Uterine necrosis in obstetrics after emergency surgical procedure: the dark side of hypogastric artery ligation

TO THE EDITORS: Karoui et al, in their report about uterine necrosis after artery ligation, described an interesting and rare case of surgical complication of the treatment of postpartum hemorrhage following the failure of medical therapy. Moreover, we appreciate the authors’ interest in reporting these surgical adverse events of postpartum hemorrhages, and we take the opportunity to highlight the problem and the consequence of uterine vascular deprivation. In our center, patients presented with analog cases of postpartum hemorrhage caused by traumatic lesion of uterine vessels following cesarean delivery. The patients underwent abdominal delivery for breech presentation, and during surgery, agenesis of the peritoneal tissue of the broad ligament was observed with exposure of the uterine arterial and venous vessels bilaterally; intraoperative hemorrhage following performance of bilateral ligation of the hypogastric artery was also observed. The patient reported uterine necrosis 40 days after the surgical procedure, and laparotomic hysterectomy was performed for sepsis. The women reported fever, abdominal pain, and purulent lochia belatedly, remaining asymptomatic during the puerperium. Literature shows limited cases of uterine necrosis after surgical uterine compression sutures, artery ligation, or artery embolization for the management of postpartum hemorrhages; the incidence and causal factors remain unknown. We may suppose that the onset of uterine necrosis could be the sum of 2 different pathways. First, in patients who underwent artery ligation, the ischemic damage derives from inadequate collateral circulation (round uterine ligament arteries) that do not allow sufficient blood supply to the uterus. Hormonal status, proinflammatory cytokine and chemokine pathway activation, and immune response probably play a key role in the uterine reperfusion mechanism. Artery ligation may cause an unpredictable modification in the endothelial tissue of uterine vessels, resulting in more severe ischemic damage. On the other hand, agenesis of the peritoneum could induce excessive laxity of the peritoneal tissue, and concomitant physiological changes in the connective and extracellular matrix during pregnancy increase the risk of modified physiological uterine involution of postpartum with an increased risk of ischemic injury. The distinctive traits of these 2 cases lie in the development of uterine necrosis after artery ligation and not following uterine compression sutures, as reported in literature data. Finally, uterine necrosis may represent a tardive event without early symptoms, as demonstrated by our cases.

In conclusion, uterine necrosis after artery ligation may be an indicative event in obstetrics to conduct counseling of postsurgical emergency procedures properly, because incomplete knowledge of the timing of uterine reperfusion represents a challenge.

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