MALIGNANT HYPERTHERMIA: A CASE REPORT OF SUCCESSFUL MANAGEMENT*

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THE MORTALITY associated with malignant hyperthermia was 77 per cent in 1967¹ but had decreased to 64 per cent in 1970² primarily because of increased awareness by anaesthetists of the early signs of this syndrome and the prompt initiation of prompt therapy. The incidence of malignant hyperthermia in children is 1:10,000, with a range of 1:5,000 to 1:70,000.³ However, our experience at the Vancouver General Hospital indicates the incidence in adults differs markedly from that in children. During the past ten years (1961 to 1970 inclusive) approximately 290,000 general anaesthetics were administered at our institution to adult patients with the development of malignant hyperthermia in only one case.⁴ Patient ages have ranged from six months⁵ to 70 years,² but the majority of patients were less than 30 years of age.

At present we are aware of 176 patients who have developed this syndrome characterized by severe pyrexia of sudden or insidious onset, often associated with skeletal muscle rigidity, during or following the administration of a muscle relaxant and/or potent inhalation anaesthetic agent. The various aspects of this syndrome, i.e. aetiology,^{3,9,10-12} pathophysiology,^{10,11} clinical manifestations,^{1,2,8,13} laboratory findings² and management^{2,7,8,13,14} have been fully described in the literature.

Our reasons for presenting this paper are threefold: first, to describe our recent experience of an unpublished case of apparently succinylcholine-induced malignant hyperthermia in a healthy child; secondly, to illustrate an unusually rapid and fulminant rise of body temperature; and finally, to re-emphasize the important features of successful management of this syndrome. Such therapy includes continuous monitoring of body temperature, early recognition of signs characteristic of this syndrome, prompt cessation of anaesthesia and operation, immediate judicious treatment and continuous follow-up for 48 to 72 hours.

The following case report describes these principles, the application of which has led to the complete recovery of the patient from this serious anaesthetic complication.

CASE REPORT

An intelligent, healthy, well-developed seven-year-old East Indian boy (weight 21.5 kg) was admitted to hospital August 16, 1971, for an elective adenoidectomy. Apart from hypertrophied adenoids there were no pertinent negative nor positive aspects in his pre-operative history or examination and the blood profile

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TABLE I
CORRELATION OF LABORATORY RESULTS WITH BODY TEMPERATURE: DURING AND FOLLOWING THE ONSET
OF MALIGNANT HYPERTHERMIA IN A CHILD

Date Time (hours)	Normal values (V.C.H.) 38°C	Results correlated with time					
		August 17, 1971			2240	Aug. 18, 1971 1300	Aug. 19, 1971 1000
		1115	1215	1340			
Rectal temperature		38.2	37.6	37.9	40	37.5	37.2
Arterial blood gases							
F _i O ₂		1.0	0.90	0.21			
pH	7.35-7.45	6.99	7.22	7.31			
PaO ₂	90-100 mm. Hg.	450	212	114			
PaCO ₂	35-45 mm. Hg.	70	39	32			
HCO ₃	24-27 mEq/L.	16.2	15.6	15.6		24.5	
Serum							
K	4.2-5.5 mEq./L.	5.2	5.6	3.9		4.5	
Na	135-145 mEq./L.	134	131	134		138	
Cl	95-105 mEq./L.	96	95	99		95	
Ca	8.3-10.7 mg. %			9.7		8.5	
P	2-5 mg. %			2.5		4.5	
Mg.	1.8-2.8 mg. %	-	-	19		-	
Lactate	6-16 mg. %			0.48			
Pyruvate	0.2-0.6 mg. %						
Creatinine	up to 1.2 mg. %					0.7	
Serum enzymes							
SGOT	5-35 I.U./L.			917		1200	
SGPT	5-35 I.U./L.			108		280	
LDH	150-350 I.U./L.			2500		4450	
CPK	5-50 I.U./L.			55000			53200
Plasma							
PCLE	550-1300 I.U./L.	-				-	-
Blood							
Hgb.	13.4 gm. %			16.9			
Free hgb.	0			0			
Haematocrit	39%			45.3			
W.B.C.	8000/cu. mm.			16800			
Platelets	125000-350000/cu. mm.			225000			

TABLE I (Continued)

	Results correlated with time				
	August 17, 1971			Aug. 18, 1971	Aug. 19, 1971
Date				1300	1000
Time (hours)				2240	
Prothrombin time (quick)					
Partial thromboplastin time	80-100% activity				
Thrombin time	30-50 sec.				
Urine	15 sec.				
Myoglobin	0				
Free hgb.	0				
Net hgb.	0				
S.G.	0				
Protein					
Glucose					
Ketones					
				0	Present
				Present	Present
				1.006	0
				0	Trace
				0	0

Symbols:
 -: test not done.
 0: absent.

and urinalysis were normal. The child had not been previously anaesthetized. The family physician informed us that there were three other healthy siblings and that the patient's mother had a radical mastectomy without incidence. It was interesting, however, that the child's father had occasionally complained of vague skeletal muscle aches and discomfort that could not be correlated with any precipitating factors.

On the day of operation the patient received morphine 4.0 mg and scopolamine 0.3 mg intramuscularly one hour pre-operatively. At this time the heart rate and respiratory rate were normal. Rectal temperature was 37.5°C.

Anaesthesia was induced at 1107 hours with sodium thiopental 100 mg, followed by intravenous succinylcholine (20 mg) to facilitate intubation. No muscle fasciculations were noted but 20 seconds after the administration of succinylcholine, instead of the expected relaxation, sudden and profound generalized skeletal muscle hypertonicity occurred characterized by episthotonus, rigidity of the muscles of all limbs, abdomen and face which caused the jaws to be clamped firmly shut, thus making oral intubation impossible. Despite the decrease in chest wall compliance it was possible to ventilate the lungs with 100 per cent oxygen using a face mask and Ayre's T-piece circuit. At 1109 hours the temperature had increased to 37.8°C and spontaneous respirations returned even though the generalized muscle hypertonicity persisted. It was intensified at this time by an additional dose of succinylcholine (20 mg). Apnoea ensued again and continued for 16 minutes during which continuous vigorous ventilation with 100 per cent oxygen was again possible using a face mask.

At 1110 hours an alarming sequence of events developed. The skin was dry but extremely warm on palpation even though the temperature was only 38.2°C. Cyanosis of the lips and perioral skin was observed. Bizarre cardiac arrhythmias from supraventricular and ventricular ectopic foci predominated for three minutes and then changed to a sinus tachycardia of 160 per minute with the blood pressure varying between 90/50 to 100/60 mm Hg. Apnoea, coma and muscle hypertonicity persisted.

Vigorous therapy at this time included continued hyperventilation with 100 per cent oxygen, establishment of an intravenous infusion of five per cent glucose in water, initiation of surface cooling by packing the entire body in crushed ice, and monitoring of all vital signs.

At 1115 hours serial arterial blood gas analyses and serum electrolyte determinations were started and are recorded in Table I. Ten ml. of 7.5 per cent sodium bicarbonate was then administered. In spite of active therapy the rectal temperature increased from 38.2°C at 1115 hours to 40°C at 1120 hours after which time the temperature gradually decreased to the pre-operative level. Figure 1 correlated these serial changes in body temperature with the clinical manifestations of skeletal muscle hypertonicity and coma.

At 1125 hours shallow rapid spontaneous respirations resumed at a rate of 44 per minute.

At 1205 hours when the patient was transferred to the recovery room the skeletal muscle rigidity had disappeared. He responded to painful stimuli. The

FIGURE 1. Graphic representation of time sequence of events in the case reported.

blood pressure was 110/60 mm Hg, the EKG showed a sinus rhythm of 88 per minute, ventilation was adequate with a respiratory rate of 24 per minute and the body temperature was 37.8°C.

Eighty-three minutes after induction of anaesthesia the patient regained consciousness and although he was slightly drowsy he responded coherently to questions and complained of severe pain in his leg muscles and over his entire chest when he was asked to take a deep breath. Shortly after regaining consciousness he voided 75 cc of reddish urine.

His subsequent progress was interesting in that he had a rebound of hyperthermia to 40°C at 2240 hours accompanied by a sinus tachycardia of 126 per minute and tachypnoea of 32 per minute. This necessitated active cooling for a short period in order to restore his temperature within the normothermic range.

On August 18th he voided a large volume of dark brown urine in the morning and by the afternoon his urine was a clear colour. He also complained of severe aches and pains in his neck, arms, legs and abdominal muscles but these pains subsided dramatically during the following 24 hours and he was discharged home walking on August 19.

Changes in serial laboratory results during and following the onset of malig-

nant hyperthermia are recorded in Table I. They illustrate the correlation of the metabolic abnormalities with body temperature and therapeutic management of this syndrome.

DISCUSSION

The clinical manifestation of profound skeletal muscle hypertonicity occurring 20 seconds after the administration of succinylcholine prompted us to make an immediate diagnosis of malignant hyperthermia and to initiate the treatment as discussed. Serial arterial blood gas and serum electrolyte analyses guided the treatment for correction of the hypoxaemia and serious respiratory and metabolic acidosis. Continuous monitoring of the body temperature directed therapy for the hyperthermia.

The temperature rise was extremely rapid as illustrated in Figure 1. The onset of pyrexia occurred only two minutes after the administration of succinylcholine and reached a maximum of 40°C thirteen minutes after induction. This finding was considerably different than the observation by Britt and Kalow² who found that in the majority of patients who developed malignant hyperthermia the rate of temperature rise varied from between 2.0° and 8.0°F per hour. It is conceivable that the rigidity experienced by our patient may have been more pronounced resulting in excessive heat production and marked skeletal muscle cell damage as evidenced by striking elevations of the SGOT (serum glutamic oxalic transaminase), SGPT (serum glutamic pyruvic transaminase), LDH (lactic acid dehydrogenase) and CPK (creatine phosphokinase) in the serum less than three hours following the onset of rigidity.

During the period of fulminant hyperthermia and persistent skeletal muscle hypertonicity the use of procaine amide was entertained but was not used when the body temperature gradually decreased after reaching its maximum of 40°C. Beldavs *et al.*,⁷ reported the successful termination of hyperthermia and muscle rigidity following administration of procaine amide. We feel this drug deserves further study and may find a place amongst the agents used to manage these difficult cases.

This case demonstrates several important features of successful management of malignant hyperthermia. These may be summarized as follows:

- (1) Routine continuous monitoring of body temperature.
- (2) Early diagnosis by prompt recognition of the signs of malignant hyperthermia.
- (3) Immediate cessation of anaesthesia and operation upon diagnosis.
- (4) Prompt and judicious treatment guided by clinical signs, serial arterial blood gas and serum electrolyte analyses and monitoring of vital signs, i.e. body temperature, cardiac rhythm, CVP, urinary output and haematological parameters.
- (5) Continuous monitoring of vital signs for 48 to 72 hours. The importance of this aspect of management was demonstrated in our patient whose body temperature rebounded to 40°C twelve hours after the onset of malignant hyperthermia necessitating treatment similar to that given before.

Edelist⁶ and Denborough *et al.*¹⁵ have also advocated the pre-operative determination of serum CPK, SGOT and LDH values to screen patients in an attempt to detect those who might be affected. A family history of malignant hyperthermia should also give rise to a high index of suspicion that the patient may manifest this syndrome during anaesthesia.

SUMMARY

A case of succinylcholine-induced malignant hyperthermia has been presented to illustrate some of the problems usually encountered in the successful management of this syndrome.

Possible screening tests to detect patients who might be affected, principles of management and the correlation of laboratory findings with body temperature during malignant hyperthermia have been presented.

Emphasis is placed on the need for early diagnosis and prompt management of malignant hyperthermia to decrease the mortality of these patients.

RÉSUMÉ

Nous avons présenté un cas d'hyperthermie maligne provoquée par de la succinylcholine pour exposer certains problèmes à résoudre habituellement pour traiter ce syndrome avec succès.

Nous avons énuméré les tests possibles permettant de dépister les malades qui pourraient être atteints, les principes de la conduite à tenir et la corrélation des résultats de laboratoire avec la température durant l'hyperthermie maligne.

Dans le but d'abaisser le taux de mortalité chez de tels malades, nous insistons sur la nécessité de faire le diagnostic précoce de l'hyperthermie maligne et d'en instituer promptement le traitement.

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