Head-banging with subsequent hemoglobinuria and acute renal failure[1]

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Acute renal failure was precipitated by hemoglobinuria secondary to head-banging in a retarded 12-year-old boy who had cyanotic tetralogy of Fallot and mild dehydration secondary to acute gastroenteritis. Hemoglobinuria is a not uncommon result of repeated pounding trauma to any extremity. Renal problems with hemoglobinuria generally occur in association with other renal risk factors such as dehydration and ischemia.

Index terms: Head, injuries • Hemoglobinuria • Kidneys, failure
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Traumatic or march hemoglobinuria has been reported following activities such as running[1,2] conga drum playing,[3,4] karate,[5] and head-banging.[6] Progression to acute renal failure is infrequently described, especially in the absence of concomitant myoglobinuria. In the patient described here, head-banging has led to several episodes of hemoglobinuria. Acute renal failure followed one such episode.

The role of isolated hemoglobinuria in precipitating renal failure is controversial. The toxicity of hemoglobin on renal tubules is probably potentiated by dehydration and ischemia, and, possibly, by increased blood viscosity. Our case report presents the unusual combination of self-inflicted blows causing hemoglobinuria in a patient with mild dehydration, cyanotic congenital heart disease with compensatory polycythemia, and acute renal failure.

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Case report

A 12-year-old black youth with severe psychomotor retardation, cyanotic congenital heart disease (baseline PO_2 60, saturation 85%) presented in October 1982 to The Cleveland Clinic Foundation with the chief complaint of an increase in self-abusive behavior, red urine, and fever. The patient chronically, forcefully bangs his temples with his fists. On several previous occasions the frequency of his behavior inexplicably increased from the usual few blows a day to blows every few seconds for many hours. On nine prior occasions, the patient's urine had turned transiently and uneventfully red with documented hemoglobinuria. With such an episode in September 1982, there was a free serum hemoglobin of 86 mg/dl (normal less than 5 mg/dl), urine Hemastix on Klini-Tek machine was strongly positive for hemoglobin, serum blood urea nitrogen (BUN) 17 mg/dl, creatinine, 1.7 9/24 hr, with negative qualitative urine test for myoglobin. The patient's urine subsequently remained clear, and his self-abusive behavior was minimal until 72 hours before admission.

The child had been in his usual state of health until three days before admission when fever, vomiting, and nonproductive cough developed. With the onset of illness, his mother noted marked increase of head-banging and the passing of dark red urine. The patient's urine remained dark for 48 hours, and he became anuric 24 hours before presentation.

Medications included a maintenance dosage of thioridazine (Mellaril), 50 mg/at bedtime and an over-the-counter preparation for cold symptoms.

The medical history was significant for congenital psychomotor retardation of unknown etiology. Surgical history included repair of esotropia and cryptorchidism, and placement of an aortopulmonary shunt for palliation of tetralogy of Fallot.

On examination the patient was combative and thin with microcephaly and abnormal facies; specifically, low hairline, long philtrum, and epicanthal folds. Skin turgor was poor, and distal extremities were cold. Temperature was 36° C; respiratory rate 20/min, pulse 116/min, blood pressure, 110/70 mm Hg; and weight, 23 kg. No rash was observed although the child had several patches of alopecia on the temporal and parietal scalp, and thick calluses on the knuc-

kles on both hands. His ear canals were atretic. His pharynx was mildly inflamed with tonsillar hypertrophy; mucous membranes were dry. The patient's neck was supple with bilateral anterior cervical lymphadenopathy. The lungs were clear. The cardiac examination revealed a continuous murmur on the right midclavicular line consistent with his history of Watterston shunt placement, and a III/VI harsh systolic murmur was audible over the entire left precordium. Pulses were regular but weak; mild cyanosis and 3+ clubbing were present. Results of the abdominal examination were normal. Genitourinary examination revealed Grade III hypospadias. Bilateral clinodactyly and flexion contractures of the knee joints also were noted.

The patient was admitted for rehydration and further evaluation. He was given an initial 20 ml/kg fluid bolus and then received maintenance fluids equivalent to 400 ml/m^2/24 hr. Fluid intake was liberalized when the patient began to produce urine. Urinary output was monitered via Foley catheterization. He required sodium polystyrene sulfonate (Kayexalate) for initial control of potassium; phosphate binders and renal dietary restrictions of protein and potassium were prescribed. Urine was negative for myoglobin on three specimens.

Renal ultrasound demonstrated normal-sized kidneys without evidence of obstruction, and renal flow scan was consistent with acute tubular necrosis. He entered the polyuric phase of ATN on the second day of his hospitalization. BUN and creatinine levels continued to rise until they peaked on the fourth hospital day (*Table*). He never required dialysis. BUN and creatinine levels stabilized, and his urine was free of dark brown sediment by the ninth hospital day. The patient did well and was discharged with no medication or dietary restrictions.

Discussion

Hemoglobin, unlike myoglobin, is thought to be relatively nontoxic to the kidney when not associated with certain other conditions. However, acute intravascular hemolysis seldom occurs as an isolated insult. Dehydration, hypotension, decreased cardiac output, and shock often occur in association with the original insult, causing hemolysis.

Hemoglobinemia and subsequent hemoglobinuria occur when the binding capacity for hemoglobin by haptoglobin and hemopexin are exceeded. This usually occurs after an episode of acute intravascular hemolysis. Free or unbound hemoglobin is then able to enter the renal tubular fluid. There it can be either absorbed (or reabsorbed) and degraded to hemosiderin by the proximal tubular epithelial cells, or excreted as hemoglobin if the tubular threshold is exceeded.[7]

Acute intravascular hemolysis with resulting hemoglobinemia/hemoglobinuria has been as-

Table. Laboratory values

	Day 1	3	5	7	8	1 Month Later
Na (mEq/L)	141	139	133	135	135	140
K (mEq/L)	6.1	4.0	4.0	3.9	4.5	4.8
Cl (mEq/L)	94	93	91	90	101	103
CO_2 (mm Hg)	23	27	...	18.1	19	23
BUN (mg/dl)	38	57	65	50.5	30	12
Creat (mg/dl)	4.3	7.3	6.8	3.0	1.8	0.7
Ca (mg/dl)	...	7.6	8.5	9	...	...
PO_4 (mg/dl)	...	8.6	6.1	3.4	...	...
CPK (mU/ml)	890	1106	>1200	284	...	...
SGOT (U/ml)	...	59	64	...	...	...
SGPT (U/ml)	...	29	...	...	...	...
Aldolase mU/ml	24	...	...	...	...	...
HCT (%)	59.6	54.5	56.5	56	...	...
WBC ($\times 10^3$ mm^3)	19	21	13.1	15.6	...	...
PLT ($\times 10^3$ mm^3)	199	269	186	191	...	...
Urine						
Hg	3+	3+	...	1+	...	...
Protein	2+	Trace	...	0	...	...
RBC	11–25	>25	...	3–5	...	...
SG	1.013	1.010	...	...	...	...
Color	Red	Brown	Yellow/brown sediment	Yellow/brown sediment	Yellow/brown sediment	...
Input (ml)	855	1252	1546	2527	850	...
Output (ml)	5	770	1460	1970	1020	...
Wt (kg)	23	23.3	23.7	22.6	23.6	...

Abbreviations: Na = sodium; K = potassium; Cl = chlorine; CO_2 = carbon dioxide; BUN = blood urea nitrogen; Creat = creatinine; Ca = calcium; PO_4 = serum phosphorus; CPK = creatine phosphokinase; SGOT = serum glutamic oxaloacetic transaminase; SGPT = serum glutamic pyruvic transaminase; HCT = hematocrit; WBC = white blood count; PLT = platelet count; Hg = free hemoglobin; SG = specific gravity; Wt = weight.

sociated with the following conditions: acute toxins, the infusion of hypotonic or hypertonic solutions such as distilled water or angiographic contrast media, sepsis with hemolytic organisms such as *Bacillus perfringens*, black water fever, G6PD deficiency, incompatible blood transfusions, burns, paroxysmal cold hemoglobinuria, and, as in our patient, march or traumatic hemoglobinuria.

Many theories exist about the mechanisms of the effect of hemoglobin on the kidneys. Tubular obstruction by pigment casts and pigment crystals was suggested as an avenue of damage by Baker and Dodds.[8] Maluf[9] felt that tubular obstruction would have to be combined with a low glomerular filtration rate before renal failure would ensue. The role of hemoglobin crystals as a cause of renal damage was subsequently discounted by Oliver et al.[10] They demonstrated that there were crystals in the distal tubules with no dilatation above them, thereby showing that the crystals were probably the *result* of the anuria instead of the cause.

Maluf[9] was unable to produce renal physiologic changes by the infusion of filtered hemoglobin or bolus infusion of distilled water in well-hydrated dogs. However, renal failure followed the infusion of hemoglobin in dehydrated dogs and the infusion of distilled water in animals in histamine shock. Blackburn et al[11] used continuous infusions of distilled water in 10 well-hydrated patients to cause intravascular hemolysis and hemoglobinuria. Seven of the 10 experienced increased vascular resistance, decreased renal plasma flow, and transient oliguria. These physiologic changes were reversed readily by discontinuation of the infusion and injection of parathormone, a renal hyperemic agent. Subsequently, the same patients were infused with hemoglobin washed free of erythrocyte stroma, and no renal physiologic changes occurred.

Baker and Davis[12] infused hemoglobin in rabbits, producing tubular epithelial necrosis in the proximal tubules with resultant decreased urine output. Theoretically, this showed that hemoglobin alone caused damage as it was being reabsorbed in the proximal tubules. However, he did not monitor fluid status in these animals, and the effects of possible dehydration cannot be ruled out. Lowe,[13] in response to Baker, studied the effects of renal ischemia and hemoglobinuria on rabbit renal tubules. She found that neither hemoglobinuria alone nor ischemia under 45 minutes duration caused tubular damage. However, 60 minutes of ischemia produced necrosis of 0.1% of the proximal tubules. Fifteen minutes of ischemia in association with hemoglobinuria produced the same changes in the same number of tubules. Hemoglobinuria and 30 minutes of ischemia necrosed 75% of the proximal convoluted tubules. Furthermore, she also demonstrated potentiation by hemoglobinemia of the renal damage associated with nephrotoxins such as potassium dichromate, mercuric chloride, mersalyl, sodium tartrate, and oxalic acid. Jaenike[14] confirmed the synergistic effects of renal ischemia, dehydration, tubular obstruction, and hemoglobinuria in the production of renal tubular lesions. Current theories on the association of hemoglobinuria and renal damage center on the role of hemoglobin as a potentiator for other disorders or toxins.

Previous case reports citing hemoglobinuria as the sole cause of acute renal failure generally have had other factors potentiating possible nephrotoxicity of hemoglobin. Rimer and Roy[15] presented two cases of hemoglobinuria in children who had been beaten. One subsequently suffered from acute renal failure, the other from transient oliguria. No myoglobin was detected in their urine, and their state of hydration was not addressed. Jackson described several cases of hemoglobinuria in acute renal failure, but these were in soldiers after strenuous exercise.[2] Presumably, dehydration and increased temperature must be considered in these cases. Hemoglobinuria associated with hand trauma has been described in congo drummers, but myoglobin, a recognized renal toxin, was also present in the urine. Hemoglobinuria as a result of head-banging has been reported previously[16] but was not associated with renal failure. The patient reported here experienced bouts of hemoglobinuria, but only progressed to acute renal failure in the presence of mild dehydration and fever.

Of particular interest is a description of hemoglobinuria[17] associated with the administration of contrast medium during cardiac catheterization in patients with complex cyanotic congenital heart disease. Precatheterization patients are often restricted to no food or water, and, as a result, become mildly dehydrated. Like the patient described here, they may have had cyanotic congenital heart disease with compensatory polycythemia. Given the association of angiographic contrast medium and subsequent hemoglobinuria in these patients, it would be particularly important to monitor their state of hydra-

tion. Possibly, routine intravenous hydration before catheterization would be indicated. In the event of a hemolytic episode as a reaction to contrast medium, aggressive hydration and alkalinization of the urine to prevent precipitation of hemoglobin and subsequent renal failure may be indicated. Our patient subsequently underwent cardiac catheterization, and, with careful hydration, maintained normal renal function without hemoglobinuria.

In summary, hemoglobinuria in the presence of risk factors such as dehydration or impaired circulation with ischemia may predispose to renal failure.

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