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Innovation in surgical technique

Treatment of acute intraoperative Budd-Chiari syndrome by placement of silicone prosthesis☆

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ABSTRACT

Complete liver mobilization for major resections sometimes causes liver tilting due to the release of the suspensory elements of the liver. Rarely this may take to a liver abnormal position with acute obstruction to venous flow at the suprahepatic level (Budd-Chiari syndrome). To avoid this complication, techniques such as post-operative stent implantation have been described. The case of a patient who underwent a complete mobilization of the liver for resection of the inferior venous cava and a right renal tumor, was reported. After that, an acute Budd-Chiari Syndrome was observed caused of the liver malposition, which was solved with the placement of two silicone prostheses in the liver cell.

Tratamiento del síndrome de Budd-Chiari agudo intraoperatorio mediante colocación de prótesis de silicona

Palabras clave:
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RESUMEN

La movilización hepática completa para resecciones mayores provoca, en ocasiones, basculación del hígado secundaria a la liberación de sus elementos suspensorios. Esto puede ocasionar una malposición hepática con obstrucción aguda al flujo venoso a nivel de las suprahepáticas (síndrome de Budd-Chiari). Para salvar esta complicación se han descrito técnicas como colocación de sondas de Foley, balón Sengstaken-Blakemore o implantación de endoprótesis vasculares en el postoperatorio. Aquí presentamos el caso de un paciente al que se le realizó una movilización completa para resección de un tumor renal derecho y resección de vena cava inferior por trombosis de ésta hasta entrada de las venas suprahepáticas. Durante el proceso se produjo un Budd-Chiari agudo por malposición del hígado que fue resuelto con la colocación de dos prótesis de silicona en la celda hepática.

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Introduction

The onset of acute or sub-acute Budd-Chiari syndrome (BCS) following liver surgery has been reported in .1% of cases[1]. Most developed after right liver resections or extended hepatectomies[2–5]. Cases have also been reported after use of the right hepatic lobe from a living donor in liver transplantation[6]. Hypertrophy or rotation of the remnant causes rotation or kinking at the outflow of the suprahepatic veins (SHV) or inferior vena cava (IVC). Treatment to date has been reintervention with fixation of the hepatic remnant to the falciform ligament, anterior wall, etc.[2–5], the use of vascular stents[6,7], placement of Sengstaken-Blakemore balloon,[8–10] Foley catheters,[11] or tissue expanders[12,13]. The onset of acute BCS has also been described in liver transplantation in the event of a mismatch between the size of the donor graft and the recipient's hepatic fossa, solved by the placement of silicone implants[14–16].

We present a case of BCS onset in a patient after complete hepatic mobilisation for excision of a right kidney tumour and complete resection of the inferior vena cava. It was treated by placing a silicone prosthesis.

Surgical technique

A 55-year-old male patient presenting with discomfort in the right renal fossa associated with constitutional syndrome with weight loss of 10 kg over the past two months and dyspnoea on minimal exertion. The CT scan showed a large left kidney mass (15 × 13 × 11 cm), destructuring the kidney, reaching the renal hilum with an image of thrombosis of the renal vein continuing with thrombosis of the inferior vena cava, thrombosis of the distal part of the left renal vein, and thrombosis up to the ileus femoris region (stage IIIB, according to the classification by Ciancio et al.[17]). The case was presented to the Oncology Committee and intervention on the renal mass with the collaboration of hepatobiliary surgeons and cardiovascular surgeons was decided. The Urology Department resected the renal mass alone, as it was impossible to remove the entire tumour en bloc. The surgical department released the right and left hepatic ligaments, dissected, ligated, and sectioned the retrohepatic veins to expose the IVC in its entirety, up to the SHV outflow. The suprahepatic IVC was dissected and surrounded by a vascular snare for control if necessary. Adequate hepatic and systemic drainage were confirmed by clamping below the outflow of the SHV. The cardiovascular surgeons excised the IVC up to the iliac bifurcation, after ligating the left renal vein. The length of the resected vein did not allow its reconstruction. On repositioning in the hepatic fossa, there was descent and cranial displacement of the liver due to the absence of the tumour and the resected thrombosed cava, which caused closure of the venous drainage of the SHV, leading to haemodynamic instability and hepatic congestion (bluish colour, increased volume, and consistency of the liver). The liver surgeons were notified of this, and, on examination, they assessed that the descent of the liver in the hepatic fossa was responsible for the unstable situation. Therefore, a silicone prosthesis was placed to elevate the liver; however, the cranial displacement of the liver towards the diaphragm maintained the difficulty of venous drainage of the liver and the unstable situation. It was decided to place a second prosthesis to keep it in position and prevent its displacement towards the diaphragm. This allowed adequate venous drainage, the liver congestion improved, and the instability disappeared. Neither flowmetry nor echo-Doppler was performed due to the lack of available equipment. During his stay in ICU the patient required haemodialysis for acute kidney failure and developed a pulmonary thromboembolism. The postoperative control CT scan showed correct position with both prostheses, good venous drainage, and adequate hepatic perfusion. Histology was clear cell carcinoma, World Health Organization/International Society of Urological Pathology (WHO/ISUP) grade 4, with extensive sarcomatoid areas (>50%) and inferior cava with lumen occupied by carcinoma and wall infiltration, and adrenal gland involvement (T4N0Mx).

Discussion

Onset of Budd-Chiari syndrome after liver surgery usually follows right liver resections or right trisectionectomies[2–5]. It is due to displacement of the liver remnant leading to kinking of the SHV or compression of the IVC, resulting in compromised hepatic venous drainage. It can also occur in liver transplant recipients when there is mismatch between donor graft size and recipient liver cell size. The most described treatment is reoperation and clamping of the liver to the hepatic ligaments, wall, or diaphragm[2–5]. Recent publications show therapeutic solutions using venous stents[6,7], balloons that are subsequently removed postoperatively[8–10], Foley catheters[11] or tissue expanders[12,13], most show good results.

We present a case of onset of intraoperative BCS after excision of a renal mass and thrombosed IVC secondary to hepatic malposition. The displacement into the hepatic and cranial fossa was not solvable with ligament or abdominal wall clamping. The placement of silicone prostheses has already been described in the literature by liver transplant groups when there is a mismatch between the size of the donor graft and the recipient's hepatic fossa[14–16]. It was performed in our Hepatobiliary Surgery and Transplant Group[14]; the patient made good short and long-term postoperative progress, with a follow-up of more than 10 years, without complications related to the silicone prosthesis used. In other studies, follow-ups of up to 50 months have been described for silicone prostheses placed without any complications[15].

This technical innovation is an additional resource to consider, especially in an intraoperative emergency with the onset of BCS that cannot be resolved with the usual support. The use of current silicone prostheses is safe, with a very low risk of infectious complications, rupture[18], or leakage[19] in the long term, according to the literature.

We conclude that the use of silicone prostheses in hepatic malposition secondary to complete mobilisation and resection of the inferior vena cava has proven effective in the management of secondary acute Budd-Chiari syndrome.

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Conflict of interests

The authors have no conflict of interests to declare.

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Appendix A. Supplementary data

Supplementary material related to this article can be found, in the online version, at doi:https://doi.org/10.1016/j.cireng.2021.08.004.

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