thus are useful analgesics [131].

Along with glucocorticoids suppressing prostaglandin production, chronic caffeine anaphylaxis generates lipolysis [6]. Caffeine stimulates lipolysis [132,133], and a study on guinea pigs showed that anaphylaxis stimulates lipolysis [134]. By stimulating lipolysis, caffeine anaphylaxis welcomes free fatty acids into the bloodstream, raising the free fatty acid level [6]. Chronic lipolysis can lead to fatty acid and prostaglandin deficits.

Moreover, during a course of caffeine anaphylaxis, when histamine decreases, prostaglandin 1 of the E series (PGE1) decreases [6]; PGE1 decreases along with histamine [135].

A prostaglandin deficit disables pain sensation. A high pain threshold contributes to allergic masking.

30. Prostaglandins, pain tolerance and psychosis

Schizophrenic patients exhibit a decreased PGE1-stimulated cyclic AMP accumulation in platelets [136,137], evidence of impaired prostaglandin production. And there is evidence of a fatty acid deficit with mania and depression [138].

The pain threshold is elevated in psychotic patients [139-141]. Decreased prostaglandin production with elevated pain tolerance suggests an ongoing process, allergic toxicity.

31. Ongoing caffeine anaphylaxis and theophylline

In addition to the above mentioned biochemical markers, a theophylline level is a useful marker for detecting ongoing caffeine anaphylaxis [6]. Acute caffeine toxicity results in the presence of caffeine in the blood. Allergic toxicity presents with theophylline in the blood [11].

Low weighing molecules have a greater tendency to generate hypersensitivity [142,143]. Caffeine's molecular weight is 194.19 [2], whereas theophylline's molecular weight is 180.17 [144].

32. Caffeine anaphylaxis and psychiatric medication

According to Abram Hoffer, M.D., Ph.D., "Drugs will subdue all psychotic patients—but they will not recover. A psychotic sick from caffeine will not get well no matter what is used until the caffeine is removed" [145]. Despite this, a rapid increase in mental illness cases and an explosion of psychiatric drugs plagues the world.

Psychiatric medication reduces symptoms of ongoing caffeine anaphylaxis. Neuroleptic drugs reduce psychotic symptoms by decreasing cyclic AMP. Serotonin selective reuptake inhibitors (each a SSRI) decrease depression by raising serotonin. But SSRI drugs should never be taken with caffeine, because combining SSRI and MAOI agents can cause serotonin toxicity and suicidal tendencies. Chlorpromazine (thorazine) and other phenothiazine drugs exhibit an anti-histamine effect similar to diphenhydramine (benadryl). Therefore, a person allergic to caffeine taking a phenothiazine medication will experience relief from ongoing caffeine anaphylaxis [3,7]. It is suggested that benzodiazepines merely mask symptoms of caffeine toxicity [17].

33. Conclusion

Ongoing caffeine anaphylaxis fight or flight toxicity is a recently discovered condition that alters homeostasis, resulting in rhabdomyolysis, *systemic edema*, progressive toxic dementia, ataxia, SIADH, adrenal exhaustion and a menagerie of other physical abnormalities. Because caffeine allergy masks its own allergic response and doctors have not considered caffeine allergy, doctors fail to diagnose caffeine anaphylaxis. Resulting from caffeine's MAOI effects, caffeine consumers lose insight,

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continue ingesting caffeine and continue at risk for developing a caffeine allergy. Allergic toxicity increases caffeine's detrimental effects and generates a progression of toxic symptoms.

Considering the amounts of caffeine ingested by psychiatric patients, it is imperative that health care workers update their chemistry knowledge and consider ongoing caffeine anaphylaxis fight or flight toxicity as the cause of abnormal physical and mental symptoms, including ADHD, anxiety, panic, OCD, depression, bipolar disorder, schizophrenic psychosis, muscle weakness, hyperglycemia, SIADH and adrenal exhaustion.

The biochemical abnormalities of ongoing caffeine anaphylaxis include ATP reduction, cyclic AMP increase that can diminish with progressed allergic toxicity, excess CPK MM, an elevated catecholamine level, a fluctuating serotonin level, an anticholinergic response, reduced GABA, reduced oxygen, excess lactic acid, increased blood glucose, likely increased aldosterone output, excess ADH, an elevated cortisol level that can decrease with adrenal exhaustion, a fatty acid deficit and reduced prostaglandin production, biochemical abnormalities accompanying the majority of psychiatric patients.

Definitions:

Acetylcholine (ACh): the parasympathetic nervous system's primary neurotransmitter located in the peripheral nervous system, spine and brain. ACh is needed for proper muscle contraction and brain functioning.

Acetylcholinesterase: an enzyme required to break down acetylcholine into choline and acetic acid.

Aldosterone: a mineralocorticoid released from the adrenal cortex in response to adrenocorticotrophic hormone (ACTH) to help control swelling. (In response to any type of stress, including stimulation, fear, infection, and inflammatory conditions, the pituitary gland releases ACTH, also known as corticotrophin. In turn, ACTH activates many biochemical responses.)

Anaerobic glycolysis: the breakdown of fuel sources in the absence of oxygen to provide a small amount of ATP for energy to sustain cell life.

Anaphylaxis: an immunoglobulin E (IgE) mediated mast cell degranulation, (when mast cells eject cell mediators into the circulating bloodstream). (A mast cell is a type of white cell.) An explosive hypersensitivity reaction, anaphylaxis is capable of affecting all organs. Anaphylaxis always affects brain function.

Anticholinergic effects: a group of abnormal symptoms resulting from insufficiency of the parasympathetic nervous system. Effects include, but are not limited to, ataxia, dry mouth, muscle rigidity, delusions and clouding of consciousness.

Antidiuretic hormone (ADH): also known as vasopressin, a hormone secreted by the posterior pituitary to help regulate water balance; ADH promotes water conservation.

Atrial natriuretic peptides: amino acid compound secreted by cardiac atrium tissue in response to high sodium chloride (NaCl) concentration, low blood volume, or high extracellular fluid content.

Catecholamine: adrenaline, noradrenaline, and dopamine, each an amino acid and neurotransmitter. As a group, they are known as the catecholamines.

Cyclic AMP: the primary second messenger synthesized from ATP.

Fight or flight response: the body's natural reaction to a potentially dangerous situation. The fight or flight response is associated with excess sympathetic activity. However, the parasympathetic nervous system is also active as it attempts to restore homeostasis.

y-Aminobutyric acid (gamma-aminobutyric acid, GABA): an amino acid and neurotransmitter synthesized from glutamate by the enzyme glutamic acid decarboxylase (GAD). Located in the brain, GABA is responsible for natural inhibition.

Glucocorticoids: a group of steroid hormones, including cortisol and cortisone, released by the adrenal cortex. ACTH activates cyclic AMP to initiate the adrenal cortex release of glucocorticoids.

Homeostasis: sustained cellular equilibrium.

Hyperglycemia: the state of increased glucose production resulting from excess stimulation.

Hypocortisolism: underproduction of cortisol as a result of damage to the adrenal glands, generally caused by chronic stimulation and immune disorders.

Hyponatremia: the state of low sodium content with cell swelling (hypertonicity), generally due to decreased Na^+/K^+-ATPase activity. Hyponatremia is a sign of brain edema.

Lipolysis: the breakdown of fat stored in fat cells for fuel. Lipolysis occurs in response to adenosine antagonism and the stimulation of cyclic AMP.

Monoamine oxidase inhibitor (MAOI): any substance, usually a toxin, which prevents the enzyme monoamine oxidase from breaking down catecholamines. As a result, a MAOI agent elevates the catecholamine level.

Oxidative phosphorylation: an aerobic enzymatic process that synthesizes ADP to ATP for metabolic energy.

Phosphodiesterase: the enzyme required to break down cyclic AMP to AMP.

Polydipsia: excessively abnormal thirst.

Prostaglandin: a hormone synthesized from arachidonic acid, an essential fatty acid, in phospholipid membranes. Stressful conditions, including anaphylaxis and fight or flight, promote tissue prostaglandin release.

Systemic edema: swelling throughout the body due to fluid accumulation.

Theophylline: a chemical classified as a methylxanthine, a bronchodilator, and a break down product of caffeine. Theophylline is also a drug commonly prescribed for chronic asthma.

About the Author

The author of this manuscript is caffeine allergic. In 1975, after caffeine cleared Whalen's system, she reintroduced it in the form of Coca-Cola®. She immediately suffered symptoms of anaphylaxis. Believing that this senior in high school was suffering from a reaction to clams, because she had ingested them earlier in the week, an emergency room doctor injected Miss Whalen with adrenaline. In doing so, he induced acute psychosis. With continued caffeine intake, Whalen's psychosis increased and progressed. Although she had desired to be a dentist, Miss Whalen became a medical laboratory technician. During her illness, she worked for 14 years in a Massachusetts hospital in the areas of chemistry and immunology. Despite Miss Whalen's desperate attempts to seek adequate medical care for idiopathic symptoms of ongoing caf-

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feine anaphylaxis fight or flight toxicity, doctors misdiagnosed her for 25 years and in 1999 Miss Whalen was institutionalized. Despite Miss Whalen's abnormal laboratory tests, direct indications of a physical disorder, suspecting mood and personality disorders, a psychiatrist forced her to take neurontin. In the privacy of her home on the advice of a friend, Miss Whalen discontinued using caffeine but took a neurontin. The lack of caffeine in conjunction with neurontin caused a seizure. On December 10, 1999, a Massachusetts doctor diagnosed "caffeine allergy/toxicity." Thus began Miss Whalen's research. Miss Whalen's recovery spanned two years. Her articles have appeared in the *Journal of Orthomolecular Medicine*, the *Townsend Letter for Doctors and Patients* and *Positive Health Magazine*.

Ongoing caffeine anaphylaxis is a masked cerebral allergy that targets organs, including vulnerable brain tissue, and alters biochemical reactions. Chronic allergic response progresses: Vascular walls break down, and toxins invade organs, including the brain. Hence, the glassy-eyed look of caffeine-consuming patients diagnosed with bipolar and schizophrenic psychosis. Mental symptoms include attention deficits, loss of insight, focus, judgment and values, delusions, procrastination, anger and a sense of self-loss. Unable to correlate the symptoms with a textbook disorder, doctors diagnose ADHD and/or another mental disorder.

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