



Research Article

Conservative Treatment of Acute Intracerebral Hemorrhage Occurred with Complicated Type B Aortic Dissection: Case Report and Literature Review

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Abstract

The concurrence of acute intracerebral hemorrhage and complicated acute aortic dissection is destructive and extremely rare. Surgical treatment poses a high risk to patients with this type comorbidity; however, a medical treatment could be an alternative in acute phase. We described a case of a 63-year-old male with hypertension who emergently presented at our institution with left-sided limb weakness. A head computed tomography (CT) scan showed 30mL of superficial intracerebral hemorrhage located in the right frontal-parietal lobe. On the third day, painless aortic dissection was found by accident in a routine chest CT examination. CT angiography confirmed the acute type B aortic dissection complicated with malperfusion syndrome of left renal. By conservative means, we controlled the systolic blood pressure varying from 100 to 120 mmHg persistently and smoothly for about a month. Until intracranial hematomas were mostly absorbed, a delayed thoracic endovascular aortic repair was performed to restore renal perfusion. Aggressive blood pressure lowering therapy would be important for improving long-term outcome. To our knowledge, this is the first report of the conservative treatment of acute intracerebral hemorrhage occurred simultaneously with complicated type B acute aortic dissection.

Key Words: Intracerebral hemorrhage; Aortic dissection; Blood pressure control; Delayed endovascular intervention.

Introduction

Intracerebral hemorrhage complicated acute aortic dissection is so catastrophic that most patients died before timely diagnosis, let alone prompt management (1). Neurological deficits

caused by intracerebral hemorrhage could notably dominate the clinical picture, masking the underlying condition of aortic dissection. In addition, there is absence of sufficient data between therapeutic approaches and long-term outcomes for acute aortic dissection (2).

Meanwhile, the concurrence of acute intracerebral hemorrhage and acute aortic dissection presents a high challenge for selection of treatment which continues to be critical to survival.

Case Presentation

A 63-year-old male with hypertension, was sent to the emergency department with a sudden onset of left-sided limb weakness approximately 3 hours prior to admission. The patient denied headache, chest pain, shoulder-back pain, and abdomen pain, and was non-fluent aphasia, but eager to show signs of left-sided weakness of lower facial muscles, hemiplegia and hemiparesis on the left side. Examination revealed blood pressure (BP) of 179/80 mmHg. The Glasgow Coma Score was 15. Head CT without contrast revealed an acute intracerebral hemorrhage in right frontal-parietal lobe (≤ 1 cm from the cortical surface of the brain) with a volume of 30 mL, a reduction in the size of the right lateral ventricle, and shallow sulci (Fig. 1 A). Until the third day of admission, thoracic aortic dissection was found by accident in a routine chest CT examination. Subsequently, emergent contrast-enhanced CT angiography (CTA) confirmed the complicated acute type B aortic dissection starting from the distal end to the origin of the left subclavian artery with involvement of left renal artery branch (Fig. 1 C) and malperfusion syndrome of renal (Fig. 1 D). No intracranial aneurysms or arteriovenous malformations were presented. Pertinent laboratory findings revealed normal functions of organs including renal function.

The patient was then immediately consulted by a neurosurgeon and cardiovascular surgeon. Specialists decided not to perform surgical intervention based on the normal midline structure of cerebrum and the thrombosed arterial lumen of aorta on CT images. By monitoring the vital signs strictly, the patient received 100 mL of 20% mannitol every 6 hours in the first week to prevent

perihematoma oedema which then tapered in the next week.

During first two days, we lowered the patient BP to around 130/80 mmHg (Fig. 1 F) with sodium nitroprusside by continuous intravenous infusion. Subsequently, intravenous antihypertensive agents were tapered till the systolic BP between 100 and 120 mmHg (Fig. 1 F) by oral antihypertensive agents including 95 mg of metoprolol every 12 hours, and 8 mg of benidipine every 24 hours. After 4 weeks of continuous treatment mentioned above, CT showed the cerebral hematoma was absorbed without ischemic nidus (Fig. 1 B). Equally, complicated type B aortic dissection on the patient stayed in the same condition as initially reported. On the 30th day of hospitalization, a delayed endovascular intervention for complicated aortic dissection was conducted successful. After 1 month, a repeated CTA scan of the thoracic aorta showed that left renal perfusion has recovered completely (Fig. 1 E). Following up for 6 months, the patient could take care of himself and do some activities of daily living with the aid of walking tools.

Discussion

Concurrence of intracerebral hemorrhage and acute aortic dissection raises a therapeutic dilemma whether surgery is essential, and which one fatal disease should be targeted in the first place. A search of PubMed with the terms “aortic dissection” and “brain hemorrhage or intracerebral hemorrhage or subarachnoid hemorrhage” produced only 6 cases reported (Table 1). 3 cases (3, 4 and 7) were treated successfully by emergent surgery for coexistence of intracerebral hemorrhage and acute aortic dissection. In 2 cases (3 and 7), surgical operations for the aneurysm of the cerebral artery were achieved successfully by the means of open surgical clipping or endovascular aneurysm obliteration, while medical treatment was

managed for aortic dissection. The prevention of aneurysm rupture was the first goal of treatment for subarachnoid hemorrhage. However, surgical treatment strategies varied as the specific status of patients changed. When traumatic subarachnoid hemorrhage involved non-aneurysm and no vascular abnormality, an emergency distal arch replacement for traumatic type A aortic dissection was performed with extracorporeal circulation on a patient with cardiac hemodynamic alter (4). Contrary to the above favorable prognosis, the patients with hernia cerebri or dissection extending into neck vessels were less likely to survive even with promptly medical assistance (5, 6).

To best of our knowledge, there has been no successful conservative treatment for simultaneous occurrence of complicated type B aortic dissection and intracerebral hemorrhage.

Selection of conservative treatment is a balance of risks and benefits. First and foremost, if aneurysm or arteriovenous malformation was not apparent, the incidence of cerebral hematoma enlargement with controlled BP would be low. Moreover, conservative treatment did not decrease the rate of death or disability at 6th month compared with early surgery (8), particularly for conscious patients with superficial lobar cerebral hemorrhage without intraventricular hemorrhage (9). Meanwhile, surgical operation of hematoma evacuation might incur neural damage and increase the risks of rupture and extension of dissection. On the other hand, thoracic endovascular aortic repair was widely applied to complicated cases and suitable anatomy (10). But usage of anticoagulants, such as heparin during stent implantation, would increase risk of residual disability in patients with acute intracerebral hemorrhage (11).

As for conservative management, the goals for BP would be responsible for prognosis of both cerebral hemorrhage and aortic dissection with renal ischemia. In sub-analysis of international clinical trial (INTERACT2) (12), to maintain BP

below 140 mmHg smoothly for 7 days was beneficial for early recovery of neurological function, but there was absence of more searches between long-term monitoring of BP and outcome. Remarkably, overaggressive treatment of BP especially below 120 mmHg in intracerebral hemorrhage might decrease perfusion of tissue or organ, predictably inducing cerebral ischemia (13, 14) and cardio-renal adverse events (15). With beta-blocking agents initiated first to counteract reflex tachycardia, lowering systolic BP (systolic blood pressure between 100 and 120 mmHg) was highly recommended for acute aortic dissection (16), which aimed at decreasing wall stress in order to limit extension of the dissection (17). We adopted this BP reduction standard and regularly evaluated patient's hemodynamic change, consciousness status, and peripheral vascular change. Eventually, the patient's condition remained stable without renal ischemia progress and worse brain injury, to prove out the systolic BP goal for this case.

Complicated type B aortic dissection refers to malperfusion syndrome, such as branch-vessel involvement resulting in end-organ ischemia, aneurysm, intractable symptoms (pain), or uncontrolled hypertension (18). Once detected, complicated acute type B aortic dissection was supposed to be repaired as soon as possible. Studies had demonstrated that the less invasive nature of endovascular repair decreased the in-hospital mortality risk of type B aortic dissection in chronic period (19), although appropriate time of endovascular repair in type B dissections remained unknown. Akin et al. speculated that the more stable clinical status of the patients, the better survival following endovascular repair (20). Our case had confirmed that intervention was delayed treating acute type B aortic dissection complicated with intracerebral hemorrhage when patients' condition permitted. An interesting finding from a cohort study was that endovascular intervention for type B aortic dissection repaired renal ischemia, in return subclinical renal

malperfusion revascularization contributed significantly to long-term BP control (21). Presumably, renal hypoperfusion could aggravate the proceeding of hypertension through nervous-humoral regulation and direct or indirect effects of vasoactive substances. Physiological adjustment mechanism was attenuated theoretically by the recovery of renal blood flow to improve BP control.

Conclusion

The concurrence of acute intracerebral hemorrhage and complicated acute aortic dissection is fatal and extremely rare. Emergent surgery poses a high risk to patients with this type

comorbidity; therefore, a conservative management could be an alternative in acute phase. Firstly, with review of the literature, we report the first case to introduce a conservative treatment for medium intracerebral hemorrhage simultaneous with complicated type B acute aortic dissection. Secondly, in patients with life-threatening medical comorbidity, non-operative treatment with early mobilization can yield acceptable results. Furthermore, this study has revealed that aggressive blood pressure lowering therapy below 120 mmHg had proved reasonable and recommended a delay endovascular repair for complicated type B aortic dissection in a more stable clinical status of the patient to improve long-term blood pressure control.

Table 1. Reported cases of acute intracerebral hemorrhage occurred with aortic dissection

Author/year	Initial symptoms	Subtype of acute aortic dissection/treatments	CNS hemorrhagic location/treatments	Available outcome
Garrard, P. (1) 1996	Neck pain	Atypical ATAAD (found Postmortem examination)	left frontal lobe and left cerebellar hemisphere hemorrhage/Conservative management	In-hospital death on the 2nd day
Inaba, S. (3) 2005	Not available	ATBAD/Conservative management	SAH/Intracranial aneurysmal clipped operation	Uneventful Perioperative course.
Ishikawa, H. (4) 2009	Slightly impaired consciousness, cardiogenic shock	Traumatic ATBAD with mediastinal hemorrhage and pericardial effusion /Emergency distal arch replacement	Traumatic SAH/Conservative management	Condition improved and discharged after 36 days of treatment.
Sonoo, T. (5) 2013	Disturbance of consciousness and vomiting	ATAAD with pericardial effusion/Conservative management	Putamen hemorrhage /Emergency decompressive craniectomy and hematoma evacuation	In-hospital death on the 3rd day

Lingqiu, K. (6) 2015	Loss of consciousness	ATAAD With extension to Bilateral common carotid arteries/Conservative management	Thalamus hemorrhage/Conservative management	In-hospital death on the 3rd day
Inamasu, J. (7) 2016	Severe headache	Painless ATAAD with extension to Bilateral common carotid arteries/conservative management	SAH/Intracranial aneurysm embolization	Follow-up for 90 days, aortic dissection without progression

CNS, central nervous system; SAH, subarachnoid hemorrhage; ATBAD, acute type B aortic dissection; ATAAD, acute type A aortic dissection.

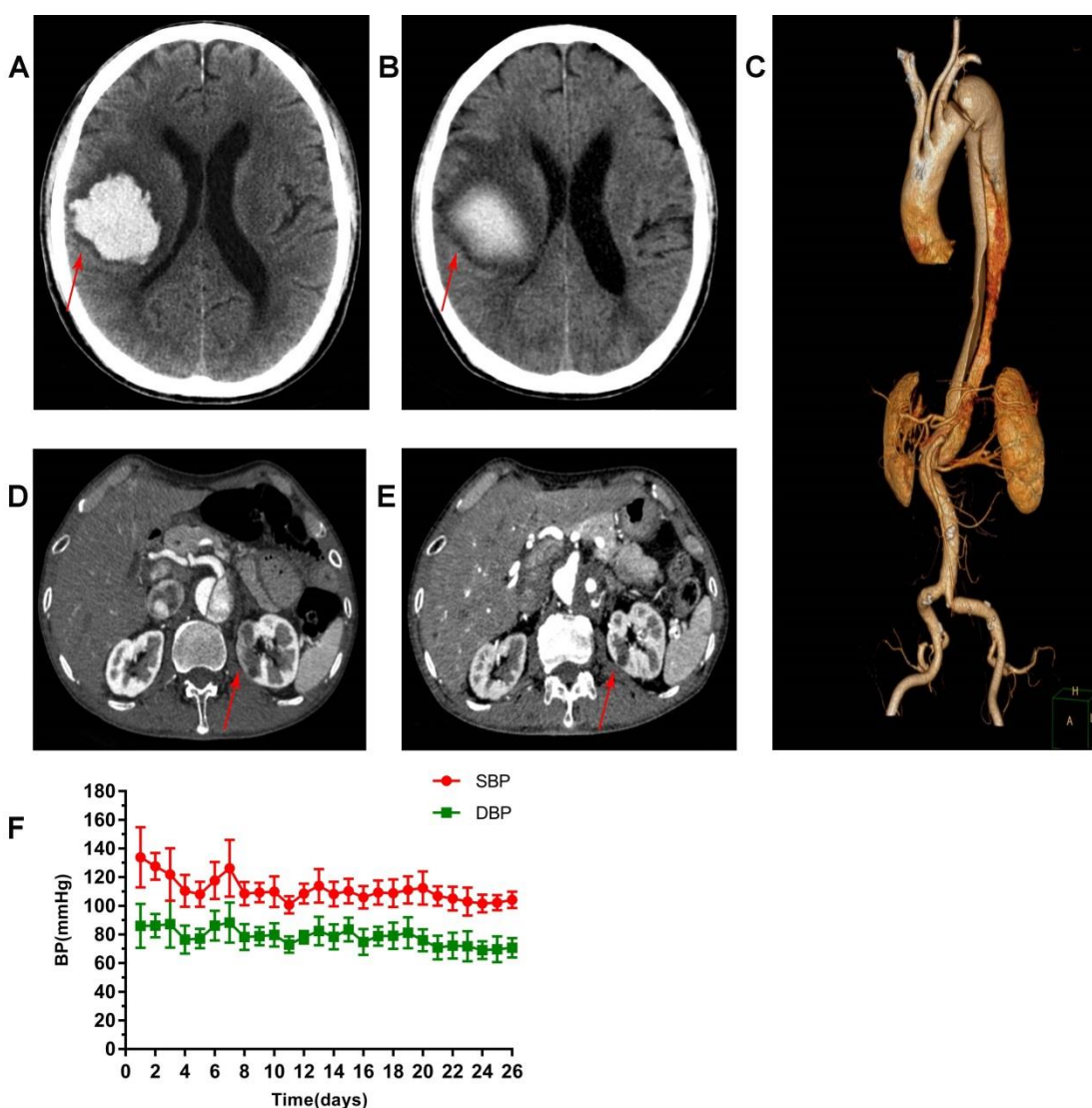


Fig. 1. The conservative treatment of acute intracerebral hemorrhage occurred simultaneously with complicated type B acute aortic dissection.

(A) Computed tomographic (CT) on admission showing the high-density hemorrhage lesion in right frontal and parietal lobe (red arrow).

(B) Follow-up head CT at 4 weeks showing that cerebral hematoma was mostly absorbed (red arrow) without ischemic nidus.

(C) Three-dimensional reconstruction of contrast enhanced CT angiography images of the acute type B aortic dissection.

(D) Contrast-enhanced CT angiography showing aortic dissection complicated with malperfusion syndrome of left renal (red arrow).

(E) Renal malperfusion revascularization (red arrow) after delayed thoracic endovascular aortic repair.

(F) Statistical graph on daily mean blood pressure and variability (a series of blood pressure every 1 hour for 26 days) showing: total systolic blood pressure at 111 ± 9 mmHg; total diastolic blood pressure at 78 ± 8 mmHg. BP, blood pressure; SBP, systolic blood pressure; DBP, diastolic blood pressure.

Declarations

1) *Consent to publication*

We declare that all authors agreed to publish the manuscript at this journal based on the signed Copyright Transfer Agreement and followed publication ethics.

2) *Ethical approval and consent to participants*

Not involved.

3) *Disclosure of conflict of interests*

We declare that no conflict of interest exists.

4) *Funding*

None

5) *Availability of data and material*

We declare that the data supporting the results reported in the article are available in the published article.

6) *Authors' Contributions*

Authors contributed to this paper with original ideas (JL), literature search (JL), manuscript writing (JL), revision (JL and DKG), editing (JL and DKG) and final approval (DKG).

7) *Acknowledgement*

None

8) *Authors' biography*

None

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