Jpn J Radiol (2010) 28:1–7
DOI 10.1007/s11604-009-0372-x

ORIGINAL ARTICLE

Nontraumatic postmortem computed tomographic demonstration of cerebral gas embolism following cardiopulmonary resuscitation

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Received: May 10, 2009 / Accepted: August 26, 2009

Abstract
Purpose. The aim of this study was to investigate cerebral gas embolism (GE) on nontraumatic postmortem CT (PMCT), regarding its frequency, location (arterial or venous), and causes.
Materials and methods. Our subjects were 404 nontraumatically deceased patients who had been in a state of cardiopulmonary arrest on arrival at our emergency room. PMCT was performed within 2 h of the confirmation of death.
Results. Cardiopulmonary resuscitation (CPR) was performed on 387 of the 404 subjects; and of these, cerebral GE was detected in 29 (7.5%) subjects (3 arterial, 25 venous, 1 undeterminable). Cerebral GE was not noted in the other 17 of the 404 subjects who did not undergo CPR. However, there was no significant difference in the incidence of cerebral GE between the subjects who underwent CPR and those who did not. The mechanism of cerebral arterial GE was presumed due to pulmonary barotrauma and/or paradoxical embolism, while the thoracic pump theory was suggested to explain the cerebral venous GE.
Conclusion. Cerebral arterial/venous GE is found in CPR cases on nontraumatic PMCT.

Key words Postmortem computed tomography · Cerebral gas embolism · Cardiopulmonary resuscitation

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This article was presented at the Japan Radiology Congress, Yokohama, April 2008

Introduction

In light of the number of autopsies currently being performed, postmortem examination using imaging modalities such as computed tomography (CT), magnetic resonance imaging (MRI), ultrasonography, and angiography has been proposed as an alternative to or a concomitant method for autopsy.[1–13] In Japan, as the medical examiner system does not extend nationwide, about 80% of major facilities with emergency rooms (ERs) use postmortem CT (PMCT) to detect the cause of death in patients arriving at the ER in a state of cardiopulmonary arrest (CPA).[14–18] As the need for and frequency of PMCT increase worldwide for the purpose of determining the cause of death, diagnostic methods that aid the interpretation of PMCT findings such as intravascular gas in the heart, great vessels, and liver need to be established.[19,20]

Cerebral gas embolism (GE) is thought to occur accidentally,[21–23] iatrogenically,[24,25] and as a result of cardio-

pulmonary resuscitation (CPR) after CPA.[26–35] On PMCT imaged immediately before or after death in traumatically deceased cases, only arterial findings of cerebral GE due to CPR have been reported.[27–33] In contrast, both arterial and venous GE findings have been reported in nontraumatically deceased cases.[26,27,34,35] Any analysis of cerebral GE induced by CPR should exclude the possible effects of trauma. However, the published literature on the subject is limited, and the nature of GE remains unclear with regard to its frequency of occurrence and whether it occurs more commonly in arteries or veins. In this study, we investigated cerebral GE on PMCT in nontraumatically deceased subjects and discussed its possible mechanisms.

Materials and methods

Our subjects were 404 nontraumatically deceased patients for whom death was confirmed after arriving at our ER in a state of CPA between January 2000 and December 2007. They included 274 men and 130 women, ranging in age from 0 to 101 years (mean 67 years). CPR was performed on 387 of the 404 subjects during transport and in our ER by artificial respiration with bag-valve masking and intratracheal intubation, continuous chest compression, and infusion. CPR was not performed on 17 of the 404 subjects because they had already exhibited early signs of death (postmortem hypostasis is usually apparent within 30 min to 2 h following death, as is rigor mortis at about 4 h after death), although there was no indication of putrefaction.

Causes of death were diagnosed based on a comprehensive understanding of the patient's present illness, clinical history, and PMCT findings. A fatal hemorrhagic lesion was defined when PMCT detected a subarachnoid hemorrhage, cerebral hemorrhage, aortic dissection, or rupture of abdominal aortic aneurysm. For diagnosis of acute heart failure, a frequent cause of death, PMCT cannot detect direct findings, such as thromboembolism of the coronary artery or ischemic myocardium.[36] Therefore, we diagnosed acute heart failure when pulmonary edema was seen on PMCT, which is an indirect finding that results from cardiac pump failure.[37] Autopsy findings were also obtained in 29 cases for which we were able to secure family consent.

PMCT was performed within 2 h of the confirmation of death in the Radiology Department of our institution with the prior approval of the institutional review board. Two clinical CT scanners were used for PMCT. Until April 2004, PMCT was performed with a single-detector CT scanner (Accel Proceed; GE-Yokogawa Medical Systems, Tokyo, Japan) in conventional scan mode without using the helical scan technique. The scan parameters for the head were as follows: 120 kV, 160 mA, 2.0 s/rotation, contiguous 5-mm sections from the orbitomeatal line to the pentagon level, and 10-mm sections in the upper area. The scan parameters for the thorax, abdomen, and pelvis were 120 kV, 250 mA, 1.0 s/rotation, and 15-mm intervals with 10 mm collimation. A neck CT scan was omitted because the attending emergency physician, who performed CPR on the patient, thought that the cervical spine was not injured based on a comprehensive understanding of the patient's present illness, clinical history, and physical examination; thus, the cause was categorized as a nontraumatic death.

From April 2004, PMCT was performed with a 16-channel multidetector row CT (MDCT) scanner (Aquilion 16; Toshiba Medical Systems, Tokyo, Japan). The scan parameters for the head were conventional scan mode 120 kV, 200 mA, 2.0 s/rotation, 1 mm collimation, and contiguous 4-mm sections. The scan parameters for the thorax, abdomen, and pelvis were helical scan mode, 120 kV, 300 mA, 0.7 s/rotation, 1 mm collimation, pitch 15, and contiguous 10-mm sections. The neck region was not scanned for the same reason as described above. All images were observed on a 21-inch monochrome monitor with 1600×1200 pixels at appropriate window settings for each region.

A board-certified radiologist and board-certified emergency medicine physicians retrospectively reviewed PMCT findings of the brain, thorax, abdomen, and pelvis. We determined whether cerebral GE was present by means of consensus. Cerebral arterial GE[26–33] was defined as when one or more of the following arteries contained gas: the internal carotid artery (cavernous and supraclinoid segments) or the anterior, middle, or posterior cerebral artery. Cerebral venous GE[34,35,38,39] was defined as when the posterior cranial fossa or venous sinuses contained gas. Cardiovascular gas and other abnormal findings associated with cerebral GE were also recorded if present.

The chi-squared test for independence was used to determine whether differences between the incidence of cerebral GE in patients who received CPR and that in those who did not were statistically significant. A two-tailed probability value of $P < 0.05$ was considered statistically significant.

Results

Cerebral GE was detected in 29 (7.5%) of the 387 subjects who underwent CPR. No cerebral GE was detected in the 17 subjects who did not receive CPR. The differ-

ence between the two groups was not statistically significant ($\chi^2 = 1.35 < 3.84 = \chi^2_{0.05}$).

Arterial GE was detected in 3 of the 29 cerebral GE subjects: an 11-day-old male baby (case 1: cause of death acute circulatory failure); a 1 month-old female baby (cause of death: congestive heart failure and pneumonia), and a 2 month-old male baby (cause of death: pneumonia). PMCT of the thorax showed cardiovascular gas in the right atrium and bilateral ventricles in all three subjects. Autopsies performed on the 11-day old male infant and the 1 month-old female infant confirmed the presence of a right-to left-shunt. Also, the autopsies in these two cases revealed gas bubbles in the leptomeningeal vessels and in the bilateral anterior, middle, and posterior cerebral arteries. Autopsy was not performed on the 2-month-old male infant for whom the cause of death was diagnosed as pneumonia because of his signs and symptoms, including leukocytosis and extensively increased attenuation in the lung on PMCT.

Venous GE was detected in 25 of the 29 cerebral GE subjects. The causes of death of these 25 subjects were acute heart failure (17 subjects), pneumonia (3), aortic dissection (2), cerebral hemorrhage (1), suicide by ingesting hypnotic drugs (1), and choking on food (1). These 25 subjects showed either a spotty or linear-shaped venous GE in the cerebellum (case 2); one subject also showed GE in the cavernous sinus. PMCT of the thorax in 23 of these 25 subjects showed vascular gas in at least one of three veins: jugular, subclavian, and brachiocephalic. Autopsy was performed on a 37-year-old woman (who committed suicide by excessive ingestion of hypnotic drugs) and a 40-year-old man (who had acute heart failure). The autopsies in these two cases were not able to identify the gas bubbles that were shown on PMCT.

Cerebral GE was detected along the surface of the left temporal lobe in 1 of the 29 subjects, but the location of the gas could not be specified as either arterial or venous (case 3).

Three typical cases are described.

Case descriptions

Case 1

An 11-day-old neonate was found not breathing in the morning. He had been diagnosed as having a heart murmur at birth. After being transferred to our ER by ambulance in a CPA state, he did not respond to CPR, and death was confirmed 60 min after arrival. To determine the cause of death, PMCT was performed. PMCT of the brain showed arterial GE (Fig. 1a). PMCT of the thorax showed cardiovascular gas in the right atrium, bilateral ventricles, and ascending aorta (Fig. 1b). Autopsy revealed multiple heart anomalies (atrial septal defect, ventricular septal defect, patent ductus arteriosus) and severe pulmonary edema. Autopsy also revealed gas bubbles in the leptomeningeal vessels and in the bilateral anterior, middle, and posterior cerebral arteries. The cause of death was diagnosed as acute circulatory failure.

Case 2

An 81-year-old woman was found unconscious. She had been undergoing dialysis for the previous 3 months. After being transferred to our ER by ambulance in a CPA state, she did not respond to CPR, and death was confirmed 30 min after arrival. Although no autopsy was performed, the cause of death was diagnosed as acute heart failure based on her medical history and clinical course. PMCT was performed to exclude definitively other possible causes. PMCT of the brain showed venous GE (Fig. 2a). PMCT of the thorax showed cardiovascular gas in the left brachiocephalic vein (Fig. 2b).

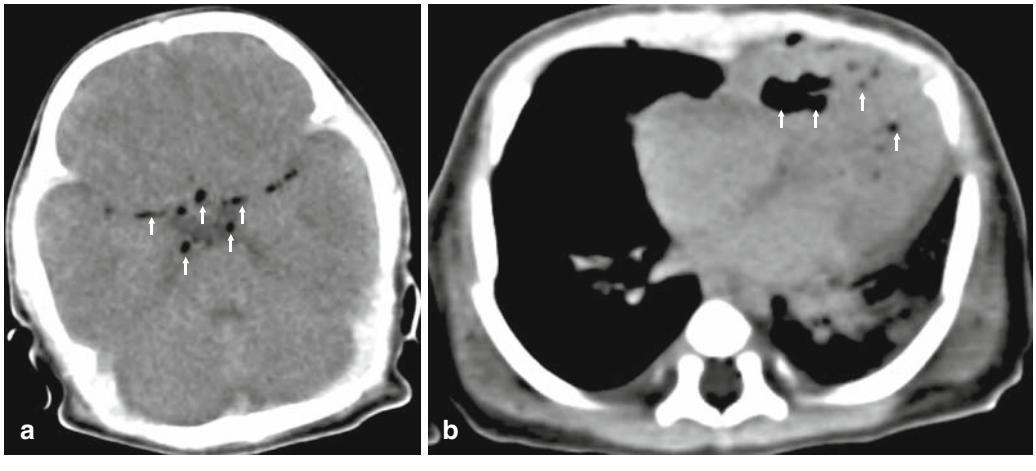

Fig. 1. **a** Postmortem computed tomography (PMCT) of the brain shows gas bubbles in the proximal parts of the bilateral anterior, middle, and posterior cerebral arteries (*arrows*). **b** PMCT of the thorax shows gas bubbles or retention in bilateral ventricles (*arrows*)

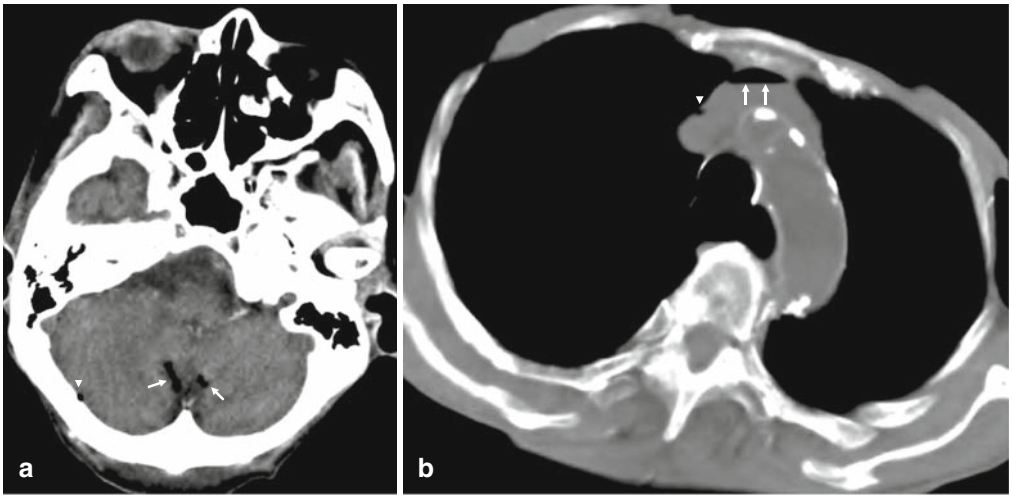

Fig. 2. **a** PMCT of the brain shows linear-shaped gas in the dorsal cerebellar vermis (*arrows*) and spotty gas along the surface of the right cerebellar hemisphere (*arrowhead*). **b** PMCT of the thorax shows a thin gas layer in the left brachiocephalic vein (*arrows*) and spotty gas in the superior vena cava (*arrowhead*)

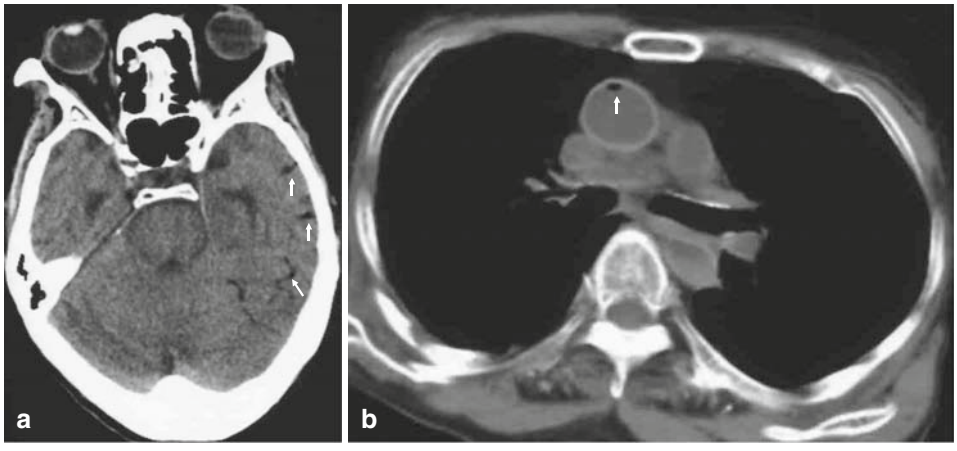

Fig. 3. **a** PMCT of the brain shows curvilinear gas along the surface of the left temporal lobe (*arrows*). **b** PMCT of the thorax shows spotty gas in the ascending aorta (*arrow*)

Case 3

An 84-year-old woman committed suicide by ingesting hypnotic drugs. After being transferred to our ER by ambulance in a CPA state, she did not respond to CPR, and death was confirmed 30 min after arrival. PMCT was performed to exclude definitively other possible causes. PMCT of the brain showed curvilinear GE along the surface of the left temporal lobe (Fig. 3a), but whether it was arterial or venous GE could not be determined. PMCT of the thorax showed cardiovascular gas in the bilateral jugular veins, right atrium, bilateral ventricles, and ascending aorta (Fig. 3b). No autopsy was performed.

Discussion

As causes of cerebral GE, which sometimes is shown by PMCT in nontraumatic death cases, the possible effects of CPR or putrefaction must be taken into consideration. In our study, the possibility of putrefaction was negated because PMCT was performed within 2 h of the confirmation of death. However, we found no statistically significant difference regarding the incidence of cerebral GE between the patients who underwent CPR and those who did not, although we did find a significant difference in the incidence of cardiovascular gas between the same two groups. This discrepancy is attributable to a low incidence of cerebral GE (7.5%) in subjects who underwent CPR, whereas that of cardiovascular gas was 71%.[19]

The cerebral arterial GE detected by nontraumatic PMCT of ER patients arriving in a CPA state is believed to be caused by the mechanical forces of CPR, such as positive-pressure ventilation and chest compression causing pulmonary barotrauma (rupturing of pulmonary vessels in conjunction with parenchymal destruction of the lung), which permits air to enter the pulmonary vein, reach the systemic circulation, and thus the intra-

cerebral arteries.[26–33] In our study, three subjects with cerebral arterial GE were pediatric cases. Arterial GE in pediatric cases can be theoretically explained as paradoxical embolism.[40] In contrast, in a study on nontraumatic PMCT, cardiovascular gas was seen in the right atrium of 36% of the subjects who underwent CPR and in the right ventricle in 41% of subjects,[19] suggesting that the gas was transported into the systemic circulation through the left atrium and ventricle by way of a right-to-left shunt such as an atrial and/or a ventricular septal defect.

Cerebral venous GE on nontraumatic PMCT can be explained by the thoracic pump theory.[34] In a study of nontraumatic PMCT, cardiovascular gas was detected in the brachiocephalic vein and superior vena cava of 45% of patients who underwent CPR.[19] The gas bubbles entering these veins are able to ascend retrogradely to the brain against the blood flow.[41] In patients for whom CPR is unsuccessful, antegrade venous flow does not occur and the gas is not carried away from the intracranial veins.

Rubinstein, et al.[38,39] reported that the distribution of cerebral GE varied among living individuals, and the causes were thought to include the time between the manipulation of intravenous lines and the CT scans, and the anatomy and position of the patient. In our study, venous GE was seen in the infratentorial region in all subjects; that is, its distribution was limited. We attributed this to the fact that the timing of PMCT was relatively constant, being within 2 h after confirmation of death, with the body remaining motionless in a supine position. Also, the diameter of the vessel is greatest from the sigmoid sinus to the transverse sinus, in the passageway from the internal jugular vein to the veins of the head.[42]

In two subjects who showed cerebral arterial GE on PMCT and who underwent an autopsy, gas bubbles in the vessels of the brain were found by autopsy, and the amount of gas was more than that shown on PMCT. Gas bubbles in the vessels of the brain are often detected at autopsy as an artifact. The causes include removal of the roof of the skull, which tears the meninges,[21] and dissection of the vessels near the medula oblongata to remove the brain from the skull. Postmortem imaging using CT or MRI is superior to autopsy for detecting gas in blood vessels and potential body cavities without altering the distribution of the gas.[43,44]

Nontraumatic PMCT findings are classified into three categories: (1) cause of death (e.g., subarachnoid hemorrhage and neurogenic pulmonary edema,[37] aortic dissection, cardiac tamponade[45]); (2) postmortem changes (e.g., hypostasis,[46] hyperattenuating aortic wall,[47] dilatation of the right heart[48]); and (3) changes that occur after CPR (e.g., cardiovascular gas,[19] gastrointestinal distention, hepatic portal venous gas[20]). In general, cerebral GE can be fatal; however, in our subjects, cerebral GE did not appear to be a direct cause of death. Autopsy was performed on two of three pediatric subjects who had arterial GE, and the cause of death was determined in each case (acute circulatory failure, congestive heart failure, and pneumonia). In the other subject who did not undergo autopsy, the cause of death was diagnosed as pneumonia based on a comprehensive understanding of the patient's present illness, blood count, and PMCT findings. Although 25 subjects showed a small amount of venous GE, such a small amount of GE is considered asymptomatic even in living individuals.[38,39]

The present study lacked neck region scans. Therefore, we could not define the relation between cerebral GE and vascular gas in the jugular vein. In a future study, we are planning to scan the neck regions of nontraumatically deceased patients using PMCT.

Conclusion

Cerebral arterial/venous GE was found in subjects who underwent CPR. Our study showed cerebral GE on PMCT in 29 subjects (7.5%), with venous GE more frequent than arterial GE. Furthermore, as the cause of the arterial GE observed in our pediatric cases, we suggest that cardiovascular gas caused by CPR had been transported into the systemic circulation by way of a right-to-left shunt.

Acknowledgments. This work was supported by a grant of the Public Trust-Foundation of Marumo ER Medicine & Research Institute. We thank Ms. Yumiko Moriyama for her help in manuscript preparation.

References

1. Brogdon BG. Research and applications of the new modalities. In: Brogdon BG, editor. Forensic radiology. 1st edn. Boca Raton: CRC Press; 1998. p. 333–8.
2. Swift B, Rutty GN. Recent advances in postmortem forensic radiology: computed tomography and magnetic resonance imaging applications. In: Tsokos M, editor. Forensic pathology reviews. 1st edn. Totowa: Humana; 2006. p. 355–404.
3. Uchigasaki S. Postmortem ultrasound imaging in forensic pathology. In: Tsokos M, editor. Forensic pathology reviews. 1st edn. Totowa: Humana; 2006 p. 405–12.
4. Dirnhofer R, Jackowski C, Vock P, Potter K, Thali MJ. Virtopsy: minimally invasive, imaging-guided virtual autopsy. Radiographics 2006;26:1305–33.
5. Hayakawa M, Yamamoto S, Motani H, Yajima D, Sato Y, Iwase H. Does imaging technology overcome problems of conventional postmortem examination? A trial of computed

tomography imaging for postmortem examination. Int J Legal Med 2006;120:24–6.
6. Oyake Y, Aoki T, Shiotani S, Kohno M, Ohashi N, Akutsu H, et al. Postmortem computed tomography for detecting causes of sudden death in infants and children: retrospective review of cases. Radiat Med 2006;24:493–502.
7. Chew FS, Relyea-Chew A, Ochoa ER Jr. Postmortem computed tomography of cadavers embalmed for use in teaching gross anatomy. J Comput Assist Tomogr 2006;30:949–54.
8. Ljung P, Winskog C, Persson A, Lundstrom C, Ynnerman A. Full body virtual autopsies using a state-of-the-art volume rendering pipeline. IEEE Trans Vis Comput Graph 2006;12: 869–76.
9. Poulsen K, Simonsen J. Computed tomography as routine in connection with medico-legal autopsies. Forensic Sci Int 2007;171:190–7.
10. Levy AD, Harcke HT, Getz JM, Mallak CT, Caruso JL, Pearse L, et al. Virtual autopsy: two- and three-dimensional multidetector CT findings in drowning with autopsy comparison. Radiology 2007;243:862–8.
11. O'Donnell C, Rotman A, Collett S, Woodford N. Current status of routine post-mortem CT in Melbourne, Australia. Forensic Sci Med Pathol 2008;3:226–32.
12. Shiotani S, Shiigai M, Ueno Y, Sakamoto N, Atake S, Kohno M, et al. Postmortem computed tomography findings as evidence of traffic accident-related fatal injury. Radiat Med 2008;26:253–60.
13. Weustink AC, Hunink MGM, van Dijke CF, Renken NS, Krestin GP, Oosterhuis JW. Minimally invasive autopsy: an alternative to conventional autopsy? Radiology 2009;250: 897–904.
14. Sakamoto N, Ohashi N, Hamabe Y, Kohno M, Shiotani S, Hayakawa H, et al. Answers to questionnaire regarding current status and future subjects of postmortem imaging in Japanese emergency center hospitals. Kyukyuigaku (Japanese Journal of Acute Medicine) 2009;33:985–9 (in Japanese).
15. Sugawara S, Mizunuma K, Kato K, Toshiyasu T. Evaluation of postmortem CT (PMCT) to diagnose the cause of death. Rinsho Hoshasen (Japanese Journal of Clinical Radiology) 2006;5:845–50 (in Japanese with English abstract).
16. Otomi Y, Kanetada K, Mukaijo T, Kourai F. Clinical experience of CT scannings to 107 postmortem examinations of CPAOA cases. Rinshogazou (Clinical Imagiology) 2008;24: 514–8 (in Japanese).
17. Sugimura H, Yano M, Takechi Y, Kawasaki Y, Tanaka T, Muranaka T, et al. Usefulness of postmortem computed tomography to investigate the cause of death in 135 cases of cardiopulmonary arrest on arrival. Kyukyuigaku (Japanese Journal of Acute Medicine) 2008;32:861–4 (in Japanese).
18. Takahashi N, Higuchi T, Shiotani M, Maeda H, Hirose Y, Iinuma Y, et al. Postmortem computed tomography in 360 deceased individuals: postmortem findings and influence of cardiopulmonary resuscitation. Rinsho Hoshasen (Japanese Journal of Clinical Radiology) 2008;53:1840–5 (in Japanese with English abstract).
19. Shiotani S, Kohno M, Ohashi N, Atake S, Yamazaki K, Nakayama H, et al. Cardiovascular gas on non-traumatic postmortem computed tomography (PMCT): the influence of cardiopulmonary resuscitation. Radiat Med 2005;23:225–9.
20. Shiotani S, Kohno M, Ohashi N, Yamazaki K, Nakayama H, Watanabe K. Postmortem computed tomographic (PMCT) demonstration of the relation between gastrointestinal (GI) distension and hepatic portal venous gas (HPVG). Radiat Med 2004;22:25–9.
21. Krants P, Holtas S. Postmortem computed tomography in a diving fatality. J Comput Assist Tomogr 1983;7:132–4.
22. Haydon JR, Williamson JA, Ansford AJ, Sherif S, Shapter MJ. A scuba-diving fatality. Med J Aust 1985;143:458–62.
23. Ozdoba C, Weis J, Platter T, Dirnhofer R, Yen K. Fatal scuba diving incident with massive gas embolism in cerebral and spinal arteries. Neuroradiology 2005;47:411–6.
24. Jensen ME, Lipper MH. CT in iatrogenic cerebral air embolism. AJNR Am J Neuroradiol 1986;7:823–7.
25. Kodama F, Ogawa T, Hashimoto M, Tanabe Y, Suto Y, Kato T. Fatal air embolism as a complication of CT-guided needle biopsy of the lung. J Comput Assist Tomogr 1999;23: 949–51.
26. Yamaki T, Ando S, Ohta K, Kubota T, Kawasaki K, Hirama M. CT demonstration of massive cerebral air embolism from pulmonary barotraumas due to cardiopulmonary resuscitation. J Comput Assist Tomogr 1989;13:313–5.
27. Shiina G, Shimosegawa Y, Kameyama M, Onuma T. Massive cerebral air embolism following cardiopulmonary resuscitation: report of two cases. Acta Neurochir (Wien) 1993;125: 181–3.
28. Iwama T, Andoh H, Murase S, Miwa Y, Ohkuma A. Diffuse cerebral air embolism following trauma: striking postmortem CT findings. Neuroradiology 1994;36:33–4.
29. Hashimoto Y, Yamaki T, Sakakibara T, Matsui J, Matui M. Cerebral air embolism caused by cardiopulmonary resuscitation after cardiopulmonary arrest on arrival. J Trauma 2000; 48:975–7.
30. Akaishi K, Hongo K, Obinata C, Kobayashi S. Pneumoangiogram in a patient with severe head injury: case illustration. J Neurosurg 2000;92:502.
31. Sakai I, Nishizawa S. Cerebral air embolism after lung contusion: case illustration. J Neurosurg 2001;95:909.
32. Ugurel S, Kocaoglu M, Saglam M, Ucoz T, Somuncu I. CT pneumoangiogram sign following cardiopulmonary resuscitation: detrimental cerebral air embolism or postmortem blood replacement with air? Eur J Radiol Extra 2003;45:114–7.
33. Hwang SL, Lieu AS, Lin CL, Liu GC, Howng SL, Kuo TH. Massive cerebral air embolism after cardiopulmonary resuscitation. J Clin Neurosci 2005;12:468–9.
34. Imanishi M, Nishimura A, Tabuse H, Miyamoto S, Sakaki T, Iwasaki S. Intracranial gas on CT after cardiopulmonary resuscitation: 4 cases. Neuroradiology 1998;40:154–7.
35. Sharma MR, Newell DW, Grant GA. Diffuse cerebral venous air embolism following subarachnoid hemorrhage: case illustration. J Neurosurg 2003;98:1320.
36. Shiotani S, Yamazaki K, Kikuchi K, Nagata C, Morimoto T, Noguchi Y, et al. Postmortem magnetic resonance imaging (PMMRI) demonstration of reversible injury phase myocardium in a case of sudden death from acute coronary plaque change. Radiat Med 2005;23:563–5.
37. Shiotani S, Kohno M, Ohashi N, Yamazaki K, Nakayama H, Watanabe K, et al. Non-traumatic postmortem computed tomographic (PMCT) findings of the lung. Forensic Sci Int 2004;139:39–48.
38. Rubinstein D, Symonds D. Gas in the cavernous sinus. Am J Neuroradiol 1994;15:561–6.
39. Rubinstein D, Dangleis K, Damiano TR. Venous air emboli identified on head and neck CT scans. J Comput Assist Tomogr 1996;20:559–62.
40. Muth CM, Shank ES. Gas embolism. N Engl J Med 2000;342: 476–82.
41. Schlimp CJ, Loimer T, Rieger M, Lederer W, Schmidts MB. The potential of venous air embolism ascending retrograde to the brain. J Forensic Sci 2005;50:906–9.
42. Pernkopf E. The head. In: Ferner H, editor. Atlas of topographical and applied human anatomy. 2nd revised edn. Munich: Urban & Schwarzenberg; Baltimore; 1980. p. 1–135.

43. Ros PR, Li KC, Vo P, Baer H, Staab EV. Preautopsy magnetic resonance imaging: initial experience. Magn Reson Imaging 1990;8:303–8.
44. Donchin Y, Rivkind AI, Bar-Ziv J, Hiss J, Almong J, Drescher M. Utility of postmortem computed tomography in trauma victims. J Trauma 1994;37:552–6.
45. Shiotani S, Watanabe K, Kohno M, Ohashi N, Yamazaki K, Nakayama H. Postmortem computed tomographic (PMCT) findings of pericardial effusion due to acute aortic dissection. Radiat Med 2004;22:405–7.
46. Shiotani S, Kohno M, Ohashi N, Yamazaki K, Itai Y. Postmortem intravascular high density fluid level (hypostasis): CT findings. J Comput Assist Tomogr 2002;26:892–3.
47. Shiotani S, Kohno M, Ohashi N, Yamazaki K, Nakayama H, Ito Y, et al. Hyperattenuating aortic wall on postmortem computed tomography (PMCT). Radiat Med 2002;20:201–6.
48. Shiotani S, Kohno M, Ohashi N, Yamazaki K, Nakayama H, Watanabe K, et al. Dilatation of the heart on postmortem computed tomography (PMCT): comparison with live CT. Radiat Med 2003;21:29–35.