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CASE REPORT

Anesthesia Management in Aneurysm Clipping Surgery with Chilaiditi Sign : a Case Report

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Abstract

Chilaiditi sign is the asymptomatic interposition of the colon between the liver and diaphragm; when accompanied by gastrointestinal symptoms, it is termed Chilaiditi syndrome. Its coexistence with aneurysmal subarachnoid haemorrhage creates competing anaesthetic priorities, including aspiration prevention, lung protection, and control of cerebral haemodynamics. We report a 79-year-old woman who presented with sudden severe headache and transient loss of consciousness. Imaging demonstrated subarachnoid haemorrhage from a ruptured left posterior communicating artery aneurysm, together with incidental colonic interposition beneath an elevated right hemidiaphragm, bilateral pleural effusions, and partial right-lower-lobe collapse. After 10 hours of fasting and pharmacological aspiration-risk mitigation, general anaesthesia was induced with thiopental, remifentanil, and rocuronium using a modified rapid-sequence approach that included preoxygenation, cricoid pressure, gentle mask ventilation, and video laryngoscopy. Pressure-controlled ventilation and close haemodynamic control were maintained throughout aneurysm clipping. No intraoperative rupture, aspiration, haemodynamic instability, or respiratory deterioration occurred. The patient was extubated 36 hours after surgery and discharged on postoperative day 8 with a modified Rankin Scale score of 1. This case highlights the need to individualise induction and ventilation when Chilaiditi sign coexists with urgent intracranial surgery. The uneventful outcome of a single case should not be interpreted as evidence that modified induction is generally safer than classical rapid-sequence induction.

Keywords: Chilaiditi syndrome; Intracranial aneurysm; Subarachnoid hemorrhage; Anesthesia, general; Craniotomy

Introduction

Chilaiditi sign is the asymptomatic radiographic interposition of the colon or, less commonly, small intestine between the liver and right hemidiaphragm. When this finding is accompanied by gastrointestinal symptoms, it is termed Chilaiditi syndrome. The reported prevalence is 0.025-0.28%, with a predominance among older adults.[1] Although usually incidental, cephalad displacement of the diaphragm can reduce functional residual capacity and impair ventilation, particularly when increased intra-abdominal pressure or positive-pressure ventilation causes further diaphragmatic displacement. Interposed bowel may also raise concern about regurgitation during induction, although evidence quantifying aspiration risk is limited.[2] Patients with coexisting pulmonary or pleural abnormalities may have even less and controlled ventilation particularly important.[3]

When Chilaiditi sign coexists with aneurysmal subarachnoid haemorrhage (aSAH), the anaesthetic plan must reconcile competing priorities. The aneurysm should be secured promptly to reduce rebleeding, while anaesthetic management aims to maintain cerebral perfusion, limit changes in transmural pressure, provide brain relaxation, and avoid hypoxaemia and hypercapnia. Airway instrumentation can provoke sympathetic stimulation and abrupt haemodynamic changes, whereas strategies used to reduce aspiration risk may themselves influence oxygenation, gastric insufflation, and intracranial pressure. Evidence specific to neuroanaesthesia in patients with this anatomical variant is limited to isolated reports; decisions must therefore rely largely on general neuroanaesthetic and aspiration-risk principles.[4] This report describes the perioperative management of an older woman with a ruptured posterior communicating artery (PCom) aneurysm and incidental Chilaiditi sign who underwent emergency craniotomy and clipping. The case highlights how aspiration precautions, lung-protective ventilation, and cerebral haemodynamic control were balanced in an individualised modified rapid-sequence approach

Case Presentation

A 79-year-old woman (60 kg, 155 cm) presented five hours after a fall with head impact, followed by sudden severe headache and about five minutes of loss of consciousness. Her last meal was consumed six hours before the pre-anaesthetic evaluation. She reported choking while eating for five years, reduced oral intake for one week, and poorly controlled hypertension. She had no gastrointestinal symptoms, dyspnoea, fever, or seizures. The Glasgow Coma Scale score was E3V5M6 (14), and pain was 7/10. Peripheral oxygen saturation was 93% on room air and 97% with oxygen at 4 L/min through a nasal cannula. The respiratory rate was 22 breaths/min, with bilateral rhonchi; bowel sounds were audible over the right sixth intercostal space. Blood pressure was 157/72 mmHg and heart rate 95 beats/min. Arterial blood gas analysis on face-mask oxygen at 8 L/min showed mild compensated metabolic alkalosis (pH 7.50, $PaCO_2$ 46 mmHg, HCO_3^- 35.9 mmol/L, and base excess +12.7 mmol/L). Other laboratory findings are presented in Table 1

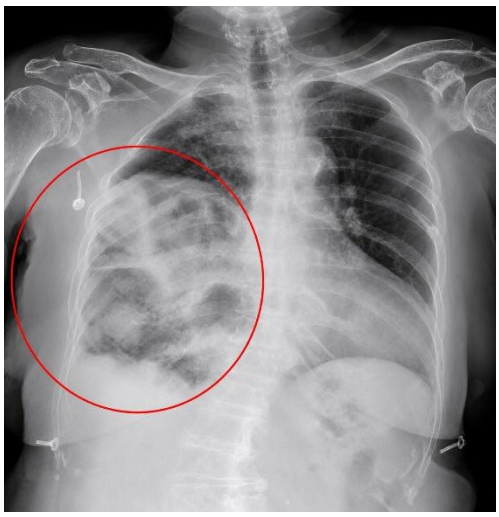

Figure 1. Posteroanterior chest radiograph showing elevation of the right hemidiaphragm with interposed bowel gas, consistent with Chilaiditi sign, together with bilateral pleural effusions and partial right-lower-lobe collapse.

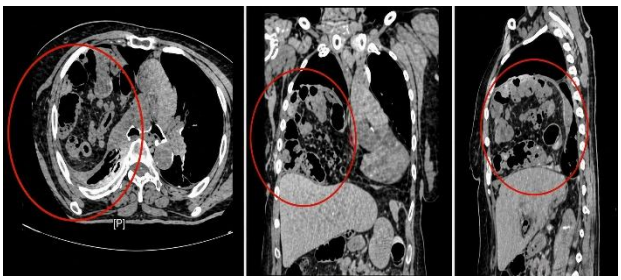

Figure 2. Contrast-enhanced thoracic CT images in axial, coronal, and sagittal planes showing interposition of the hepatic flexure between the liver and right hemidiaphragm (Chilaiditi sign), with cephalad displacement of the right hemidiaphragm

Table 1. Laboratory Findings

Test names	Values	Units
Complete Blood Count		
White Blood Cell	10.04	$\times 10^3/\mu L$
Hemoglobin	11.4	g/dL
Hematocrit	34.5	%
Platelets	267	$\times 10^3/\mu L$
Coagulation Profile		
PT	9.5	s
APTT	23.3	s
International Normalized Ratio (INR)	0.88	–
Clinical Chemistry		
BUN	17	mg/dL
Creatinine	0.65	mg/dL
eGFR	84.4	mL/min/1.73 m^2
AST	38	U/L
ALT	41	U/L
Electrolytes		
Sodium	137	mmol/L
Potassium	3.8	mmol/L
Chloride	103	mmol/L
Glucose	97	mg/dL
Arterial Blood Gas (FM 8 L/min)		
pH	7.5	–
pCO_2	46	mmHg
pO_2	189	mmHg
BEecf	12.7	mmol/L
HCO_3^-	35.9	mmol/L
SaO_2	100	%
TCO_2	37.3	mmol/L

ALT : alanine aminotransferase; APTT : activated partial thromboplastin time; AST : aspartate aminotransferase; BEecf : base excess; BUN: blood urea nitrogen; eGFR : estimated glomerular filtration rate; PT : prothrombin time; SaO_2 : arterial oxygen saturation; TCO_2 : Total carbondioxide

Chest radiography (Figure 1) showed an elevated right hemidiaphragm and bilateral pleural effusions. Thoracic computed tomography (CT; Figure 2) confirmed Chilaiditi sign, with the hepatic flexure in the subphrenic space, leftward mediastinal shift, and partial right-lower-lobe collapse.

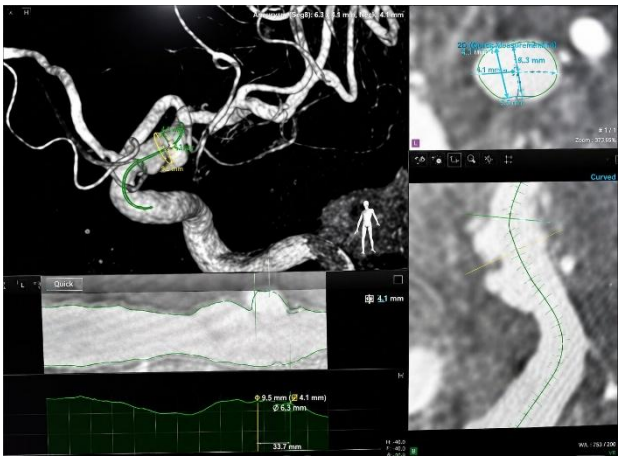

Figure 3. Three-dimensional rotational angiography and multiplanar reconstructions showing a saccular aneurysm at the origin of the left posterior communicating artery.

CT angiography demonstrated intracranial haemorrhage and subacute infarction involving the anterior limb of the internal capsule and left lentiform nucleus. Digital subtraction angiography (Figure 3) showed a saccular left PCom aneurysm, left vertebrobasilar-junction stenosis, and no additional anterior-circulation aneurysm. Severity was World Federation of Neurosurgical Societies grade 2, Hunt and Hess grade 2, and modified Fisher grade 1.

After 10 hours of fasting, premedication comprised midazolam 1 mg, omeprazole 40 mg, and dexamethasone 20 mg. The patient was classified as American Society of Anesthesiologists physical status IIIE. Standard monitoring was supplemented with a right radial arterial line. Preoxygenation used tidal-volume breathing with 100% oxygen at 6 L/min through a face mask. Anaesthesia was induced with thiopental 180 mg (3-5 mg/kg) and remifentanil target-controlled infusion (TCI; Minto model, 5-7 ng/mL). Cricoid pressure and gentle mask ventilation were applied. After rocuronium 40 mg, tracheal intubation was achieved on the first attempt using a video laryngoscope (Cormack-Lehane grade II). This modified rapid-

sequence approach maintained aspiration precautions while attenuating the pressor response to laryngoscopy. A right internal jugular catheter was inserted under ultrasound guidance.

Anaesthesia was maintained with air-oxygen (FiO_2 0.5), pressure-controlled ventilation (tidal volume 350 mL, approximately 7 mL/kg predicted body weight [50.5 kg]; respiratory rate 14 breaths/min; positive end-expiratory pressure 5 cmH_2O; plateau pressure 20 cmH_2O), thiopental infusion (3-4 mg/kg/h; total 600 mg), remifentanil TCI (3-5 ng/mL), and intermittent rocuronium. End-tidal carbon dioxide was 30-35 mmHg and mean arterial pressure 80-90 mmHg, without vasopressors. Ondansetron, tranexamic acid, and 20% mannitol 150 mL (30 g; approximately 0.5 g/kg) were administered before dural opening. A single permanent clip was applied, without temporary clipping or intraoperative rupture. Estimated blood loss was 300 mL, urine output 200 mL, crystalloid administration 1,800 mL, and temperature 36.1°C. Surgery lasted 2 hours 30 minutes.

The patient remained intubated and sedated and was transferred to intensive care. Analgesia comprised fentanyl 400 μg in 50 mL 0.9% saline at 2.1 mL/h and paracetamol 1 g every 8 hours. Nimodipine, started on admission, was continued for 21 days. Deep sedation was maintained for 24 hours; extubation was deferred until she was awake, cooperative, and breathing adequately. She was extubated 36 hours after surgery, stayed in intensive care for 5 days, and was discharged on postoperative day 8 with a modified Rankin Scale score of 1. Follow-up CT was not performed because of clinical improvement. No aspiration, pneumonia, or respiratory or neurological deterioration occurred. Figure 4 summarises the course.

Discussion

This case involved urgent clipping of a ruptured left PCom aneurysm in a patient with incidental Chilaiditi sign, an elevated right hemidiaphragm, bilateral pleural effusions, and partial right-lower-lobe collapse. The principal concerns were reduced pulmonary reserve and possible regurgitation if gastric insufflation or increased intra-abdominal pressure occurred. Chronic choking added uncertainty about aspiration risk despite fasting. The team retained aspiration precautions while avoiding hypoxaemia, hypercapnia, and abrupt haemodynamic changes that could worsen cerebral physiology.[5,6] Fasting was confirmed for 10 hours, but does not guarantee an empty stomach given her choking history, so aspiration precautions were maintained.

The modified rapid-sequence approach allowed controlled preoxygenation, cricoid pressure, low-pressure mask ventilation, and video laryngoscopy. Omeprazole could reduce gastric acidity but could not prevent aspiration.[7] Dexamethasone 20 mg was given as institutional practice intended to reduce cerebral oedema, with incidental antiemetic benefit.[8] Midazolam provided anxiolysis while lowering cerebral blood flow and oxygen consumption.[9] Gentle ventilation preserved oxygenation while minimising airway pressure and gastric insufflation. This approach differs from classical rapid-sequence induction and should be understood as an individualised balance of risks, not a generally preferable technique.

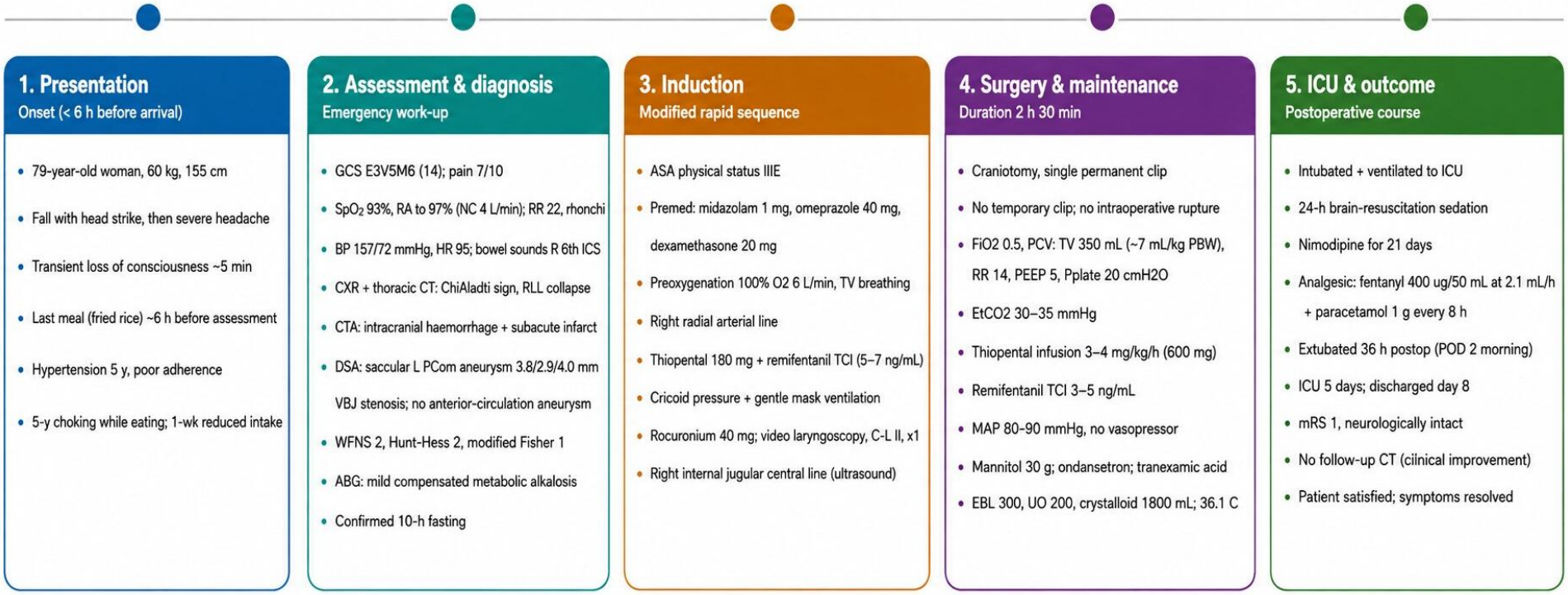

Figure 4. Timeline of the patient's presentation, perioperative management, and postoperative outcome.

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Induction aimed to prevent rupture while preserving cerebral perfusion, avoiding a rise in transmural pressure and blunting the laryngoscopy pressor response.[4] General neuroanaesthetic principles for aneurysm surgery guided agent selection.[10] Thiopental, remifentanil, and rocuronium were selected for rapid hypnosis, attenuation of sympathetic responses, and prompt tracheal intubation. Thiopental reduces cerebral metabolic rate and blood flow, and observational data from unruptured aneurysm clipping have associated its use with fewer neurological complications; those findings cannot be directly extrapolated to ruptured aneurysms.[11] Remifentanil permits rapid titration with limited accumulation, whereas rocuronium provides rapid intubating conditions without clinically relevant histamine release.[12,13]

Classical RSI is usual when aspiration risk is high, but its main hazard here is the pressor response to laryngoscopy, with coughing, hypoxia and hypercapnia raising intracranial and transmural pressure. A modified rapid sequence approach, combining preoxygenation, cricoid pressure, gentle mask ventilation, rapid-onset agents and video laryngoscopy, preserved aspiration precautions with haemodynamic stability.[4]

Pressure-controlled ventilation with a tidal volume of approximately 7 mL/kg predicted body weight, positive end-expiratory pressure of 5 cmH_2O, plateau pressure of 20 cmH_2O, and end-tidal carbon dioxide of 30-35 mmHg provided lung-protective support. The elevated diaphragm, pleural effusions, and right-lower-lobe collapse were likely as relevant as the bowel interposition. Extubation was delayed until awakening and adequate respiratory assessment. The effusions were not further investigated or drained before urgent surgery, and no postoperative CT or formal pulmonary follow-up was performed; these are important limitations. Mannitol 30 g before dural opening gave satisfactory brain relaxation.[14]

Tranexamic acid was also administered; current aSAH guidance does not recommend routine antifibrinolytic therapy to improve functional outcome, so its use should be interpreted cautiously.[15] Depth-of-anaesthesia and quantitative neuromuscular monitoring were unavailable. These limitations and the uneventful outcome in one patient preclude causal conclusions about the selected strategy.

Published anaesthetic experience with Chilaiditi sign or syndrome is sparse. One abdominal surgery case emphasised aspiration prophylaxis and careful ventilation, and two patients underwent total intravenous anaesthesia without complications.[16,17] A cerebral-aneurysm report described an acceptable outcome with propofol-fentanyl TCI.[18] These heterogeneous reports support individualised planning but do not provide comparative evidence for any induction technique. This case adds a neuroanaesthetic example involving pulmonary compromise and modified rapid-sequence induction but remains limited by its single-patient design and incomplete postoperative imaging.

The principal lesson is that Chilaiditi sign should prompt assessment of respiratory mechanics and aspiration-related factors without allowing these concerns to compromise cerebral perfusion or aneurysm stability. In this patient, a carefully titrated modified rapid-sequence

approach, lung-protective ventilation, and delayed extubation were associated with an uneventful perioperative course. This outcome should not be interpreted as evidence that modified induction is safer than classical rapid-sequence induction; management should be individualised to the patient's pulmonary, gastrointestinal, and neurological risks.

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Declaration of Patient Consent

The authors confirm that all necessary patient consent forms have been obtained. In these forms, the patient(s) provided informed consent for the publication of their images and relevant clinical information in the journal. The patients have been informed that while their names and initials will not be published and reasonable efforts will be made to protect their identity, complete anonymity cannot be guaranteed.

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Conflict of Interest

The author(s) report no conflict of interest.

Data Availability Statement

Deidentified patient data from this case report/series will be made available upon reasonable request to the corresponding author following publication, subject to institutional data-sharing policies and ethics approval.

Author's Contributions

Conceptualization: WW. Data curation: WW. Investigation: WW. Writing – original draft: WW. Writing – review & editing: IPPS. Supervision: IPPS. All authors have read and approved the final version of the manuscript.

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