Content Analysis of Fever Handouts Online: Could Parent Education Materials Perpetuate Fever Phobia?

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Abstract

Background. Fever is one of the most common reasons children present to their physicians. Fever phobia has been identified as a major contributor to parental concern over the presence of fever and many parents identify the internet as a preferred source of information on fever.

Objective. The purpose of this study was to identify the extent to which information on the internet contributes to the parental misconceptions which perpetuate the fever phobia phenomenon.

Methods. Articles addressing fever in children in the top 20 hits on all three of the top search engines were included. A content analysis of ten sites was performed using emergent coding methods and ten categories were determined. Reliability was acceptable (Cohen's Kappa = 0.791). Discrepancies were corrected by consensus. Frequencies of individual categories were computed.

Results. The categories and their respective frequencies were assessment of fever (22.2%), when to call the doctor (17.2%), how to treat fever (16.9%), definition of fever (15.9%), other (6.4%), how not to treat fever (6.2%), parents' perception of fever (4.7%), workup of fever (4.4%), symptoms of fever (3.7%), and febrile seizures (2.4%).

Conclusions. Internet articles relating to fever in children do not appear to contain information that could perpetuate the phenomenon of fever phobia. While alarming material regarding fever is present in the literature available on the internet, there appears to be a larger proportion of content dedicated to educating parents of the positive value of fever and to eliminate fear of fever.

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Introduction

Fever is one of the most common complaints in any pediatric patient encounter.[1] This may be due to the level of understanding of "what constitutes a fever" and the "causes of fevers" which vary across parents and the medical community. As a result, parental fear of fever and its potential outcomes are prevalent. Schmitt[2] first described "fever phobia" in 1980 and, since that time, many studies across many countries have examined this phenomenon.

As recently as 2008, a study found 91% of parents believe fever can cause harm.[3] Another study found parents have poor knowledge of fever and measure it inaccurately leading to needless consultations and hospital admissions.[4] However, the underlying causes of fever phobia are unclear.

As technology advances, more individuals are turning to resources such as the internet to learn about their medical

conditions.[5,6] In a study of internet use for health information, as many as 87% of respondents had routine internet access available and as many as 60% were using the internet for health related information.[6] In addition, information available online often is formatted as a handout or a portable document file (PDF) for easy printing and dissemination. Such handouts are often the mainstay of parent education programs[7], and have been shown to improve parent's knowledge and compliance with care.[8]

While the internet can serve as a great resource for parents, it can give inaccurate and dangerous information. Only 34% of parents who search for medically-related information on the internet discuss the information they find with their physician.[6] Moreover, as the generations who have grown up using computers begin to have children, it is likely the internet will become a more common source of information. Currently, internet use in younger parents is more than double that of older generation parents.[9]

Previous studies primarily focused on defining fever phobia and delineating misconceptions of fever. However, it is important to determine why these misconceptions arise and where inaccurate information is obtained. With expanding technology, one must explore the quality and content of information presented outside the physician's office. The purpose of this study was to determine what information is available to parents searching for fever health information via the internet and whether any of the material may enhance misconceptions of fever.

Methods

To identify fever websites, the term "fever" was searched utilizing the top three search engines identified by Nielsen NetRatings: Google, Yahoo, and MSN.[10] Search terms included all combinations of "fever" or "temperature" and "child" or "children". Websites were included if the information (a) was formatted as a handout, PDF, or had a printable version available, (b) was specific to fever, (c) had an English version available, and (d) was directed at a parent/guardian/adult. Websites were excluded if (a) it took more than three additional links to reach the handout, (b) the handout was more than four pages long, or (c) the handout applied to a limited population (those with epilepsy or seizure disorders). Handouts were included in the analysis only if all three search engines resulted in a hit. Ten handouts were identified and a Flesch-Kincaid[11] readability level was assessed for each. Then, individual ideas were unitized. A total of 1311 separate units were identified. Each website contained between 35 and 289 units with an average of 126.5 (SD=73.33).

A code book of different theme categories found within the fever sections of the websites was created. Emergent coding methods[12] were utilized following preliminary examination of data. Three researchers independently reviewed the materials to design a set of features identifying the initial checklists. Emergent coding methods resulted in ten categories (see Table 1 for complete list of categories). This method allowed the content itself to determine the categories. Two investigators independently reviewed the content and compiled a list of emerging themes. Next, they compared lists and reconciled any differences to develop a final checklist.

This consolidated checklist was used independently to apply coding and a periodic quality control check was established. The reliability code, utilizing the Cohen's Kappa, was acceptable at 0.791. Any discrepancies in coding were discussed by the two original coders and continuing disputes were settled by a third researcher.

Results

The Flesch-Kincaid[11] reading grade level ranged from 7th to 11th grade (M=9.37, SD=1.43). Frequency analysis of each theme category revealed that "assessment of fever" was mentioned most often and accounted for 22% of the coded units (see Table 1). Other frequently-mentioned categories included "when to call the doctor", "how to treat fever", and "definition of fever" which accounted for 17%, 17%, and 16% of coded units, respectively. Categories mentioned the least included "febrile seizures", "symptoms of fever", "workup of fever", "parents' perception of fever", "how not to treat a fever", and "other".

Varying representation of the categories was noted among the ten articles chosen for analysis (see Table 1). Two of the categories, "definition of fever" and "assessment of fever", were mentioned in all ten articles. However, two categories, "workup of fever" and "febrile seizures", were mentioned only in three of ten articles.

Discussion

Only four websites had readability levels below the 9th grade level. The average reading ability for the general population is 8th grade,[13] and information presented at higher levels may be misinterpreted, misunderstood, or ignored due to a lack of comprehension. Therefore, below the 9th grade level and preferably closer to the 5th grade level is recommended for written patient information.[14]

The themes in the articles chosen for analysis represent appropriate topics relating to fever in children. The content of some themes, such as "definition of fever" and "parents' perception of fever", largely focus on educating parents about the nature of fever and its role in fighting infection. Information of this sort should allay parental fears regarding fever in their children. The categories "how to treat fever", "how not to treat fever", and "when to call the doctor" help parents sort through the methods of caring for their child during illness and allow them to choose appropriate therapeutic techniques. Together, these five categories account for 60.9% of the total units analyzed. Therefore, a majority of the content within the ten articles chosen for analysis was intent upon educating parents and diminishing inappropriate or unnecessary treatment of fever.

While still supplying parents with accurate information, other categories such as "febrile seizures" and "workup of fever", tended to highlight the rare, but serious, effects of fever. Such information may serve as a source of parental concern.[1-5] However, as these themes accounted for only 2% and 4% of content respectively, this source of potentially fever phobia-inducing content is unlikely to be a major contributor to the phenomenon of fever phobia.

Content analysis of ten popular web articles relating to fever in children suggested such articles likely do not perpetuate or contribute to the phenomenon of fever phobia. While alarming material regarding fever was present in the literature available on the internet, there appeared to be a larger proportion of content dedicated to educating parents of the positive value of fever and the elimination of fear over fever.

This study had several limitations. Unitizing statements is a subjective process, even with independent reviewers. Codes may have been assigned inappropriately to certain units or important codes may not have been included. Assumptions were made regarding how parents access fever information and the use of the three internet search engines and online handouts. Other limitations regarding internet content also apply, such as rapidly changing information and content links.

Table 1. Frequency of theme categories on ten fever handouts.

Theme Category	Frequency (%)	# of Articles (%)	Definition	Examples	
Assessment of Fever	291 (22.2%)	10 (100%)	Concrete ways to take a temperature, types of thermometers used and the benefits/dangers and accuracy of each type, and how readings compare to one another.	If your child is younger than three months, you'll get the most reliable reading by using a digital thermometer to take a rectal temperature.	To test your thermometer's accuracy, bring it to your next visit to your pediatrician and compare the reading it gives against the one that your pediatrician uses.
When to Call the Doctor	226 (17.2%)	9 (90%)	Sign/symptoms of when to call the doctor or go to the ER, and when not to call a doctor.	If your child is less than three months old and has a temperature over 100.4 degrees F, call your doctor right away even if he or she doesn't seem sick.	You should also call the doctor if your child doesn’t wake up easily.
How to Treat Fever	221 (16.9%)	9 (90%)	Directions/guidelines to parents on assessment of fever, and directions on when to treat and how to treat fever, what to do if you suspect a fever, and how these methods work.	Because your body loses more water with a fever, be sure to drink plenty of fluids to avoid dehydration.	Treat fevers only if they seem to be causing discomfort.
Definition of Fever	209 (15.9%)	10 (100%)	What defines a fever, how fever is produced, and body changes and etiology of fever.	Because fevers may rise and fall, a child with a fever might experience chills as the body tries to generate additional heat as its temperature begins to rise.	Fever is a part of the body's defense mechanism against viruses or bacteria.

Other	84 (6.4%)	9 (90%)	Any statement that does not fit in the other categories.	The following information is for education only and should not replace the advice of your child's doctor.	Reye's syndrome is a serious illness that can lead to death.
How Not to Treat Fever	81 (6.2%)	9 (90%)	Directions/guidelines on how to not treat fever and explanation of why.	Infants under two months old should not be given any medications for fever without being evaluated by a doctor.	Do not use aspirin to treat fever in children.
Parents' Perception of Fever	61 (4.7%)	8 (80%)	How parents feel about fevers, irrational/rational fears about fevers excluding seizures, statements addressing parent fears, general information to allay fears.	Many parents also fear that untreated fevers will keep going higher and higher.	It is unlikely that your child's temperature will get high enough to be dangerous.
Workup of Fever	58 (4.4%)	3 (30%)	How to assess the etiology or cause of fever and what these tests mean, and how the tests are administered, what parents can expect from the testing procedure.	Treatment depends on the duration and cause of the fever and on other accompanying symptoms.	Your doctor will then perform a physical examination and may order tests.
Symptoms of Fever	48 (3.7%)	6 (60%)	Symptoms of fever.	Sick children are often tired and bad-tempered.	Sometimes kids with a fever breathe faster than usual.
Febrile Seizures	32 (2.4%)	3 (30%)	Definition and physiology of febrile seizures and consequences.	Although they're alarming for parents the vast majority of febrile seizures cause no lasting effects.	Call for emergency medical assistance if a seizure lasts longer than 10 minutes.
Total	1311 (100%)				

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Impact of Increased Amino Acid Intake on Very Low Birth Weight Infants

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Abstract

Background. A consensus has not been reached on whether higher doses of parenteral amino acids improve growth in preterm infants without causing adverse side effects. The objective of this study was to evaluate the impact of a more rapid increase in amino acids dosing in very low birth weight (VLBW) infants during their first few days of life.

Methods. The study design was a 1:1 ratio case matched retrospective analysis of data from two distinct periods when the hyperalimentation practice differed. During phase I, a five-day incremental dose of amino acids was utilized. In phase II, a two-step dose increase of amino acids was practiced. Duration of total parenteral nutrition (TPN), weight at 28 days, hepatic and renal function tests, and rates of complications were compared. This study was approved by two local IRBs.

Results. The phase II protocol resulted in an increased dose of amino acid delivered by day 7 (16 g/k vs 12.8 g/k, $p < 0.001$). This increased dose of amino acids resulted in a shorter duration of TPN usage (16 days vs 18 days, $p = 0.001$). The increased dosage did not have a detrimental effect on hepatic or renal function. It also did not increase the incidence of feed intolerance or bowel perforation. However, an increase in amino acid dosing did not increase absolute weight gain (15 g/k/day vs 13 g/k/day, $p = 0.282$).

Conclusions. No unfavorable outcomes were demonstrated with this increased amino acid regimen. VLBW infants may be able to tolerate even higher doses in the first month after birth.
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Introduction

Considerable research in the area of nutrition for preterm infants has resulted in a sea change in clinical practice over the last several years. Infants born prematurely miss out on an important period of in-utero protein accretion. Multiple etiologies for growth failure in the Neonatal Intensive Care Unit (NICU) have been recognized and one of them is the lack of early administration of intravenous amino acids.[1] Evidence suggested this influences growth and long term developmental outcomes.[2,3]

Most infants between 24 and 29 weeks gestation do not achieve the median birth weight of the reference fetus of the same gestation at the time of discharge.[4] For these preterm infants to match an intrauterine rate of growth, it has been postulated that amino acid infusion rates as high as 4 g/k/day would be required.[5] Amino acid supplementation at 3 g/k/day, soon after birth, results in a significant decrease in the number of infants falling below the 10th percentile for weight at 36 weeks corrected gestation and shortens time to full enteral feeds.[6] This may be responsible for improved weight gain.

Early introduction of amino acids with an increase up to a maximum of 3.5 to 4 g/k/day did not result in a higher incidence of adverse effects like hepatic or renal dysfunction.[7,8] Despite this, fears remain about the ability of very low birth weight (VLBW) infants to cope with high doses of amino acids.

Increased blood amino acid levels have been reported using 3.5 g/k/day when compared to 2.5 g/k/day, without the much desired improvement in weight gain.[9] So, does an increased blood amino acid level imply toxicity? Amino acid infusions of 3 g/k/day in neonates weighing less than 1300 grams resulted in blood profiles of amino acids that were equal to or less than those seen in 2nd and 3rd trimester fetuses sampled by cordocentesis.[10] This is supported by an earlier observation that enteral intakes approximating 3.2 to 3.5 g/k/day more closely mimicked intrauterine estimations for nitrogen retention.[11] Thus, amino acids at these doses are likely to be tolerated physiologically.

Historically, amino acids were introduced cautiously by increasing the dose gradually over several days. An evidence-based change in practice was made to administer a higher dose of amino acid at an earlier time in the first week. This change presented an opportunity to compare these two distinct periods for weight gain and the incidence of adverse events like hepatic and renal dysfunction. We hypothesized that this newer practice had resulted in an increased administration of amino acids, better weight gain, and minimal or no adverse effects. This investigation sought to replicate the findings of previous smaller studies with a larger population size.[7]

Methods

The phase I practice was to introduce intravenous amino acids at 0.5 g/k/day on day 1 and increase by 0.5 g/k/day up to a maximum of 2.5 g/k/day. The phase II practice was to start on day 0 with 2 g/k/day and increase to 3 g/k/day once the total intake reaches 100 ml/k/day. Beyond the first week, infants were advanced based upon individualized decision making to a maximum of 3.5 g/k/day in their total parenteral nutrition (TPN).

There is an inherent high degree of variability in protein administration. Infants in phase II are started at 2 g/k/d and only increased to 3 g/k/d when the fluid intake is increased to 100 ml/k/d. This increase in fluids is dependent on the infant's cardiovascular and respiratory status, hence is variable. Consequently, the "desired protein intake at 7 days" is not a set amount but the most the individual infant is clinically able to tolerate.

The study aim was to compare the two groups retrospectively with a convenience sample. Infants between the gestational ages of 23 and 33 weeks with birth weights between 501g and 1500g were selected. Infants who were small for gestational age, had congenital anomalies, died, or were transferred acutely were excluded.

The two groups were case matched 1:1 based on same week of gestation at birth and within 50g birth weight (see subject selection in Figure 1). The groups were compared for differences in weight gain, days of total parenteral nutrition (TPN) usage, and laboratory evidence of renal and hepatic dysfunction. Laboratory levels more than three times in excess of the upper reference range were considered clinically relevant. The incidence of complications like feed intolerance and bowel perforation were compared between the two groups. This study received approval from two local Institutional Review Boards.

Data analysis. Comparisons were made between the demographic characteristics of phase I and phase II subjects to establish similarity. Total protein doses at day 7 and day 28, duration of enteral feeding, total days of TPN, and weight gain at 28 days were compared between the two groups. The rates of adverse effects, namely hepatic and renal dysfunction, as well as feeding intolerance and bowel perforation also were compared. The Statistical Package for the Social Sciences (SPSS) 17.0 was used for

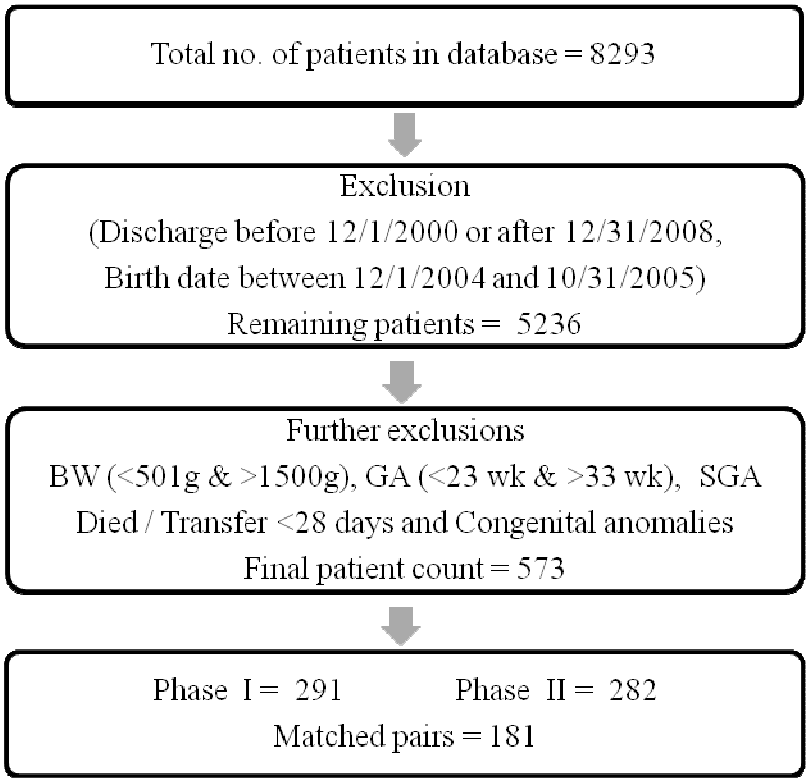

Figure 1. Characteristics of subject selection. Abbreviations: BW - birth weight, GA - gestational age, SGA - small for gestational age.

data analysis. Categorical comparisons were made using the chi square test and continuous comparisons by the Wilcoxon signed rank test. A p value of less than 0.05 was used to indicate statistical significance.

Results

Table 1 shows the demographic characteristics of the study sample. There were no significant differences between the two phases of clinical practice in gestational age, birth weight, gender, multiple gestation, and ethnicity. The duration of intravenous nutrition was significantly less in phase II (16 days vs 18 days, p = 0.001; see Table 2). The dose of amino acids received in phase II was significantly higher in the first 7 days (16 g/k vs 12.8 g/k, p < 0.001). However, when measured at 28 days, there was no significant difference.

As shown in Table 3, there was no significant increase in the incidence of feeding intolerance (20 vs 13, p = 0.201) or bowel perforation (0 vs 2, p = 0.499) in phase II. An increase in BUN was noted on day 1 in phase II (18 mg/dl vs 14 mg/dl, p < 0.001; see Table 3). Clinically irrelevant but statistically significant increases were noted in the day 7 BUN (19 mg/dl vs 18 mg/dl, p = 0.022) and creatinine (0.8 mg/dl vs 0.7 mg/dl, p < 0.001) in phase I. No significant differences in BUN or creatinine levels were noted at day 28. There were no significant differences in serum bilirubin, AST, ALP and GGT between the two groups at day 1, day 7, or day 28. There was no increase in the average weight gain noted at 28 days in phase II (15 g/k/day vs 13 g/k/day, p = 0.282).

Table 1. Demographics characteristics.*

	Phase I	Phase II	
Total number	181	181	
Gestational age in weeks (S.D.)	29 (2)	29 (2)	
Birth weight in kg (S.D.)	1.152 (0.252)	1.156 (0.247)	
Male (percent)	87 (48%)	105 (58%)	$X^2(1) = 3.593$, $p = 0.058$
Multiple gestation (percent)	10 (6%)	19 (11%)	$X^2(1) = 3.036$, $p = 0.081$
Ethnicity			
White (percent)	133 (73%)	142 (78%)	$X^2(3) = 2.139$, $p = 0.544$
Black (percent)	19 (10%)	16 (9%)	
Hispanic (percent)	23 (13%)	16 (9%)	
Other (percent)	5 (3%)	7 (4%)	
Missing (percent)	1 (1%)	0 (0.0%)	

* X^2 = chi square test

Table 2. Differences between Phase I and Phase II.*

	Phase I	Phase II	
Total protein dose (S.D.) 1st 7 days in g/kg	12.8 (6.7)	16 (7.5)	w = -4.686, $p < 0.001$
Total protein dose (S.D.) 1st 28 days in g/kg	84 (30)	85 (32.6)	w = 0.086, $p = 0.932$
TPN day count (S.D.)	18 (7)	16 (7)	w = -3.347, $p = 0.001$
Enteral feed day count (S.D.)	21 (5.3)	20 (5.5)	w = -0.788, $p = 0.431$
Weight gain in g/ day (S.D.)	13 (6)	15 (10)	w = 1.076, $p = 0.282$

* w = Wilcoxon signed rank test

Discussion

The change in protocol was associated with an increase in the total dose of protein administered by day 7 and a more rapid transition to enteral feedings resulting in a shorter duration of TPN use. Enteral intake reduces the protein intake given that Neosure (22 Kcal/ oz) at 150 ml/k/day yields 3 g/k/day of protein. Hence, improved enteral tolerance resulted in an earlier transition to enteral feeds and resulted in a lower total dose of protein at day 28.

As with a previous study, no hepatic dysfunction was noted.[7] No detrimental effects in the form of an increase in the incidence of feeding intolerance, bowel perforation, or renal dysfunction were noted. Contrary to our hypothesis, no clear advantage in terms of absolute weight gain was noted. This was consistent with one previous study.[9] In our study, infants were not followed to dismissal given the high transfer rate out of the practice to other sites

Table 3. Adverse effects and laboratory results.*

	Phase I	Phase II	
Feeding Intolerance (percent)	13 (7%)	20 (11%)	$X^2(1) = 1.634$, p = 0.201
Bowel perforation (percent)	2 (1%)	0 (0%)	$X^2(1) = 2.011$, p = 0.499
Day 1 lab results			
BUN mg/dl (S.D.)	14 (7)	18 (8)	w = 5.181, p < 0.001
Creatinine mg/dl (S.D.)	0.8 (0.3)	0.8 (0.3)	w = -1.753, p = 0.080
Bilirubin mg/dl (S.D.)	0.2 (0.2)	0.2 (0.2)	w = -0.222, p = 0.824
AST U/L (S.D.)	21 (23)	22 (28)	w = 0.229, p = 0.819
ALP U/L (S.D.)	118 (124)	134 (135)	w = 1.195, p = 0.232
GGT U/L (S.D.)	65 (92)	62 (76)	w = -0.011, p = 0.992
Day 7 lab results			
BUN mg/dl (S.D.)	19 (10)	18 (12)	w = -2.283, p = 0.022
Creatinine mg/dl (S.D.)	0.8 (0.3)	0.7 (0.3)	w = -4.023, p < 0.001
Bilirubin mg/dl (S.D.)	0.2 (0.2)	0.3 (0.2)	w = 0.208, p = 0.835
AST U/L (S.D.)	17 (15)	16 (14)	w = -0.646, p = 0.518
ALP U/L (S.D.)	213 (197)	210 (188)	w = 0.009, p = 0.993
GGT U/L (S.D.)	59 (94)	45 (52)	w = -0.603, p = 0.564
Day 28 lab results			
BUN mg/dl (S.D.)	3 (5)	3 (4)	w = 0.604, p = 0.546
Creatinine mg/dl (S.D.)	0.2 (0.3)	0.2 (0.2)	w = -0.940, p = 0.347
Bilirubin mg/dl (S.D.)	0.2 (1)	0.2 (0.6)	w = 0.880, p = 0.379
AST U/L (S.D.)	9 (16)	10 (18)	w = 0.055, p = 0.956
ALP U/L (S.D.)	172 (264)	171 (241)	w = 0.100, p = 0.920
GGT U/L (S.D.)	26 (55)	33 (64)	w = 1.674, p = 0.094

*w = Wilcoxon signed rank test, X^2 = Chi square test

and other care providers.

A limitation of this study was its retrospective design and the inherent possibility of failure to capture elements of care which might be recognized as influential. In both phases, the total dose of amino acids administered at times exceeded the maximum amount dictated by the protocol. The total dose was left to the individual clinician's discretion. Another such clinical decision was the timing of transition from intravenous to enteral nutrition. These factors might have contributed to some variability in the data.

Conclusion

Given that no unfavorable outcomes were demonstrated with the increased amino acid regimen, VLBW infants may be able to tolerate, and benefit from, even higher doses of amino acids in the first month after birth.

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Keywords: parenteral nutrition, amino acids, very low birth weight infants

Cardiac Complications of Acute Stroke

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Introduction

Cardiovascular abnormalities are common after a stroke.[1] Disorders of the central nervous system cause a wide array of cardiovascular system dysfunction ranging from electrocardiogram (ECG) changes and transient myocardial dysfunction to sudden cardiac death. Because the majority of stroke patients have risk factors that predispose them to have pre-existing cardiac abnormalities,[2] it is important to distinguish whether cardiopulmonary abnormalities cause the stroke, are caused by the stroke, or unrelated to the stroke.

Case Report

A 39-year-old, African-American male presented to the emergency department with expressive aphasia. A computed tomography scan of the brain was negative. A magnetic resonance image confirmed the presence of an acute peripheral infarct involving the posterior left frontal lobe inferiorly without other acute intracranial abnormalities. Further evaluation revealed a positive troponin T with a peak of 1.75 ng/dl (normal range = 0.00-0.06 ng/dl) indicating possible heart injury.

An electrocardiogram (Figure 1) showed the presence of peaked waves in lead V3 to V5 with 2 mm ST segment elevation in lead V3. The ECG findings suggested acute myocardial infarction. Emergent coronary angiography was negative for coronary artery disease. The ECG findings and the positive troponin were deemed to be due to the acute ischemic stroke. The patient was started on aspirin with resolution of his neurological symptoms and improvement of the ECG abnormalities in two days (Figure 2).

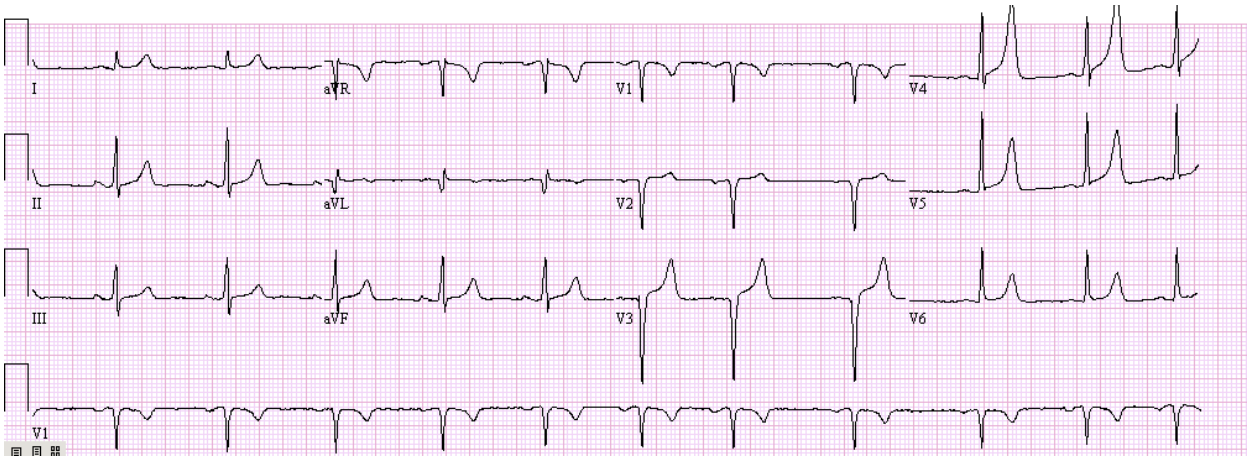

Figure 1. ECG showed peaked T wave in leads V3-V5.

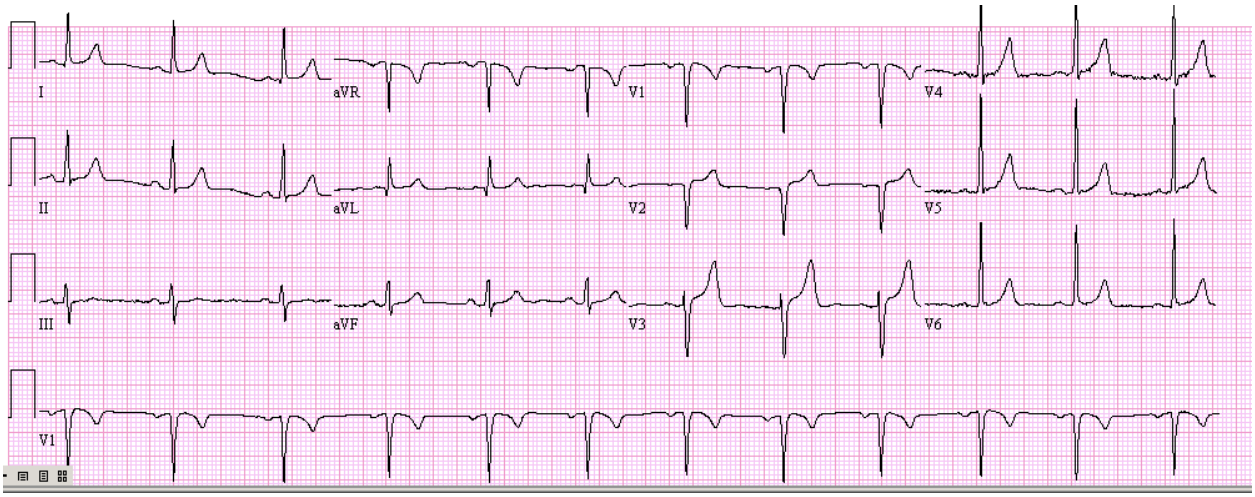

Figure 2. The follow-up ECG was normal.

Discussion

Two to six percent of all stroke patients die from cardiac causes in the first three months after ischemic stroke.[1] Regulation of the normal functioning of the heart by the central nervous system (CNS) is well recognized.[2,3] In fact, the CNS regulates the heart rate, blood pressure, vasomotor tone, and cardiac output and plays an important role in myocardial metabolism and cardiac contraction. Further, the CNS can affect the cardiovascular system by altering fluid and electrolytes balance. Cardiac systolic function also is affected after an acute CNS event.[4]

Myocardial injury following a neurological event may be due to catecholamine-mediated cardiac dysfunction.[2,5] An increase in intracranial pressure causes an acute surge of catecholamine release both systemically and at the neuronal synapses.[6] This surge is hypothesized to affect coronary microcirculation and alter membrane permeability. The latter is manifested by an increase of intracellular calcium that leads to a continuous stimulation of actin and myosin filaments that leads to cell death. In addition, free radicals released by the metabolism of catecholamine contribute to membrane damage and cell death.[2]

Coronary vasospasm caused by catecholamines has been evaluated as a possible mechanism for cardiac dysfunction, but studies have failed to confirm this hypothesis.[7] Other proposed mechanisms include disruption of the hypothalamic pituitary axis with depletion of thyroid hormones and cortisol.[2]

Cardiac dysfunction after a stroke is manifested by a wide variety of arrhythmias, ECG changes, elevated cardiac biomarkers, and hemodynamic alteration that can lead to cardiogenic shock and pulmonary edema.[2] Arrhythmias are common after stroke occurring in 78% of patients after a hemorrhagic stroke and 51% after an ischemic stroke.[8] Patients may present with sinus bradycardia, supraventricular tachycardias, atrial flutter, atrial fibrillation, ectopic ventricular beats, multifocal ventricular tachycardia, torsades de pointes, ventricular flutter, and ventricular fibrillation.[2,8] Cardiac arrhythmias are due to increased sympathetic tone and decreased vagal activity that are common in stroke.[2]

ECG changes were seen in 80% to 92% of patients with acute stroke.[2,9] Abnormalities included ST segment elevation and depression, T wave abnormalities, U wave, prolonged QT

interval, and pathological Q waves. New T wave abnormalities appeared in approximately 15% of patients with acute stroke. Upright prominent and flat or inverted T waves have been described.[2] The suggestion that these abnormalities are neutrally-induced is supported by the observation that inverted T waves may normalize if brain death occurs.[10] Nonspecific ST changes were the most common ECG abnormalities, excluding arrhythmia. ST changes are generally most apparent in the precordial and lateral leads and are usually transient.[11]

Physicians should be aware of another ECG manifestation called the J or Osborn wave. The J wave is a slow upright deflection that appears in the ECG at the end of the QRS complex or early portion of the ST segment. It generally can be observed in hypothermic patients. However, brain injury[12] and subarachnoid hemorrhage[13] have been reported to cause J waves. It is thought that heterogeneous distribution of a transient outward current–mediated spike-and-dome morphology of the action potential across the ventricular wall underlies the manifestation of the electrocardiographic J wave. The presence of a prominent action potential notch in epicardium, but not endocardium, is shown to provide a voltage gradient that manifests as a J wave or elevated J-point in the ECG.[14, 15]

Cardiac enzymes may be elevated after an acute intracranial neurological event. In an analysis of 149 patients with symptoms of acute stroke, 27% had elevated serum troponin.[16] Elevated troponin T is a poor prognostic sign after acute ischemic stroke. Troponin elevations correlated with the severity of neurologic injury and cardiovascular abnormalities including left ventricular dysfunction, pulmonary edema, and hypotension requiring pressors. In-hospital mortality among patients with an elevated troponin T was significantly higher than patients with a normal troponin T.

Echocardiographic wall motion abnormalities have been described in patients with stroke.[2] Some patients develop a transient regional left ventricular dysfunction that mimics myocardial infarction. In the absence of significant coronary artery disease this condition is known as takotsubo cardiomyopathy.

Conclusion

A wide variety of cardiac manifestations occurs in patients with stroke. The majority of stroke patients have risk factors that predispose them to have pre-existing cardiac abnormalities. It is important to distinguish the cause of the cardiopulmonary abnormalities. As seen in the illustrative case above, such abnormalities may be the complications of acute stroke.

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Keywords: acute stroke, cardiovascular disease, complications, case report

Urinary Vanillylmandelic Acid Levels in the Workup of Adrenal Incidentaloma

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Introduction

Although the majority of patients with pheochromocytoma have hypertension, 5-15% of patients are normotensive and this percentage may be higher in patients with adrenal "incidentalomas".[1-3] The diagnosis of asymptomatic pheochromocytoma is increasing in incidence, most likely due to widespread use of sectional imaging.[4] The most reliable method for diagnosing pheochromocytoma or paraganglioma is measurement of 24-hour urine catecholamines and metanephrines, with a sensitivity and specificity of roughly 98%.[5-7] The urinary vanillylmandelic acid level though may retain some value in the diagnosis of pheochromocytoma, particularly when paired with characteristic findings on imaging.

Case Report

A 68-year-old male underwent computed tomography (CT) of the abdomen as part of a work-up for back pain and an unintentional 20 pound (9 kg) weight loss over the previous six months. The scan revealed a right-sided adrenal mass 1.8 cm in maximum dimension. A follow-up CT scan after six months showed an increase in the size of the mass to 2.2 x 1.8 cm, with a non-contrast attenuation value of 13 Hounsfield units (HU). The mass was noted to have an irregular border. CT scanning of the chest was performed and demonstrated no evidence of other masses or adenopathy.

The patient denied fevers, flushing, palpitations, abdominal pain, or other constitutional symptoms. He was a previous smoker, with roughly a 30 pack-year history.

Physical examination and vital signs were normal. Laboratory revealed a normal random serum cortisol of 7 mcg/dl. A 24-hour urine collection for catecholamines and metanephrines was within normal limits, with total metanephrines of 772 mcg/24 hours (normal range 224-832 mcg/24 hours) and catecholamines of 55 mcg/24 hours (normal range 26-121 mcg/24 hours). The 24-hour urine vanillylmandelic acid (VMA) was elevated minimally at 9 mg/24 hours (normal range <9 mg/24 hours). Because of the patient's weight loss and the history of tobacco abuse, he was scheduled for laparoscopic surgical resection of the mass with malignancy high in the differential diagnosis.

Intraoperatively, the patient developed tachycardia of 140 beats per minute and hypertension, with a systolic blood pressure exceeding 260 mm Hg. He was given IV metoprolol and nitroglycerin with good results.

Pathology revealed a 1.5 cm tumor located in the adrenal medulla and compressing the adrenal cortex, consistent

with a pheochromocytoma. Low mitotic activity was noted. Neither tumor necrosis nor invasion into the periadrenal tissue was noted. Subsequent laboratory analysis, including a calcitonin level, was normal.

The patient recovered without incident and was discharged on no antihypertensives the day following surgery.

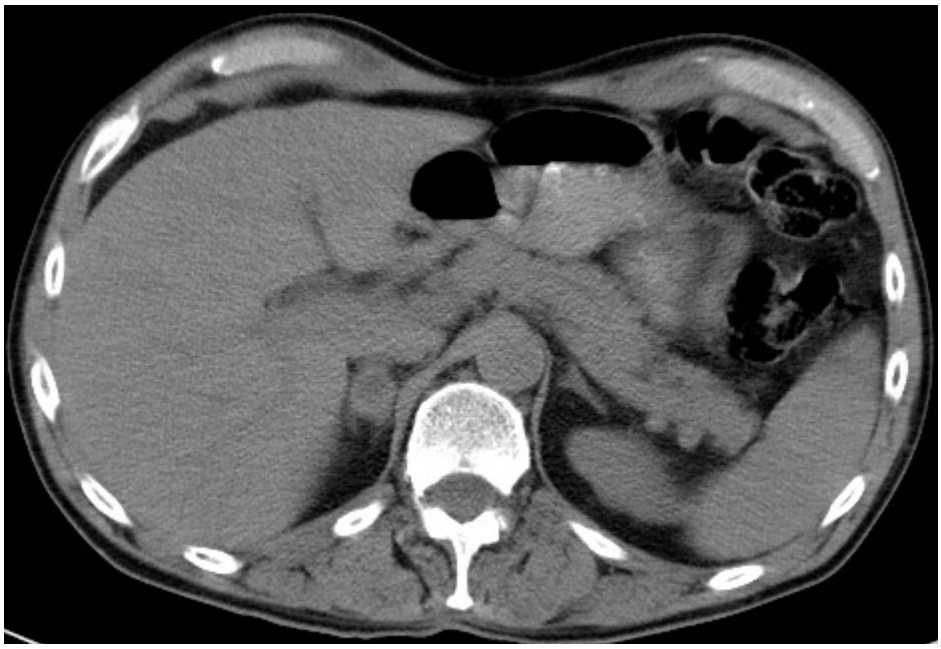

Figure 1. A 1.5 cm tumor located in the adrenal medulla and compressing the adrenal cortex, consistent with a pheochromocytoma

Discussion

Pheochromocytoma may be present in up to five percent of all patients with an adrenal incidentaloma.[7] Imaging characteristics typically help to differentiate between pheochromocytoma and adrenal adenoma. A Hounsfield attenuation value less than 10 HU on unenhanced CT scanning typically is associated with a benign cortical adenoma. A value of greater than 10 HU is associated with malignancy, pheochromocytoma, and less commonly adenoma (roughly 30 percent of adenomas do not have a large lipid content).[9]

Contrast washout may help differentiate between adenomas and nonadenomas.[10] Typically, adenomas are associated with rapid (> 50 percent at 10 minutes) contrast material washout in contrast-enhanced CT, whereas carcinomas or pheochromocytomas are associated with delayed (< 50 percent at 10 minutes) contrast material washout.

Beside the patient's slightly elevated urinary VMA and moderately high pre-contrast attenuation, little in our patient suggested the presence of a pheochromocytoma. The indication for surgery was suspicion for malignancy.

The urinary VMA has fallen out of favor as a routine test for pheochromocytoma. The elevated urinary VMA, though, should have changed management of this patient, as it has been shown to have a specificity of 96 percent for the presence of a pheochromocytoma. It is limited by its poor sensitivity of 76 percent.[11]

Conclusions

We depend on history, physical exam, laboratory, and radiological characteristics to diagnose pheochromocytoma, but sometimes the definitive diagnosis is regrettably established by pathology. The patient's history of weight loss led to the discovery of the neoplasm, but did not prompt the examiners to consider pheochromocytoma highly on their differential diagnosis. Closer attention to detail in this case, namely, notation of the relatively high attenuation of the lesion in combination with the elevated urinary VMA, may have led to appropriate alpha blockade and thus prevented an intraoperative hypertensive emergency.

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Keywords: vanillylmandelic acid, adrenal gland neoplasms, pheochromocytoma, case report

Conservative Management of Spontaneous Bladder Rupture

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Introduction

Spontaneous rupture of the bladder (SRUB) is an uncommon disease. The cornerstone of management is a high index of suspicion. In an era where alcohol intoxication is common, SRUB should always be in the differential diagnoses of acute abdominal pain, inability to void, and ascites. We report a case of SRUB secondary to alcohol intoxication presenting with elevated serum creatinine and abdominal ascites. Although reparative surgery was performed in most reported cases, conservative management was successful in this case.

Case Report

A 29-year-old, previously healthy man was admitted to the hospital for oliguric acute kidney injury. He had been intoxicated with alcohol the day prior to presentation. He reported having lost consciousness and passing out on the kitchen floor for more than seven hours. Upon awakening, he had abdominal pain and low urine output over the next 24 hours.

He presented to the emergency department where he received four liters of normal saline and subsequently was admitted to the hospital with an elevated creatinine of 4.86 mg/dL. He complained of mild diffuse abdominal pain, but denied hematuria or dysuria. He denied any recent nonsteroidal anti-inflammatory drug use.

His past medical history was negative. He smoked cigarettes and did not report any illicit drug use. He drank alcohol inconsistently, but had a tendency to binge drink every 2-3 weeks.

On physical examination, his vital signs were normal. His abdomen was tender, but soft to palpation. He had a positive wave sign, indicating the presence of ascites.

His white blood cell count was 13,700 ml with 80% neutrophils. His complete metabolic profile revealed a serum sodium level of 130 mEq/L, creatinine of 4.86 mg/dL, and BUN of 33 mg/dL. His urinalysis demonstrated 3+ blood and protein, with more than 50 red blood cells (RBCs)/hpf and 10-20 WBCs/hpf. Serum creatinine phosphokinase (CPK), uric acid, and phosphorus levels were within normal limits. A bedside bladder scan showed no urine in the bladder. A non-contrast abdominal CT scan revealed normal kidneys with a large amount of ascites (Figure 1).

A Foley catheter was placed and drained three liters of urine. A subsequent liver ultrasound showed complete resolution of the ascites. The patient was diagnosed with a spontaneous bladder rupture causing apparent renal failure and abdominal

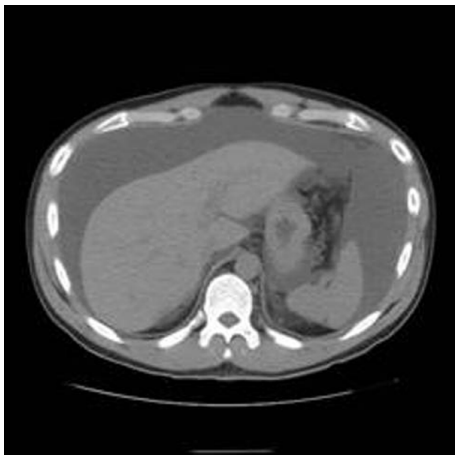

Figure 1. Abdominal CT scan revealing large amount of ascites

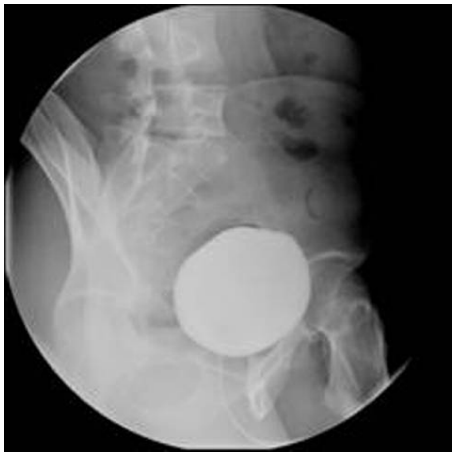

Figure 2. VCUG showing no urine leak from the bladder.

"urinary ascites". His serum creatinine dropped to 1.2 mg/dL the next day. He was discharged home with an indwelling Foley catheter. Two weeks after his injury, he had no bladder leak on a voiding cystourethrogram (VCUG; Figure 2).

Discussion

Rupture of the urinary bladder is an uncommon event. It often is secondary to blunt trauma, pelvic irradiation or bladder wall diseases.[1] SRUB is very rare and most of the reported cases occurred in association with alcohol intoxication. Late diagnosis and treatment of SRUB is associated with high morbidity and mortality of 50%.[2]

SRUB is caused mainly by bladder overdistention and thinning of the dome wall secondary to a large volume of urine produced by the diuresis effect of alcohol.[2] In addition, patients with alcohol intoxication have an impaired sensorium and may not feel the urge to void. Moreover, nausea and vomiting associated with alcohol abuse may increase intra-abdominal pressure and the likelihood of bladder rupture in the peritoneal cavity.

SRUB symptoms usually include severe abdominal pain and macroscopic hematuria[1], although only microscopic hematuria was present in our patient. The elevation of creatinine in this case likely was caused by direct absorption of urinary creatinine from the ascitic fluid.

SRUB should be suspected in patients with heavy alcohol intoxication, abdominal pain, and low or intermittent urine output. A computed tomography cystogram or retrograde cystography is the procedure of choice to confirm the diagnosis. Paracentesis also can be done and an ascitic fluid creatinine/serum creatinine ratio greater than 1.0 suggests intra-peritoneal urine leak.[3] Reparative surgery was the standard of care in most of the reported cases.[4] However, spontaneous healing of the lesion can be achieved if urinary leakage into the peritoneal cavity is prevented. This was accomplished in our patient with bladder catheterization for two weeks. Early diagnosis and treatment of this condition is crucial, since any delay may increase mortality and morbidity secondary to renal failure manifestations.

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Keywords: urinary bladder, spontaneous rupture, alcohol intoxication, case report

Therapeutic Benefits of Induced Hypothermia: In and Out of Hospital Cardiac Arrest

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Introduction

Cardiac arrest accounts for approximately 32,500 deaths per year and has a survival rate of 5% to 35%.[1] Following the arrest, if there is no oxygen to brain within few minutes, there is generation of free radicals and chemical mediators initiating a cascade of reaction resulting in cerebral injury (hypoxic injury).[2] Reoxygenation following arrest causes further damage due to necrosis and apoptosis of neural tissue (reperfusion-induced cell death).[3,4] Injury to the brain can continue up to 24 hours after the initial event and its extent depends on arrest time, reperfusion time, and severity of injury.[5] Only 11% to 17% of patients discharged following cardiac arrest have a good neurological outcome.[6] Therefore, post cardiac resuscitation should involve cerebral resuscitation.

Therapeutic hypothermia is believed to prevent this chain reaction and protect against ischemic/reperfusion injury resulting in better outcome[6] and cost effectiveness[7]. The patient should be cooled within 3-6 hours after cardiac arrest and maintained at a target temperature of 32-34°C for 24 hours.[1,8] Adverse effects of hypothermia can range from bradycardia to coagulation abnormalities, immunosuppression, and infections.[9]

Use of hypothermia in neurological surgeries and the knowledge that tissues recover from hypoxic injury better at low temperatures have been known for years.[9] However, it was only after 2002, when two groundbreaking studies were published that made induced hypothermia a routine part of post resuscitation care.[9,10] In both studies, patients who were in the hypothermic group had a favorable neurological outcome. Bernard and colleagues[10] found 49% of patients on the hypothermic protocol as compared to 26% of patients in the control group had better survival to hospital discharge with good neurological recovery. Good neurological recovery was described as patient discharged to home or rehabilitation. A European study[2] found that 55% of patients on the hypothermic protocol as compared to 39% in the control group had a favorable neurological outcome according to Pittsburg cerebral performance category of 1 (good recovery) or 2 (moderate disability) on five-point scale. Secondary end points were 6-month mortality and complications within a week of cardiac arrest.

In 2005, the American Heart Association recommended including induced hypothermia as a standard of care in treatment of both in and out of hospital cardiac arrest.[1,8] Yet, it has not gained popularity among American physicians. Merchant and colleagues[11] found the use of hypothermic intervention among physicians was 36% in Europe and only 26% in the USA. The

reasons that physicians gave for not using hypothermic intervention included they had insufficient data about the procedure, cooling was not part of Advanced Cardiac Life Support because lack of supporting data[10], the procedure was technically difficult, they lacked devices that cool rapidly[8], and there were insufficient human studies.

Case Report

A 40-year-old man presented to the emergency room with ventricular fibrillation. The patient had been found unresponsive at his neighbor's door (3:15 am). Emergency services arrived within five minutes (3:20 am). According to report, the patient had been motionless for unknown period of time. He was in ventricular fibrillation and compressions were provided by emergency services personnel. Rhythm was converted to ventricular tachycardia. He coded en route to the hospital. Another 200 joules cardioversion was given and he finally converted to sinus tachycardia (3:48 am).

The patient arrived to the hospital approximately 40 minutes after his discovery (3:54 am). On presentation, vital signs included a blood pressure of 176/136 mmHg, a pulse of 150 bpm, and an oxygen saturation of 99%. On examination, the lungs were congested with diffuse rales in all lung fields. He exhibited decorticate posture, spontaneous breathing, and a positive corneal reflex. Hypothermia immediately was induced with ice packs applied to his axilla and groin. Iced saline was started with an intravenous bolus for rapid infusion. The patient's core body temperature was maintained between 32-33° C by rectal thermometer.

The patient was placed on a ventilator and sedated with midazolam. Vecuronium was used to suppress shivering. He was monitored for any side effects of hypothermia including arrhythmias, electrolyte imbalance, and infection. Hypothermia was maintained for 24 hours followed by passive rewarming (0.25-0.5° C per hour over an 8-hour period). The cooling device was removed with monitoring for any rebound hypothermia and adverse effects.

According to family, the patient had been ill for the past month and complained of shortness of breath, abdominal pain, and chest pain precipitated by anxiety, emotion, and anger. Family history was positive for hypertension. The patient had a history of heavy alcohol use (greater than 20 beers/ day) and smoking (1-2 packs/day).

The patient had a slow response to hypothermic intervention. He awoke with continued neurologic findings of posturing and non-responsive arousing 48 hours after rewarming. Arousal with painful stimuli began to manifest on the third day. Spontaneous arousal, purposeful movement, and communication occurred on the fourth day.

A cardiology evaluation identified a 90% stenosis of the left anterior descending artery. Stenting was performed one week later. By week's end, the patient was walking and discharged with full neurological function.

Discussion

This case illustrates our experience with induced hypothermia as a part of post-resuscitation management. It emphasized the ease of administration and clinical benefit of a protocol for induced hypothermia. Early identification of eligible patients, inclusion criteria, and a rapid response in initiating hypothermia optimizes patient outcomes. Protocol procedures may be initiated by keeping iced saline in the emergency room refrigerator and applying ice bags to the axilla and groin. A rapid response time in initiating hypothermia and the importance of sedation in limiting cellular neurological damage have been

accepted widely and have become standard of care.[8] Hospital staff should review patient criteria and consider initiating the protocol as part of their initial evaluation of this patient population (see Table 1).

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keywords: induced hypothermia, out of hospital cardiac arrest, case report, Kansas

Table 1. Induced hypothermia protocol at Mercy Regional Health Center, Manhattan, KS.

Inclusion Criteria

- Post ventricular tachycardia/ventricular fibrillation arrest with return of spontaneous circulation (ROSC) within 60 minutes and Glasgow scale less than 10 for more than 45 minutes after ROSC where ACLS was started within 10 minutes form witnessed arrest.
- Platelet Count > 75,000.

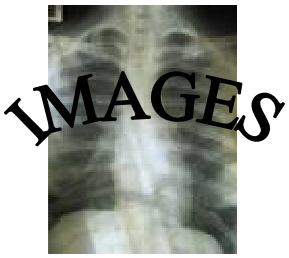
Exclusion Criteria

- Increasing neurological status (able to respond to command after ROSC).
- Pregnancy
- Trauma
- Known coagulopathies (check PT/PTT, fibrinogen, D-dimer)
- Oxygen saturation < 85% for more than 15 minutes after ROSC.
- Core temperature < 85^{o} F on admission.
- Hypotension for more than 30 minutes after ROSC or systolic BP < 90 mmHg despite use of vasopressors.

If the Criteria are Met

The patient is cooled for 24 hours to a goal temperature of 32□ - 33□C. The target time to reach the goal is 3-6 hours to achieve maximum effectiveness and should be started as soon as possible.

- Initiate Mechanical ventilator order set and place arterial line and Central Venous catheter.
- Apply external cooling with cooling blankets and application of ice packs to the groin, neck, and axilla. Completely expose the patient and place cooling blanket above and below with nothing between the blanket and skin. Cold saline infusion can be performed via a peripheral line to achieve the temperature goal.
- Monitor vital signs every 15 minutes during active cooling, then every 30 minutes during maintenance of target temperature. Place a continuous temperature monitoring device (rectal, bladder, or pulmonary artery).
- Medicate including analgesia, sedation, and paralytic per mechanical ventilation order.
- Obtain blood work include electrolytes, glucose, and complete blood count every six hours during the cooling period until normothermia. Blood cultures should be drawn at 12 hours after initiating of cooling.
- Rewarm after 24 hours of initiating cooling. The patient is rewarmed at a temperature of 1.8-3.6 degrees Fahrenheit per hour over an 8-hour period.
- The goal is normothermia. Once 36□C (96.8□ F) is reached, discontinue medications (titrate down slowly). Monitor for rebound hyperthermia, hypotension (due to vasodilatation), and hyperkalemia.
- Stroke services will continue to follow throughout and reassess the neurological status.



Air-Fluid Level

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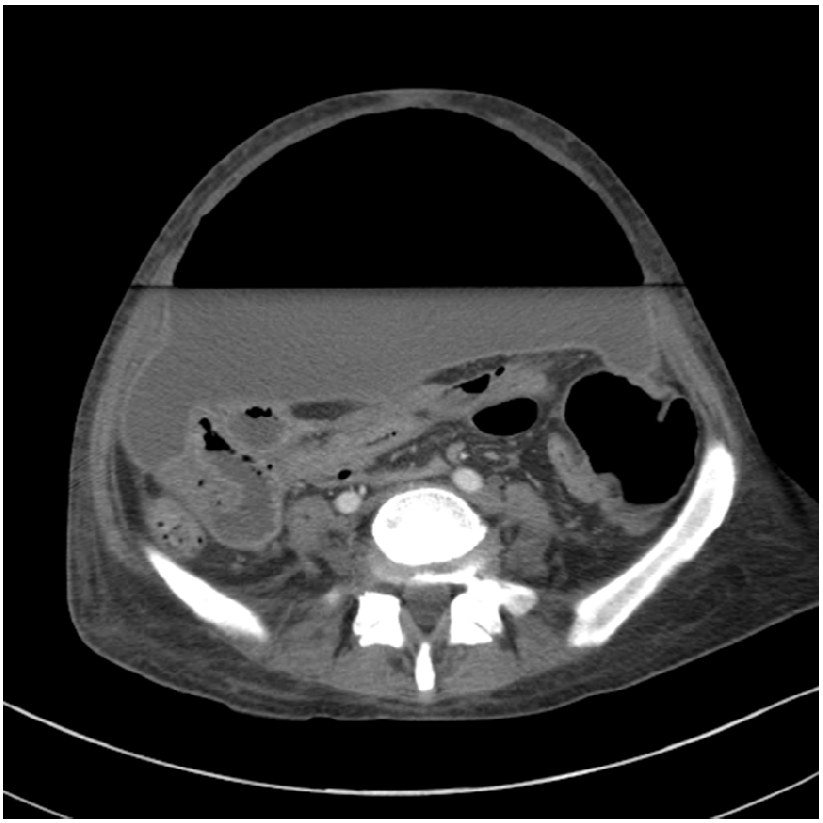

A 38-year-old woman with a history of Crohn's disease presented with progressive abdominal distention, nausea, and constipation of two months duration. She reported intermittent fever, diarrhea, and weight loss of ten pounds. The physical examination showed a tympanitic, distended abdomen with diffuse tenderness and guarding, but no rigidity. Bowel sounds were absent. The rectal examination was unremarkable, with guaiac-negative brown stool.

Laboratory investigations were pertinent for a potassium level of 2.4 meq/l, sodium level of 130 meq/l, white-cell count of 7,000 per cubic millimeter, hemoglobin level of 8.9 g/dl, platelet count of 534,000 per cubic millimeter, and a C-reactive protein of 20.4 mg/dl.

A plain abdominal radiograph of the abdomen revealed dilated small bowel loops with multiple air-fluid levels. Computed tomography of the abdomen showed a massive air-fluid level occupying much of the lower abdomen and pelvis (see image above) with several dilated small bowel loops. No mass or sign of pneumoperitoneum were identified.

The patient was started on intravenous fluid, ciprofloxacin, and metronidazole. A nasogastric tube was placed. Minimal improvement in the patient's symptoms was noted after 24 hours. An exploratory laparotomy showed a large intra-abdominal abscess with 1200 ml of purulent fluid drained. Culture of the fluid yielded growth of *Klebsiella Pneumoniae* and *Streptococcus Viridans*.

The development of intra-abdominal abscess has been reported in 7-28% of patients with Crohn's disease.[1] Spontaneous abscesses are the result of transmural extension of fissure ulcers, while postoperative abscesses may arise from intraperitoneal contamination during surgery, or from anastomotic leaks. Management traditionally has been surgical, but recent series have suggested that percutaneous drainage may have a role to play in the management of this complication in selected patients.[2] If the abscess is incompletely drained, 6-mercaptopurine was suggested to be preferred over infliximab. Sparse information, however, was available to guide medical therapy in Crohn's disease patients with intra-abdominal abscesses.

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Keywords: Crohn disease, abdominal abscess, case report

A Cardiac Cause of Exertional Dizziness

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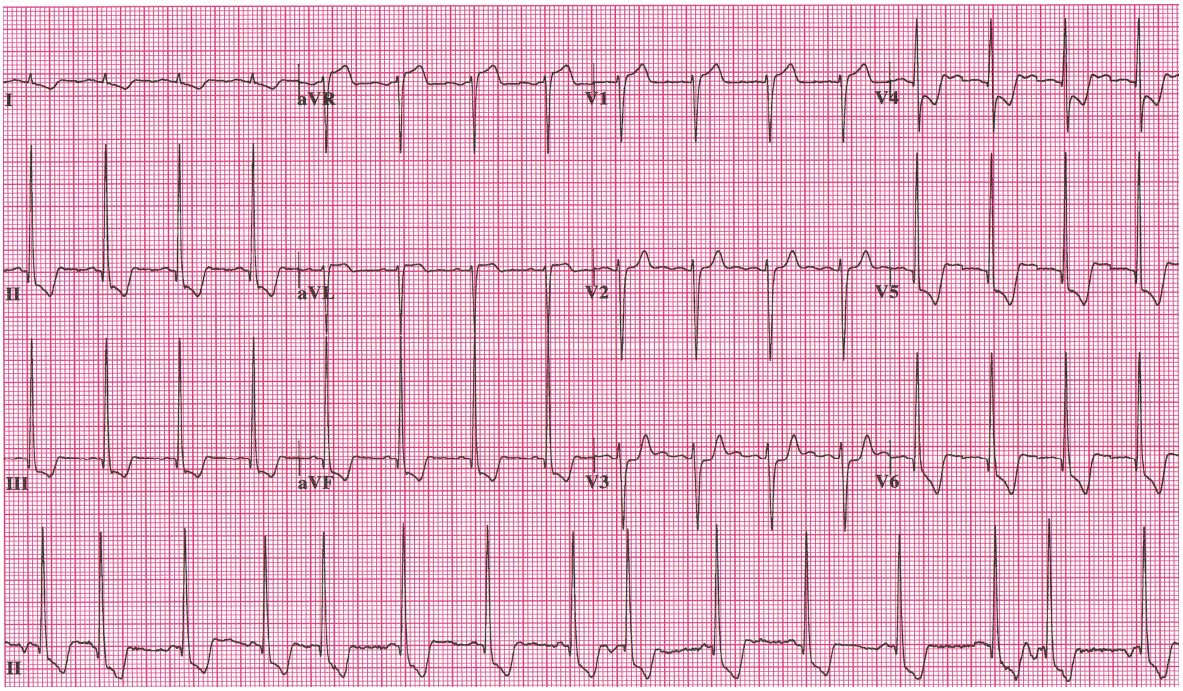
A 22-year-old male without obvious previous medical history presented for recurrent episodes of dizziness during the last year. He noted that his dizziness worsened with exertion, mainly when he was running or working in his garage. His dizziness also worsened during the summer time, especially when he was in the sun. The initial electrocardiogram is below.



What is the diagnosis?

A) Severe aortic valve stenosis
B) Inferolateral wall infarction
C) Hypertrophic cardiomyopathy
D) Atrial fibrillation with atrioventricular block

CORRECT ANSWER: C

The ECG reveals normal sinus rhythm with normally conducted atrial premature beats (beats 2, 5, 9, and 14). Left ventricular hypertrophy is seen (S wave in aVR ≥ 15mm; R wave in aVF ≥ 21mm; R wave in V5 + S wave in V6 ≥ 20mm) with associated ST-T abnormalities are evident (marked T wave inversion are noted thought the leads). A marked increase in QRS voltage and ST-T abnormalities with severe T wave inversion in a young patient with dizziness is highly suggestive of hypertrophic cardiomyopathy (the anterior and anterolateral Q waves are secondary to hypertrophic cardiomyopathy, not previous infarction). This patient underwent an echocardiogram revealing asymmetric septal hypertrophy with dynamic left ventricular outflow tract gradient (resting gradient 40 mmHg, post Valsalva gradient of 112 mmHg) consistent with the diagnosis of hypertrophic cardiomyopathy.

Discussion

Hypertrophic cardiomyopathy (HCM) is an uncommon genetic disorder that typically is inherited in an autosomal dominant fashion with variable penetrance and expressivity.[1] It is characterized by altered myocyte shape, size, and alignment, which along with increased myocardial fibrosis, results in marked ventricular hypertrophy, stiffness, and diastolic dysfunction. The vast majority of patients have abnormal ECGs with LVH in 50-65%, left atrial abnormalities in 20-40%, and pathological Q wave (especially leads I, aVL, V4-V5) in 20-30%. ST and T wave changes (repolarization changes secondary to left ventricular hypertrophy) are the most common ECG findings, while right axis deviation is rare.[2] The most frequent cause of mortality is sudden cardiac death, with risk factors including young age and a history of syncope and/or asymptomatic ventricular tachycardia on ambulatory monitoring.[1,2] Dizziness is common in patients with HCM with elevated pressure gradients across the left ventricular outflow tract. It is worsened by exertion and may be exacerbated by hypovolemia following high levels of exertion or increased insensible fluid loss (e.g., during extreme heat).[2]

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Keywords: hypertrophic cardiomyopathy, electrocardiography, dizziness