Gluteal compartment syndrome

A report of 4 cases

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Four cases of gluteal compartment syndrome are presented, 1 traumatic and 3 after an overdose of sedatives. Associated lesions included sciatic nerve palsy in 2 cases and acute renal failure in 1. Three cases with intramuscular pressure exceeding 60 mmHg underwent emergency fasciotomy. Intramuscular pressure monitoring is useful for evaluating the effect of fasciotomy.

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Gluteal compartment syndrome is rare and sometimes difficult to diagnose and treat. The onset of this syndrome is acute, with pain and swelling in the gluteal region, which is frequently associated with sciatic nerve palsy or acute renal failure, or both. Such patients account for three fourths of all the reported cases of gluteal compartment syndrome (Owen et al. 1978, Vukanovic et al. 1980, Rommel et al. 1986). Because of the severity of this syndrome, early diagnosis and treatment are imperative. The 4 cases reported here were analyzed as regards diagnostic methods and treatment of this syndrome, including measurements of intramuscular pressure.

Patients and methods

I reviewed the data on 4 patients who were admitted for gluteal compartment syndrome between 1987 and 1989 (Table 1). The cause of the syndrome was an overdose of sedatives in 3 cases and trauma in 1. The age range was 25–57 years, and 1 was a woman. Case 4 had a concomitant right ulnar nerve palsy from compression at the wrist. Three patients were treated by fasciotomy, which was performed through a posterolateral or posterior approach depending on the location of the damaged muscles. Using intraoperative intramuscular pressure monitoring, the fascia and the epimysium were incised until the pressure dropped below 30 mmHg.

Measurement of intramuscular pressure was carried out in the following way: a transducer is connected to a three-way stopcock and a recorder, a 22-gauge needle filled with heparinized saline is inserted in the muscle at the same height as the transducer, and the value indicated in the recorder is read. To determine a normal value with this method, intramuscular pressure of the gluteal muscles was measured in 28 patients, aged 70 (17–87) years, who did not have a compartment syndrome or any other muscle involvement. When they were measured, these patients were lying with the hip in a neutral position; moreover, they were under anesthesia immediately before an orthopedic operation.

The mean (SD) intramuscular pressure in the control cases was 13 (6.4) mmHg in the gluteus maximus and 14 (6.2) mmHg in the gluteus medius muscle.

Results

The histologic examinations revealed congestion, bleeding, and thrombosis in the small vessels, and swelling, degeneration, and necrosis in the muscle fibers (Table 2, Figure 1). The 3 cases subjected to pressure measurements had values 3–5 times higher than normal (Table 2).

The outcome in all of them was normal or near normal.

Discussion

Gluteal compartment syndrome is usually caused by prolonged immobility from drug overdose or by severe contusion; more rarely, it can be caused by inadequate operative positioning (Owen et al. 1978, Vukanovic et al. 1980, Rommel et al. 1986). The onset of circulation after long-term compression causes extravascular accumulation of the fluid with elevated tissue pressure, resulting from increased permeability of the capillary membrane (Hargens et al. 1978). In the gluteal compartment syndrome, all three separate sections of the tensor compartment, medius/minimus compartment,

Table 1. Observations in 4 patients with gluteal compartment syndrome

A	B	C	D	E	F	G	H	I	J	K	L	M	N	O	P	Q	R	S	T	U	V	W	X	Y
1	57	M	3	7	3	2	2	+	0	2	37	480	2.91	15	54	+	+	50	1,2,3,4	2	2	70	–	2
2	28	M	4	/	2	2	1	–	0	0	411	128	300	2.5	11	–	–	30	1	1.3	1	18	–	1
3	39	M	1	70	1	1	0	–	1	0	3.6	550	599	24	12	+	–	/	1	0	1	8	–	1
4	25	F	2	54	3	2	2	+	2	1	5.6	540	479	25	7	–	–	8	1,2,5	1	1	15	±	1

A Case
B Age
C Sex
D Cause: 1, 2, 3 coma induced by hypnotic overdose while in a supine (1) or a lateral (2) position, or by hypnotic overdosing and alcohol intoxication while remaining in a tailor position (3). (4) contusion caused by a fall from a 2-meter height
E Hours of compression
F Walking ability
  1 mild limp
  2 antalgic gait
  3 impossible
G Symptoms in the gluteal region
  1 painful swelling and muscle weakness
  2 same as 1 with tense muscles
H Pain upon motion of the hip joint
  0 no
  1 upon flexion
  2 upon flexion and upon adduction
I Diffuse edema in the lower extremity
J Bedsore
  0 no
  1 mild
  2 severe
K Sciatic nerve palsy
  0 no
  1 moderate
  2 severe
L CPK. Normal < 185 U/mL (Cases 1, 3, 4 – thousands)
M Serum myoglobin. Normal < 60 ng/dL
N LDH. Normal < 470 W–U (Case 1 – thousands)
O C-reactive protein. Normal < 0.4 mg/dL
P BUN. Normal < 23 mg/dL
Q Urinary occult blood
R Acute renal failure
  – no
  + with 5.5 mg/dL of serum creatinine
S Hours between awakening (or contusion) and operation
T Involved muscle
  1 gluteus maximus
  2 gluteus medius
  3 gluteus minimus
  4 short external rotators
  5 vastus lateralis
U Surgical treatment
  0 no
  1 fasciotomy with closed wound
  2 fasciotomy with open wound followed by secondary suture a month later
  3 muscle debridement and removal of a 344-gram hematoma
V Management for nephrotoxity
  1 transfusion
  2 hemodialysis performed 17 times
W Period from injury to recovery (days)
X Complication
  – no
  + 6 weeks later, subcutaneous infection cured by incision and drainage
Y Outcome
  1 normal
  2 normal gait but with mild weakness external rotators of the hip and numbness the foot

Table 2. Intramuscular pressure and histologic findings of the most severely damaged area in the muscle

Case	Site	Pressure (mmHg)			Histology	
		A	B	C	D	E
1	Gluteus max.	62	59	30	1.2	1.2
	Gluteus med.	74	71	22	2	3
	Gluteus min.		50	28	1.2	1.2
	Piriformis		47	28	3	3
	Gemelli		47	13	1	1
	Subcutaneous	7			1	
2	Gluteus max.	60		7	2	3
4	Gluteus med.	35	29	20	1	1
	Vastus lat.	66	61	9	2.3	3
	Subcutaneous				2.3	

A Pressure just before operation
B Pressure after incision of the fascia lata
C Pressure after incision of the epimysium
D Vascular change
  1 congestion
  2 bleeding
  3 thrombosis
E Muscle change
  1 swelling
  2 degeneration
  3 necrosis

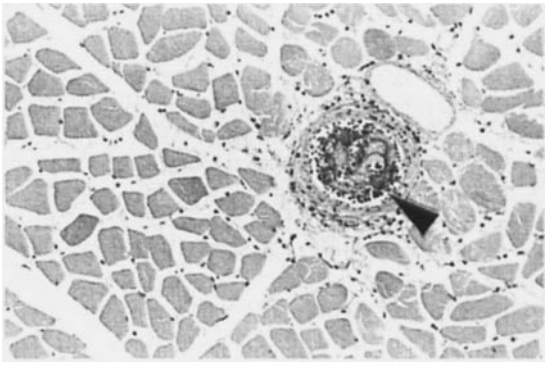
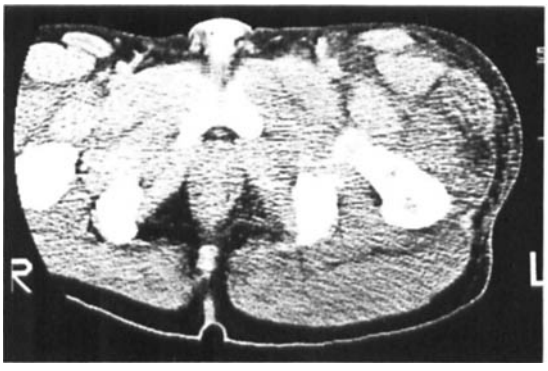
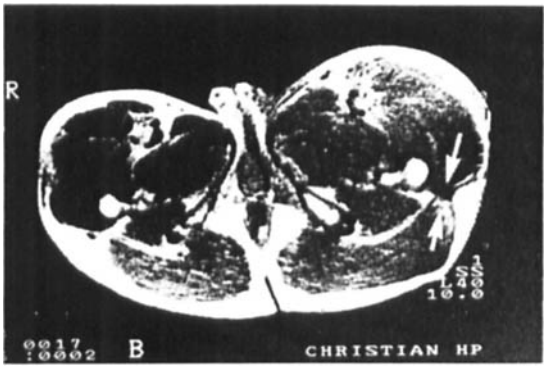


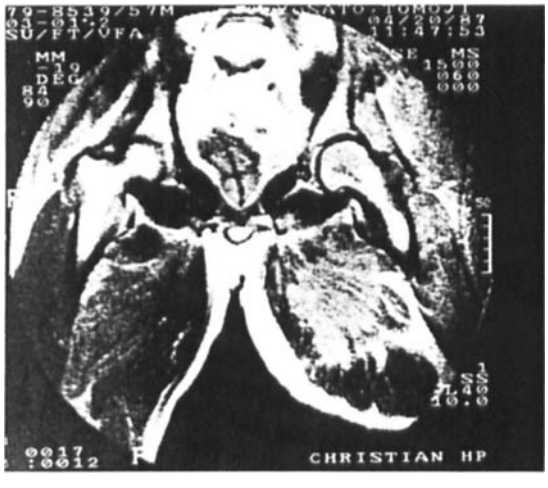
Figure 1. Case 4. Thrombosis of the arteriole indicated by black arrow and muscle necrosis in the vastus lateralis, HE ×150.



CT at the level of the greater trochanter, showing enlargement of the gluteus maximus and the short external rotators.



Axial magnetic resonance image with a T1 spin echo, showing a generalized increase in the signals in the gluteal muscles with enlargement. The white arrows point to a bright signal, indicating fluid retention beneath the fascia lata.



Coronary magnetic resonance image with a T2 spin echo showing a generalized increase in the signals in the gluteal muscles with enlargement.

Figure 2. Case 1.

and maximus compartment are affected (Owen et al. 1978), and also the piriformis may be involved (Vukanovic et al. 1980). Gluteal compartment syndromes take place in only one compartment or in any combination of compartments of the muscles of the gluteal region.

The problem of diagnosis lies in a poor understanding of this rare syndrome, which results in mistaking it for mere swelling by compression or contusion. In usual cases, painful swelling, pain associated with passive stretching, and muscle weakness are indications that might point to a compartment syndrome. However, in drug-induced comatose patients, a diagnosis of gluteal compartment syndrome is often delayed, and serious complications set in quickly. In those patients, attention should be focused on bedsores, which are an indication of prolonged skin pressure, and tenseness of the muscles, which suggests a high probability of this syndrome. The diagnosis is confirmed by ascertaining increased intramuscular pressure. CT and MRI are valuable examinations for determining the extent of muscle damage (Vulanovic et al. 1980; Figure 2).

All of my cases had hypermyoglobulinemia, and 2 of them had urinary occult blood, which is a simple and quick indicator of urinary myoglobin. Case 1 was not transfused with a sufficient volume of fluids, and contracted acute renal failure. Cases 2, 3, and 4 were transfused promptly. Transfusion with an alkalizer and a diuretic is necessary to prevent renal tubular damage by myoglobin (Rommel et al. 1986).

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