Acute Cervical Spinal Cord Injury, Dopamine and Venous Hyperoxemia

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Cervical spinal cord transection causes major derangement of the sympathetic control of blood pressure, heart rate and body temperature. Postural hypotension is frequent due to peripheral pooling and augmentation of blood flow through the skin of the limbs, which forms 65% of the body surface [1]. Sympathetic fibers are numerous in the vicinity of arteriovenous shunts and arterioles, and sympathectomy results in loss of vessel wall tone and consequent peripheral AV shunting that lasts about 3 weeks [1]. This phenomenon, as well as venous hyperoxemia, has been reported after spinal anesthesia at the T10 sensory level [2]. Quadriplegic traumatically sympathectomized patients may additionally suffer from pulmonary ventilation-perfusion disturbances and pulmonary edema. Dopamine is recommended in those cases if the hypotension and oliguria persist despite volume replacement.

We describe a patient with high cervical spinal injury, quadriplegia and hypotension in whom dopamine hemodynamic support caused significant transient peripheral AV shunting and possible extension of the spinal cord damage.

Patient Description

A 23 year old previously healthy man was admitted after a vehicular accident with brain concussion and acute post-traumatic tetraplegia and anesthesia compatible with spinal cord damage at the C3 level. Intubation had been performed at the scene of the accident. On admission he was already fully conscious with blood pressure 90/40 TORR and sinus bradycardia of 43 beats/minute. He was given lactated Ringer's (Hartmann) solution, as well as an infusion of dopamine, 3 g/kg weight/min. Atropine in doses of 0.5–1.0 mg was given intermittently. Computed axial tomography demonstrated mild generalized brain edema, comminuted fracture of the C2 vertebra with posterior dislocation of the dens, and minimally displaced fractures of the C4. The patient was treated with a Gardner-Wells caliper and 1.5 kg weight skeletal traction. He was ventilated with 40% oxygen, using a Bennet MA-1 ventilator in the control mode.

Sixty hours after admission left-sided pneumonia developed. This was followed by diabetes insipidus which was treated with intramuscular pitressin. Routine venous blood sampling for electrolytes demonstrated bright red blood. An arterial blood sample was taken immediately and both the venous and arterial samples were sent for gas analysis, with identical results. Similar findings were noted 8 hours later when blood was simultaneously sampled from the femoral artery and from three separate venous sites: left and right superficial palmar veins and right dorsalis pedal venous arch. Twelve hours later the venous hyperoxemia disappeared and did not recur. Two days later the patient suffered irreversible cardiac arrest and died.

Comment

Sympathetic outflow derangement is a well-documented sequel of spinal cord injury. However, complete arterialization or hyperoxemia of the cutaneous venous blood in this context has not, to the best of our knowledge, been described previously. Although complete sympathectomy alone may have been responsible for this phenomenon in our patient, we believe that the concomitant use of dopamine may have played a role as well, since tetraplegics are known to be hypersensitive to norepinephrine [3].

Dopamine, 2–5 g/kg weight/min, causes vasodilatation in the renal, splanchnic and mesenteric vascular beds by stimulation of the dopaminergic receptors. Dopamine treatment may cause ischemia or gangrene of the fingertips due to stimulation of the alpha-receptors of the limb vessels and by increasing NE levels when given in higher doses [4].

Sympathectomy alone may cause a fourfold increase in the flow through the pre-capillary physiologic AV shunts (from 7 to 30 ml/min), resulting in hyperemia and elevated skin temperature due to filling of the subdermal

AV = arteriovenous

NE = norepinephrine

Table 1. Timetable, clinical and laboratory findings, and pharmaceutical treatment

	Time, post-trauma (hr)						
Parameters	**30 min**	**12**	**60**	**68**		**80**	
Blood pressure (TORR)	90/40	100/60	100/60	100/60		100/60	
Pulse rate (beats/min)	43	53	60–50	60–50		100–80	
Rectal temperature (C)	–	35.2	36.5	38.5		40.7	
Medications							
Dexamethasone (mg/day)	48	48	48			24	
Cimetidine (mg/day)	400	200 x 5	200 x 5			200	
Cefazoline (g/day)	2	1 x 3	1 x 3			1	
Atropine (mg)	0.5 + 1.0	1	–			–	
Pitressin (i.m.) (U/day)	–	5	5 x 2			10 x 2	
Dopamine (g/kg/min)	3	4	8–7			10–9	
Blood gases:							
pH:	7.38	7.40	7.42*	7.33			7.52
Arterial							
Venous	–	–	7.42	7.32	7.33	7.32	7.44
PO_2:	116	100	94*	102			200
Arterial							
Venous	–	–	90	71	80	80	43
PCO_2:	39	36	35*	69			31
Arterial							
Venous	–	–	45	65	61	65	34
Saturation:	98.5%	98.\5%	97%*	97%			99%
Arterial							
Venous	–	–	97%	91.7%	94.3%	94%	80%
HCO_3:	23.5	24	22*	37			25
Arterial							
Venous	–	–	28	33.5	32.0	33.5	28

* Approximately 20 minutes difference between venous and arterial samplings.

venous plexi [9]. We assume that the dopamine, although given within the vasodilating dose in our patient, caused capillary vasoconstriction, either alone or via NE to which such patients are hypersensitive. As a result, most of the arterial blood was shunted to the venous system, as demonstrated by the similarity of the blood gas analysis of samples from the two systems.

The combined effects of traumatic complete sympathectomy and administration of dopamine may have implications beyond extensive peripheral shunting. The bypass of arterial blood may be responsible for both high output cardiac failure and for the continuing hemodynamic instability in such patients. Dopamine may cause significant elevation of the intraspinal peri-lesional NE levels, and thus contribute to proximally progressing ischemic damage in the already injured spinal cord [5]. Fatal complications in such patients may be the end result of the proximally extending spinal damage itself, and/or blood gas abnormalities, cardiac hypersensitivity to NE, or the electrolyte disturbances associated with diabetes insipidus.

While the "spontaneous" disappearance of the venous hyperoxemia may have been due to constriction of the precapillary physiologic shunt induced by the elevated levels of dopamine and pitressin [Table 1] that were administered to the patient 80 hours after admission, it may only reflect some degree of sympathetic re-innervation. We suggest that dopamine may aggravate peripheral arteriovenous shunting and increase peripheral vasoconstriction via NE in traumatically sympathectomized patients.

Theoretically, isoproterenol or ephedrine is more suitable for cardiovascular support in these patients as these drugs have far less, if any, peripheral activity. However, this cannot be supported by the existing literature, and -adrenergic agonists are usually recommended for cardiovascular support in patients with hypotension due to failure of the sympathetic nervous system [6].

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