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CASE REPORT
Cosmetic

Acute Renal Failure following Novel Subcutaneous Fat Reduction with Injected Ice Slurry

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Summary: A new fat-freezing injection may pose significant health risks. Subcutaneous injection of partially frozen normal saline and glycerol has been shown to significantly reduce adipose tissue. This article reports the first human case and adverse reactions following this new procedure. *(Plast Reconstr Surg Glob Open 2021;9:e3719; doi: 10.1097/GOX.0000000000003719; Published online 12 July 2021.)*

A new fat-freezing "ice slurry" injection may become the next big weight-loss trend.[1] The Harvard-associated inventors of CoolSculpting have pioneered a new technique for freezing away unwanted fat. Subcutaneous injection of a partially frozen ice slurry containing normal saline and 10% glycerol has been shown to reduce adipose tissue thickness by an impressive 54.5% ± 5.9%.[2] This new method of cryolipolysis has only been studied in swine models, but its application for cosmetic weight-loss has quickly captivated national attention.[1]

Little is known regarding the safety of this new weight-loss method. It was first reported in the literature by Garibyan et al in early 2020.[2] A careful review of the literature does not reveal any human trials or FDA-approved use of ice slurry injections for weight-loss. We present the first human case and severe adverse reactions following this new procedure.

CASE REPORT

A 66-year-old woman was evaluated in the emergency room four days after receiving a new "fat-freezing injection." She was an excellent historian and recalled having approximately 1 L of partially frozen normal saline and 10% glycerol subcutaneously injected periumbilically and on each flank using a 15 gauge needle. This two-part solution was formulated from commercially purchased sterile saline and glycerol and administered by a healthcare provider. No other substances were used except local lidocaine. She developed nausea, emesis, and diarrhea within six hours of the injections, which persisted until presentation.

She visited the emergency room four days later after realizing she was no longer making urine. Her past medical history and medications included chronic kidney disease Stage 3A, atrial fibrillation (apixaban/metoprolol tartrate), and controlled essential hypertension (losartan). She denied taking any over-the-counter medications, including non-steroidal anti-inflammatory drugs. She was afebrile with a blood pressure reading of 105/60, heart rate of 69, respiratory rate of 16, and appeared ill with diffuse, mild abdominal tenderness on palpation. Laboratory diagnosis showed leukocytosis and acute renal failure (Table 1). CT imaging showed stranding in the subcutaneous fat throughout the flanks and abdomen with bilateral perinephric stranding.

She was admitted to the hospital service, and nephrology was consulted. Hemodialysis was initiated seven days postprocedure given indications of fluid overload and no improvement in renal function despite aggressive hydration and diuresis. A renal biopsy obtained on day 15 revealed acute interstitial nephritis (AIN) (Fig. 1). Seven glomeruli, two of which were globally obsolescent, demonstrated no proliferative changes. The interstitium contained moderate mixed inflammatory cell infiltrates consisting of small lymphocytes and eosinophils. No granulomas were seen. The proximal tubules contained intraluminal PAS+ casts without atypical or fractured light chain casts. Immunofluorescent histology showed focal vascular wall staining for C3 (3+). There was no significant staining for IgG, IgM, C1q, albumin, or fibrinogen. Electron microscopy showed no proliferative changes. The mesangial areas consisted of normal cell elements without immune complex deposits, fibrillary material, or

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Table 1. Laboratory Findings on Admission (Obtained 4 Days after Undergoing Ice Slurry Injections)

CBC		CMP		Urine*		Misc.	
WBC	14.65 K/mm^3	Na	126 mmol/L	Protein	3+	Lactate	0.7 mmol/L
Hgb	10.8 g/dL	K	4.6 mmol/L	Blood	3+	PTH	90.5 pg/mL
Hct	32.20%	Cl	93 mmol/L	Leuk	Trace	ANA	(−)
Plt	190 × 10^9/uL	CO_2	20 mmol/L	Nitrite	Neg	C3	100 mg/dL
MCV	89.4 fl	BUN	95 mg/dL	WBC	31–49/hpf	C4	18 mg/dL
Gran %	74.3%	Cr	10.18 mg/dL	RBC	0–2/hpf	CPK	111 u/L
Lymph %	17.2%	eGFR	3.7 mL/min/1.73 m^2	Cr	25.78 mg/dL	Phos	6.2 mg/dL
EOS %	1.4%	Glu	96 mg/dL	Total protein	>250 mg/dL	Mg	2 mg/dL
Gran #	10.89 × 10^9/uL	Ca	7.9 mg/dL	Na	132 mmol/L	Lipase	698 U/L
		Alb	3.3 gm/dL	K	6 mmol/L	FOBT	(+)
		T.bili	0.4 mg/dL	Cl	107 mmol/L		
		AST	33 U/L	Urea	37 mg/dL		
		ALT	34 U/L	EOS Smear	(−)		
		Alk phos	87 U/L				

* Urinalysis findings were obtained 8 days after the ice slurry injection was adminstered.

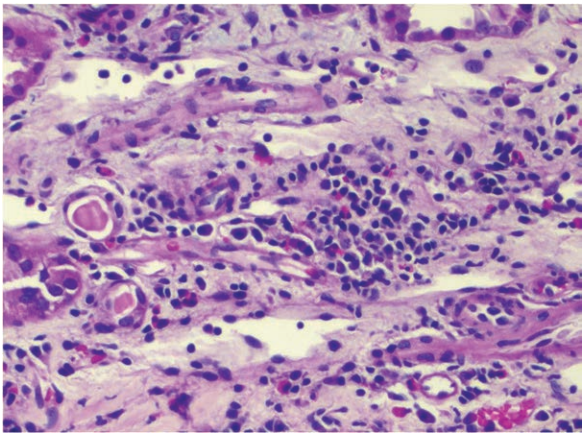

Fig. 1. High power renal biopsy image showing acute interstitial nephritis with a moderate mixed inflammatory cell infiltrate consisting of small lymphocytes and eosinophils (obtained 15 days after undergoing ice slurry injections).

amyloid. The visceral epithelial cells had intact podocyte foot processes.

Aggressive treatment for AIN was initiated with three days of methylprednisolone followed by 12 weeks of outpatient prednisone. She also required daily insulin injections for iatrogenic steroid-induced diabetes. Her kidney function improved to chronic kidney disease Stage 4, which allowed for discontinuation of hemodialysis 2.5 months after the original procedure.

DISCUSSION/CONCLUSIONS

Subcutaneous fat reduction with injected ice slurry may pose significant health risks. This patient developed a severe inflammatory response marked by nausea, vomiting, and diarrhea. Her AIN-induced renal failure with uremic buildup exacerbated her symptoms and resulted in dilutional anemia/hyponatremia (Table 1).

Alternative causes for this patient's AIN were absent. She reported no history of systemic inflammatory disease(s), recent infections, or new prescription/over-the-counter medications. Her baseline renal function 10 months prior (29 BUN, 1.16 creatinine, and 49 eGFR) was significantly different from that measured on presentation four days postprocedure (Table 1). Considering serum creatinine typically rises 1–2 mg/dL per day in severe acute kidney injuries, the provoking renal injury likely occurred around the time of her injections. Urine electrolytes did not offer significant diagnostic value because they were obtained on day 8. AIN was ultimately confirmed by renal biopsy on day 15 (Fig. 1). Three months of high dose steroid therapy was subsequently initiated, resulting in marked renal improvement and discontinuation of hemodialysis 2.5 months later.

The mechanism by which this novel procedure caused AIN is unclear. Intramuscular glycerol is a well-established experimental model for inducing rhabdomyolysis-associated acute kidney injury marked by intense cortical acute tubular necrosis and inflammatory cell infiltration.[3,4] Her urinalysis suggested myoglobinuria but the renal biopsy and admission creatinine phosphokinase level revealed no evidence of rhabdomyolysis (Table 1, Fig. 1). Instead, her biopsy was consistent with an inflammatory reaction to the normal saline/glycerol ice slurry injections. In swine models, Garibyan et al noted no signs of infection, laboratory abnormalities, or systemic side effects following this procedure.[2] These discrepancies highlight an urgent need for further research regarding the safety of this new weight-loss method.

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