





From preoperative evaluation to stroke center: Management of postoperative acute ischemic stroke



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ABSTRACT

Perioperative acute ischaemic stroke is a rare complication with potentially catastrophic outcomes. It has been shown that paying attention to perioperative neurological deficits and acute ischaemic stroke prevention proves to be beneficial in avoiding these catastrophic outcomes and may lead to determining early therapeutic interventions. This article reviews the perioperative management (covering diagnosis and treatment), prevention (covering surgery postponement, management with anticoagulant/ antiplatelet and the growing interest in statins and beta-blockers) and intraoperative recommendations (covering anaesthetic techniques, ventilation strategies, transfusion and blood pressure management) specifically for the general surgical population. A summary of current treatments is enlightened by recently described evidence for the effectiveness of mechanical thrombectomy.

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1. Introduction

Ischaemic brain injuries are frequent and sometimes occur during the perioperative period. Compared to non-surgical control groups, anaesthetized patients undergoing surgery are at a higher risk for acute ischaemic stroke (AIS) [1]. This can have disastrous consequences in terms of disability, while mortality is eight times

* Corresponding author at: La Pitié Salpêtrière, Neurosurgical Anaesthesiology and Intensive Care Department, 47–83, boulevard de l'Hôpital, 75013 Paris, France. *E-mail address:* vincent.degos@inserm.fr (V. Degos). greater for those patients having received surgery than for those who have not [2]. It has also been observed that patients undergoing carotid endarterectomy or heart surgery are at higher risk of perioperative stroke (defined as the occurrence of ischaemic or haemorrhagic brain injury, during, or within 30 days after surgery) [3,4].

Whilst much attention has been paid to the prevention of AIS in surgical patients, incidence is still non-negligible, with approximately 1 in 1000 patients suffering from stroke following surgery types excluding cardiac, carotid, major vascular surgery and neurosurgery [2] versus 6 in 1000 patients having had major vascular surgery below the diaphragm [5]. For patients undergoing non-cardiac, non-vascular and non-neurological surgery, the risk factors related to AIS include older age, kidney failure, stroke or a history of transient ischaemic attack [3]. It is also important to take into consideration the fact that perioperative strokes following surgery are mainly ischaemic rather than haemorrhagic in nature [6]. Neuro-rehabilitation is a key element for decreasing the consequences of strokes and is sometimes difficult to perform in the context of postoperative AIS, underlining another specificity of the latter.

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Abbreviations: aPTT, activated partial thromboplastin time; ACE i, angiotensinconverting-enzyme inhibitors; AF, atrial fibrillation; AIS, acute ischaemic stroke; BMI, body mass index; CPAP, continuous positive airway pressure; CS, conscious sedation; CT-scan, computerized tomography scan; ECG, electrocardiogram; GA, general anaesthesia; GCS, Glasgow coma scale; HBP, high blood pressure; INR, international normalized ratio; LMWH, low molecular weight heparin; LVH, left ventricular hypertrophy; MAP, mean arterial pressure; MRA, magnetic resonance angiography; MRI, magnetic resonance imaging; NIBP, noninvasive blood pressure; NIDDM, non-insulin-dependent diabetes mellitus; NIRS, near infrared spectroscopy; NPV, negative predictive value; PPV, positive predictive value; PT, prothrombin time; rtPA, recombinant tissue plasminogen activator; TOF, time of flight.

In this review, we will primarily focus on the prevention and management of ischaemic strokes occurring in patients undergoing non-cardiac, non-carotid and non-neurological surgery.

2. Perioperative acute ischaemic stroke management

Fig. 1 presents a clinical scenario including postoperative AIS after total knee replacement. Upon stroke onset, a preestablished procedure facilitates the necessary rapid diagnosis and effective communication between the different specialists involved (anaesthesiologist, neurologist, radiologist and interventional neuroradiologist). Such procedures limit secondary deterioration and improve prognosis. Common predisposing factors include age, a previous stroke, atrial fibrillation, and vascular/metabolic diseases.

Evidence shows that the sooner ischaemic stroke patients are treated, the more they benefit from said treatment [7,8]. In this

context, intravenous recombinant tissue plasminogen activator (rtPA) has a favourable benefit/risk balance only if administered within the 4.5 hours after the onset of symptoms [9].

2.1. Diagnosis

The presence of a new focal deficit or delayed awakening (30 min more than the expected awakening) in the absence of confounding factors should be a red flag for clinicians. Several pharmacological, physiological or pathological factors may potentially mask stroke symptoms during the post-anaesthetic period. Screening tools like the Recognition of Stroke in the Emergency Room scale (ROSIER) can be effectively used in the recovery room (Fig. 2, [10]) and help distinguish stroke suspicion from other acute neurological diseases such as seizures or headaches. The patient will then undergo a complete neurological



Fig. 1. Mechanical thrombectomy in a case of basilar artery occlusion. A 65-year-old obese women (body mass index = 40) experienced severe gonarthrosis. Her medical record highlights high blood pressure-treated with angiotensin-converting-enzyme inhibitors and rilmenidine, dyslipidemia treated with statins, non-insulin-dependent diabetes mellitus treated with metformin and continuous positive airway pressure-treated obstructive sleep apnea. The planned surgery was a left total knee replacement. Preoperative electrocardiogram showed signs of left ventricular hypertrophy with no repolarization problems and transthoracic ultrasound confirmed concentric left ventricular hypertrophy with normal left ventricular ejection fraction but no foramen ovale permeability visualized. The procedure was performed under general anaesthesia associated with femoral block lasted around 45 minutes with approximately 1.2 L blood loss. Final hemoglobin was 9.8 g/dL. The systolic blood pressure dropped from 170 mmHg to 100 mmHg after induction and remained constant until the end with a mean arterial blood pressure above 60 mmHg. During surgery, her temperature fell down to 35°. The patient was therefore continued to be sedated in the recovery room for reheating. There, she received 10 UI of rapid acting insulin for hyperglycaemia (280 mg/dL). After 90 min, she was extubated and transferred from recovery room to ward with a morphine pump when the pain went down to 4 on the Numerical Rating Scale. Physiotherapy began at day 1. Drains were planned to be removed after 72 hours if no bleeding appeared. At 10:32, day 2, she suddenly developed diplopia and left leg motor impairment during physiotherapy. Left leg symptoms disappeared after 5 minutes but then the patient developed right leg motor impairment. After ruling out peripheral causes (postoperative nerve compression, local anaesthetic side effects), hypoglycaemia, postictal state, and migraine aura, the patient had to be managed for possible stroke. The event chronology and the symptoms pleaded for a central lesion (paresis and imbalance evoke basilar artery occlusion). The patient was urgently driven to magnetic resonance imaging. At 11:02, Glasgow Coma Scale dropped to 6 on the way to radiology. The patient was then managed in the recovery room and intubated in emergency before being driven to the computed tomography (CT) scan at 11:22. CT-angiography showed a basilar thrombus. After a collegiate discussion balancing pros and cons arguments for thrombolysis after orthopaedic surgery, she was quickly treated by thrombolysis followed by mechanical thrombectomy. Post-procedure recanalization was optimal and the patient woke-up at 13:15 without any sequelae. A. Left vertebral artery digital subtraction angiography showing an occlusion of the distal aspect of the basilar artery (black arrow). B. Snap shot of the road map after the deployment of the stentriever across the thrombus (white arrow: endovascular catheter and black arrow: distal aspect of the stentriever). C. Representative picture of the thrombus removed by strentriever device (scale bar: 2 mm). D. Control digital subtraction angiography after stentriever retrieval showing a complete recanalization of basilar artery and posterior cerebral arteries.

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