Eur Spine J (2016) 25 (Suppl 1):S257–S266
DOI 10.1007/s00586-016-4562-9

CASE REPORT

Temporary liver and stomach necrosis after lateral approach for interbody fusion and deformity correction of lumbar spine: report of two cases and review of the literature

Haris S. Vasiliadis[1] · Regula Teuscher[1] · Mark Kleinschmidt[1] · Susanne Marrè[2] · Paul Heini[1]

Received: 11 February 2016 / Revised: 31 March 2016 / Accepted: 1 April 2016 / Published online: 6 April 2016

Abstract
Introduction Corrections of spinal deformities have been associated with a potential of postoperative vessel-originating complications. Reports of occlusions of celiac artery seem though to be very rare.
Case reports We present two cases that developed acute arterial supply impairment and subsequent liver and gastric necrosis due to an occlusion of celiac artery, after a spine deformity correction. In both patients a lateral surgical approach to the superior lumbar spine, lateral placement of cages and then a subsequent posterior fixation were performed.
Review The literature search reveals the report of three similar cases, with potentially different pathogenetic mechanisms and with a different prognosis. This complication is assumed to occur due to a Medial Arcuate Ligament syndrome (MALS) developed postoperatively that leaded to compression of the celiac artery against the Medial Arcuate Ligament. The pathogenesis though remains unclear and two theories are assumed to explain the acute appearance of the syndrome; the alteration of the anatomic relationship between the vessels and the surrounding tissues due to the spine deformity correction and an intraoperative direct or indirect traction injury of the celiac trunk that caused or increased its pressure against the medial arcuate ligament.
Conclusion The spine surgeon should be aware of the possibility of postoperative ischemia of the liver and stomach by occlusion of the celiac artery or its supplying branches. Specifically when a large correction of a kyphotic/kyphoscoliotic spine is planed, the surgeon should be alert for an appearance of a MALS.

Keywords Spine deformity · Lumbar spine · Celiac artery · Liver necrosis · Complication

✉ Haris S. Vasiliadis
charilaosvasileiadis@sonnenhof.ch

[1] Spine Service, Sonnenhof Hospital, Buchserstrasse 30, 3006 Bern, Switzerland

[2] Radiology Department, Sonnenhof Hospital, Buchserstrasse 30, 3006 Bern, Switzerland

Introduction

Capener was the first to introduce lumbar interbody fusion using an anterior surgical approach [1]. Later on, the posterior approach became more popular among surgeons [2]. Recently the extreme Lateral Interbody Fusion (XLIF®, NuVasive Inc., San Diego, CA, USA) was introduced as an alternative technique for an anterior interbody fusion, by Pimenta et al. [3, 4]. A main advantage is considered to be the theoretically reduced risk of complications related to a major vessel injury [5]. As the experience in the technique increases, the indications are also widened covering currently a wide range between indirect spinal canal decompression to spine deformity corrections [6, 7]. Although the lateral approach for interbody fusion may provide in selected cases several advantages over isolated posterior fusion, the surgeon should be aware of the potential complications [8–10].

Corrections of spinal deformities have been also per se associated with a potential of postoperative vessel-originating complications due to a consequent acute altering of anatomical relations in the abdominal cavity. The Anterior superior mesenteric artery syndrome (ASMAS) is a well described, however uncommon complication. It is caused by an alteration of the relationship between aorta, anterior

superior mesenteric artery and the duodenum which passes between them [11–13].

However, reports of severe arterial tears or occlusions seem to be even more rare. The literature search reveals only three reports of celiac artery occlusions leading to liver and stomach necrosis after a lateral approach of the spine for deformity corrections. Careful assessment of these cases may reveal though various pathogenetic mechanisms and a different prognosis [14–16]. In all three cases, a lateral thoracophrenicotomy was performed for the approach of the thoracolumbar and/or superior lumbar spine.

We report two cases of patients treated with a lateral approach to the superior lumbar spine, with lateral placement of cages and subsequent posterior fixation that developed an arterial perfusion impairment of the liver and stomach shortly postoperatively. We review all the related literature and discuss the existing theories regarding the pathogenetic mechanisms that may lead to such a complication.

Case reports

Case 1

A 74-year old female presented with chronic back- and leg pain. Radiological control confirmed a left-convex degenerative lumbar scoliosis with Cobb angle of 30° (Fig. 1a, b). She had undergone regional facet infiltrations, three times in the past, with only temporary relief of symptoms. The clinical history of the patient included an operated breast cancer currently under hormonal therapy, thyroidectomy and osteoporosis under oral medical treatment.

A combined lateral and posterior surgical approach was performed. The patient was placed in left lateral decubitus position and the surgical approach was performed on the right side through a muscle splinting approach. Approach to the vertebral bodies was achieved retroperitonealy, through the iliopsoas muscle according to the principals of XLIF technique (Extreme Lateral Interbody Fusion) [3]. It was followed by a complete discectomy and placement of cages (Oracle-Cages, Depuis-Synthes®) filled with a BMP II embedded matrix (InductOS®). This was performed first at level L1/2 and then to L2/3, L3/4 and L4/5. After the cages' placement the surgical incision was closed and the patient was turned to the prone position for the posterior stabilisation. A percutaneous minimally invasive screw fixation from L1 to L5 was then performed, under radiological control (Matrix-System, Synthes®). There were no intraoperative complications; the operation lasted 140 min, was well tolerated by the patient and the postoperative

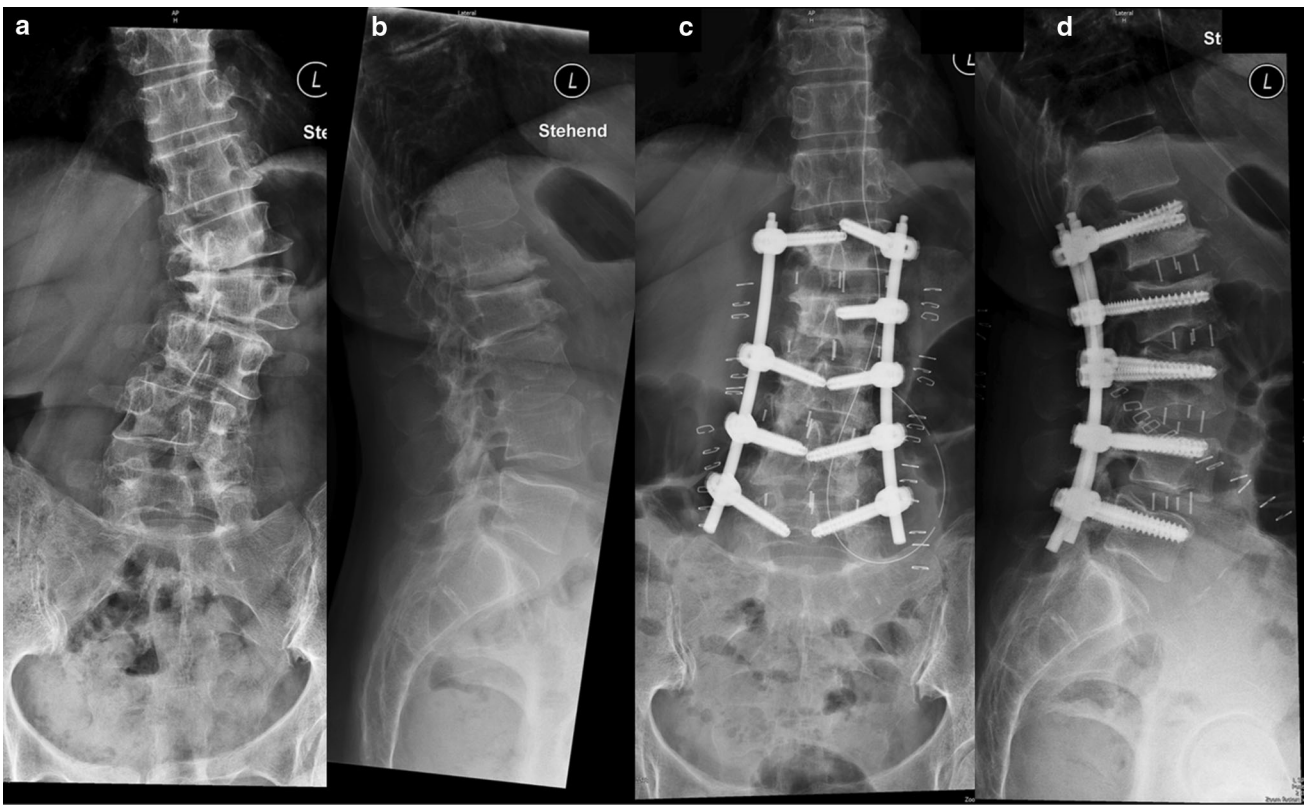

Fig. 1 Preoperative standing X-rays of case 1 (**a**, **b**). Postoperative standing X-rays of case 1 (**c**, **d**)

Table 1 Laboratory outcomes of the postoperative course of case 1

Patient 1	preop	17 h	25 h	30 h	Day 2	Day 3	Day 4	Day 5	Day 7	Day 9	Day 13
AST (15–37 U/L)	26	303	3212	4390	6848	2961	2208	1576	733	31	23
ALT (14–59 U/L)	22	367	3016	3830	5439	3581	880	387	65	352	60
LDH (81–234 U/L)		473		2775	3454					306	
CK (26–192 U/L)	123		2635	2762	3009	2557	958				
CK-MB (0.5–3.6 U/L)			36.6								
INR				1.57	2.11	1.98	1.25	1.04	1.02	1.05	1.00

X-ray shows a correct placement of the implants (Fig. 1c, d).

On first postoperative day the patient complained of exhaustion (heartbeat rate; 140 beats/min, arterial pressure; 90/40. Haemoglobin level was stable (11.6 g/dl). Laboratory control revealed also an increased level of all liver related enzymes. An acute substantial increase of transaminase levels occurred within the following 8 h (Table 1).

Abdominal ultrasound failed to show arterial flow in the porta hepatis. The origin of the celiac trunk and the superior mesenteric artery could not be identified because of overlaying enlarged gas and fluid-filled stomach. Therefore a CT angiography was performed. It shows the origin of the celiac trunk at the level of the diaphragm with stenosis/compression and also a stenosis at the origin of the superior mesenteric artery. The left gastric artery and the splenic artery were filiform but patent. In the porta hepatis and the liver filiform arteries were identified. The liver enhancement was inhomogeneous, liver veins and portal veins were patent. There was marked dilatation of the stomach, a large gallbladder without wall thickening and an impaired enhancement of the spleen. No intraperitoneal air could be shown. There was extraperitoneal air at the site of the surgical approach on the right side (Figs. 2, 3, 4).

On second postoperative day the transaminases did increase further along with LDH and CK. The patient gradually developed a light dyspnoea but retained a normal mental orientation. Coagulation mechanism started to be impaired with a gradual increase of INR to 2.11 (despite the administration of Konakion Phytomenadione). Clinically the patient developed a light pain in right subcoastal abdomen and tachypnoea with light dyspnoea (O_2 Saturation 88 % under O_2).

From the third postoperative day we noticed a slight and gradual decrease of transaminase levels and a stabilisation of clotting mechanism. Ultrasound control revealed an increased arterial perfusion of the liver comparing to the previous evaluation. However blood flow in the celiac truck was still not detectable.

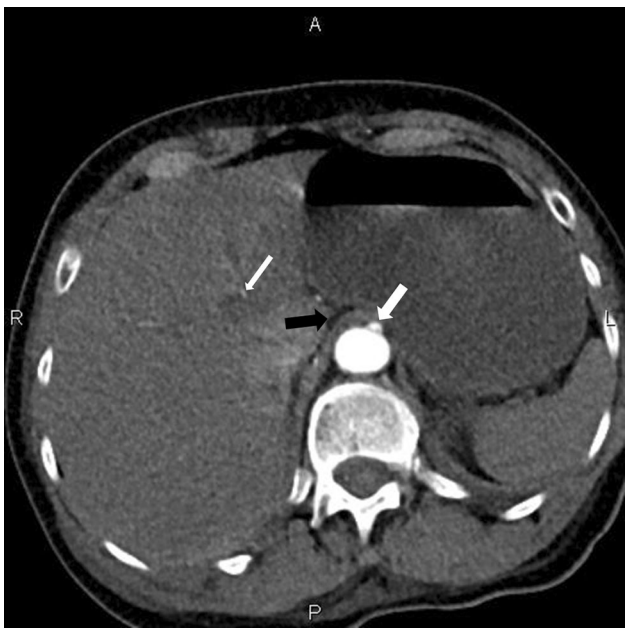

Fig. 2 Axial CT scan, iv contrast, arterial phase: image shows the origin of the celiac trunk (*thick white arrow*) in relation to the diaphragm (level of the median arcuate ligament) (*black arrow*). Filiform intrahepatic arteries (*thin white arrow*)

At fourth day INR returns to almost normal but we noticed that the dyspnoea was increased accompanied by a generalised oedema. Urea and creatinine levels remain not critically influenced (Urea 14.9 mmol/L, Creatinine 58 µmol/L, with normal values of 2.5–6.4 mmol/L and 53–88 µmol/L respectively).

A gastroscopy performed at seventh day showed multiple ischemic ulcers in the stomach and duodenum wall, confirmative of an arterial ischemia. No signs of perforation were apparent.

The transaminases and other liver indexes gradually approached the normal levels up to the ninth postoperative day. The patient complained of no abdominal pain or nausea and food uptake was gradually started after the patient was nourished parenterally before. She complained though of diarrhea and meteorismus which remained also

for the next days. Clinical examination revealed no signs of peritonism. At thirteenth day the parenteral nutrition was stopped and 2 days later the patient was discharged with normal clinical and laboratory findings.

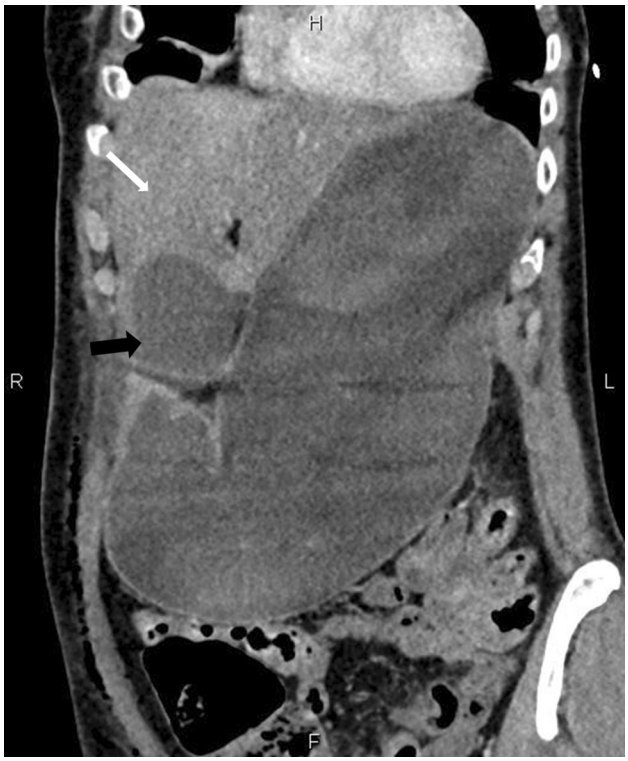

Fig. 3 CT scan arterial phase, coronal reformation: image shows marked dilatation of the fluid filled stomach and enlargement of the gallbladder without wall thickening (*black arrow*). Patchy inhomogeneous enhancement of the liver (*thin white arrow*)

Case 2

A 71 year-old male patient presented with chronic low back pain and radicular pain on left leg. Radiologic control revealed a right convexed degenerative lumbar kyphoscoliosis (with coronal Cobb angle of 40°) with facet joint arthritis and multiple level spine canal stenosis (image 5a, b). Medical history of the patient included a high blood pressure (arterielle hypertonie).

The patient underwent a two-stage operation in the same anaesthesia. An XLIF was performed from the left side with the patient in right decubitus position and three cages (Oracle-Cages, Depuy-Synthes®) filled with morselized bone allograft were inserted under X-ray control at levels L1–2, L2–3 and L3–4. The incision was performed below the thoracic cage and the approach to the spine was achieved retroperitonealy and through the iliopsoas muscle. After an uneventful positioning of the cages, the patient was turned to prone position for a posterior stabilisation. A fixation from Th12 to L4 was performed with minimal invasive techniques under X-ray control with the use of transpedicular screws and rods (Matrix-System, Synthes®). The postoperative X-ray control showed a correct instrumentation placement and a good deformity correction (Fig. 5c, d).

The patient had an uncomplicated recovery from the anaesthesia initially, but gradually developed constipation along with dyspnoea and cough, the next postoperative days. The laboratory control at 4th postoperative day revealed an increase of transaminases and CPR. Coagulation was slightly affected. Renal function was slightly deteriorated, but not critically, compared with preoperative (Table 2).

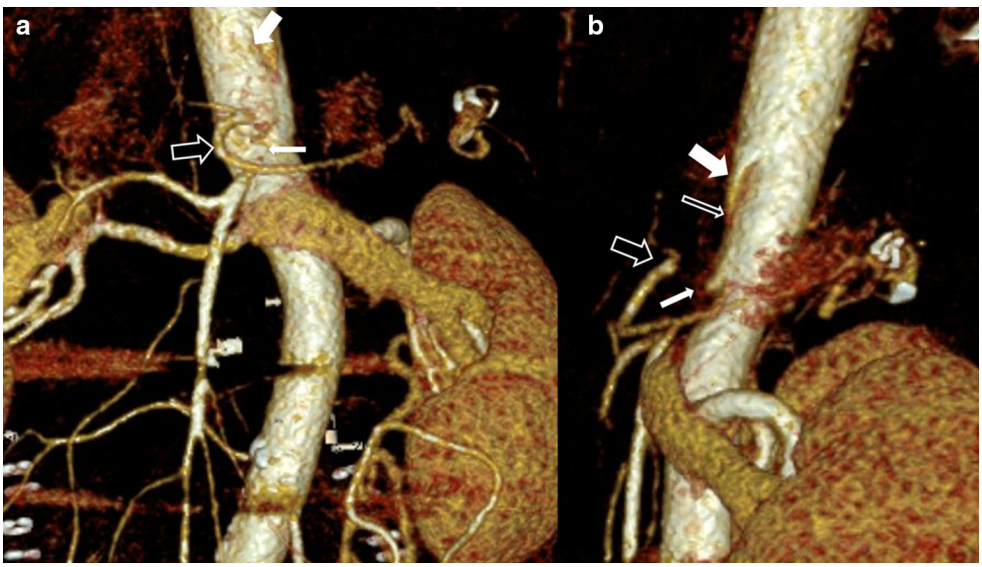

Fig. 4 **a**, **b** 3D VRT of the CT—angiography in coronal and sagittal view. Stenosis/compression (*thin black arrow*) near the origin of the celiac trunk (*thick white arrow*), high grade stenosis of the SMA (*thin white arrow*), patent but narrow splenic artery (*thick black arrow*)

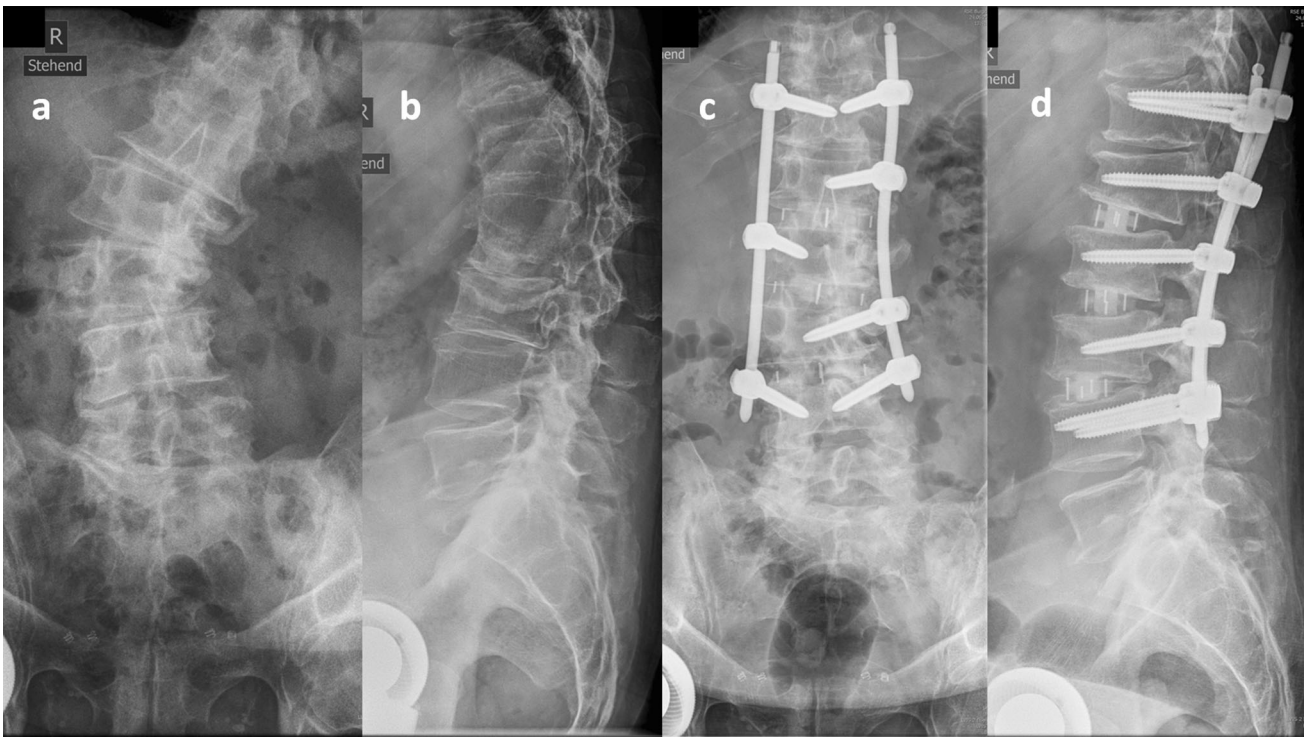

Fig. 5 Preoperative standing X-rays of case 2 (**a**, **b**). Postoperative standing X-rays of case 2 (**c**, **d**)

Table 2 Laboratory outcomes of the postoperative course of case 2

Patient 2	preop	Day 4	Day 5	Day 7	Day 10	Day 15
AST (15–37 U/L)		427	166	64	54	25
ALT (14–59 U/L)		1341	802	372	171	59
LDH (81–234 U/L)			294		325	252
CK (26–192 U/L)			202			
INR	1.2	2				

The patient became severely sick. A CT control at day 5 shows a large hypodense area at the left lobe of the liver (9 × 4 × 11 cm) and also an already known hiatal hernia (upside-down stomach) but with air entrapment within the wall, indicative of wall necrosis and incipient perforation (Fig. 6a, b). The hypodense liver area was attributed to fatty infiltration potentially indicative of perfusion insufficiency. None of these findings were apparent in previous CT control 7 years earlier). The size of the spleen was normal. The patient started receiving parenteral nutrition and a gastroscopy showed a necrotic zone of Stomach-fundus and erosive-ulcer gastritis. Ultrasound evaluation with Doppler underwent the sixth postoperative day was indicative for a perfusion necrosis of the left lobe of liver. The image of gall-bladder contributes also to the hypothesis of an ischemia. However, no current central arterial or vein occlusion was found. Subsequently ultrasound 2 weeks later showed an increase of the size of the hypoechoic area with an expansion to right liver lobe and a subcapsular fluid collection. Major veins had a free flow. The patient under conservative treatment, showed an uneventful clinical progress, with gradual subsidence of the symptoms. Transaminases showed a peak at fourth postoperative day and then followed a declining trend, reaching almost normal levels by the fourteenth day.

Discussion

Interbody fusion provides theoretical advantages over isolated posterolateral fusion. It requires a less traumatic surgical approach, resulting in a reduced intraoperative blood loss, less pain, shorter hospital stays and faster recovery [3, 17, 18]. From a biomechanical point of view, the fusion area is more extended than in posterior fusion techniques [18–20]. Lateral approach also provides a

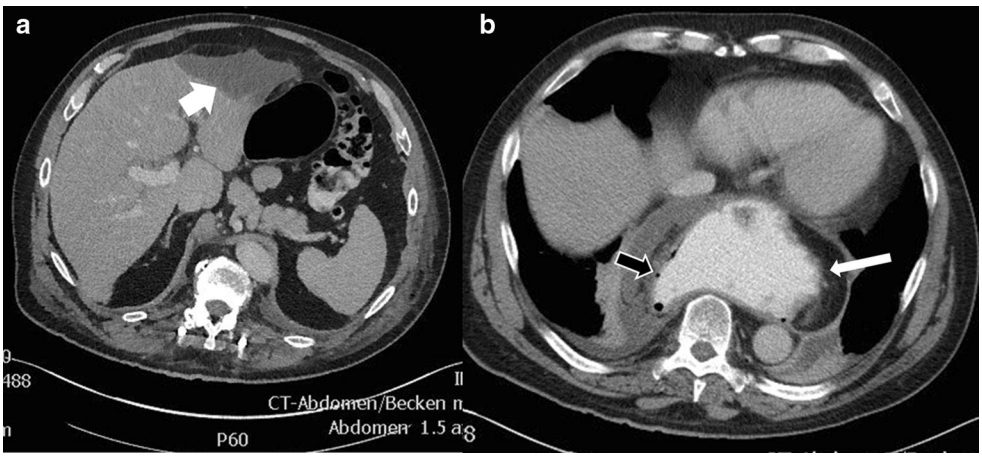

Fig. 6 **a**, **b** Large hypodense area at the left lobe of the liver (9 × 4 × 11 cm) (*thick white arrow*), hiatal hernia (upside-down stomach) (*thin white arrow*), air entrapment within the gastric wall (*black arrow*)

theoretical advantage over the anterior (ALIF), due to the avoidance of peritoneal cavity contents and especially of the mobilization of the great vessels.

Therefore, the lateral surgical approach appears to be an excellent alternative to achieve an interbody fusion. There are though a few intraoperative steps that require awareness in order to avoid potential complications directly related to the surgical approach. The related literature is so far mainly focused on the risk of nerve injuries intraoperatively. This may involve either the injury of the superficial nerves or the injury of the main branches of the lumbar plexus itself [21–23]. The injury of the lumbar plexus and genitofemoral nerve is considered the main risk factor of the transpsoas approach [24–31]. Transient groin or thigh paresthesias are probably the most common but also benign postoperative findings in even 15–25 % of the treated patients, probably attributed to the iliohypogastric or ilioinguinal nerve during preparation through the abdominal wall or in the retroperitoneal space [9, 31–34]. Transient groin or thigh pain or hip flexors weakness is also equally common caused by direct trauma to the psoas [9, 34]. Other complications are quite rarely reported, as bowel perforation or vessel injury [35].

Only three case reports of patients sustaining a liver necrosis related to a spine approach have been found in the literature [14–16]. The first case [16] was a revision correction of a post-traumatic Kyphosis (Cobb angle of 30°) treated with an anterior release and placement of an interbody cage with bone graft at L1 and posterior fixation between T12 and L2. The access was a left transthoracic approach between the 10th and 11th ribs, with a lateral intercostal thoracotomy. Access to L1 was achieved completely retroperitoneal following a circular dissection of the left diaphragm. By 48 h postoperatively the patient developed an acute abdomen accompanied with elevated liver enzyme levels. CT and angiogram evaluation revealed the partial liver necrosis with inhomogeneous arterial perfusion and diffuse arterial hypovascularization due to stenosis of the celiac trunk and the superior mesenteric artery. A subsequent surgical exploration revealed a perforated gallbladder with necrotizing cholecystitis and bilious peritonitis, the liver partially necrotic and the hepatic artery with an inexplicably weak pulse. Further surgical exploration showed that the medial arcuate ligament of the diaphragm overlapped, compressed, and stenosed the proximal 2.0 cm of the celiac trunk, thereby compromising blood flow into the trunk. The superior mesenteric artery was found to have a 2.0-cm intraluminal chronic proximal stenosis. The inferior mesenteric artery provided collateral supply to the upper abdominal vessels, as well as retrograde flow into the superior mesenteric artery celiac trunk and the superior mesenteric artery. A partial transection of the right diaphragmatic arcuate ligament was followed by instantaneous alleviation of the celiac arterial stenosis and obvious return of blood flow. The transaminase levels decreased shortly after this intervention and a CT 1 week later revealed a fully patent celiac artery and minimal residual liver necrosis.

A second reported case [15] was a correction of a severe kyphosis (Cobb 95°) of a type VI Ehler-Danlos patient through an anterior release between T6 and L3 through a left thoracophrenicotomy. The patient had an uneventful postoperative course and 10 days later he underwent a posterior release and fusion from T2 to L5 (correction to Cobb 24.5°). Two days later he developed an abrupt occlusion of the celiac axis just after its origin and hepatic infraction, along with an impaired perfusion of the stomach. The common hepatic artery was occluded at its origin but became more patent distally (probably due to collateral circulation). Peritonitis was developed due to stomach

perforation. Subsequent surgical exploration confirmed the radiological findings. The patient was weaned out of ventilation 3 weeks later with tetraplegia (under C7–C8), probably related to an MRI-confirmed hyperintensity at C5–C6 (oedematous or ischemic process).

Daniels et al. [14] reported an additional case of a rigid Scheuermann kyphosis (T2–L2 Cobb 106°). The patient was obese with type II diabetes mellitus and an intentional weight loss of 13.6 kg over the previous year. An anterior release through a right thoracotomy at the level of 9th rib (complete discectomy-annulectomy-bone grafting from T8 to T12) was followed in the same anaesthesia by a transpedicular screw fixation from T5 to L3 with bilateral hook claw constructs at T2 and T3. Neither the abdomen nor the retroperitoneal space was entered during the anterior approach. Spinal correction to Cobb 50° was achieved. At the second postoperative day clinical symptoms likely due to gastric perforation were developed. Surgical exploration revealed ischemia of the stomach, the gall bladder, the spleen, and a poorly perfused liver. An angiography confirmed an occlusion of the celiac artery at its origin with distal reconstitution of flow to the common hepatic and proper hepatic arteries.

The exact pathogenetic mechanism that may lead to this complication is not completely clear. From the existing literature the reasons may be considered rather heterogeneous among the existing case reports.

Summarising the existing evidence, one theory attributes the occlusion of the celiac truck and the subsequent liver necrosis to the spinal correction per se and to the alteration of the anatomic relationship between the vessels and the surrounding tissues. Such an alteration is already reported in the literature to cause a secondary post-correction superior mesenteric artery syndrome (SMAS) [11–13]. SMAS is caused by reduction of the normal angle between the superior mesenteric artery and aorta scissoring the third part of duodenum, at level of L3 vertebra. A congenitally short ligament of Treinz is considered a predisposing factor, contributing to compression of the third and final portion of the duodenum by the abdominal aorta (AA) and the overlying superior mesenteric artery [11].

A similar anatomic entity, the medial arcuate ligament (MAL) may cause, with a similar pathogenetic mechanism, the external pressure and occlusion of the celiac trunk after a kyphotic or kyphoskoliotic spine correction. The MAL is a fibrous arch that shapes under the diaphragm and usually passes over the aorta at the level of L1, just superior to the origin of the celiac trunk. In about 6–7 % or the general population the MAL has a relatively caudal insertion or the celiac trunk a relatively cephalic origin, leading to compression of the celiac artery [36–39]. This much rarely becomes symptomatic causing a light impairment of perfusion to the upper abdomen (celiac artery compression syndrome (CACS), or medial arcuate ligament syndrome, MALS) [37, 38]. The symptoms are usually not typical and not diagnostic, involving abdominal pain, nausea or vomiting. In chronic cases a weight loss may be also evident.

The change of the relative anatomy due to the correction of Cobb angle may acutely increase the pressure of the MAL to the celiac artery. The correction leads to a functional elongation of the spine which may pull the aorta and the celiac trunk cephalad against the diaphragmatic arcuate ligament. Retrospective analysis of the CTs of our two patients showed an increase of the distance between the lower endplate of Th12 and the sacrum by 28.4 mm (from 173.4 mm) and 12.9 mm (from 176.4 mm) respectively. The lordosis in the operated area was increased from 23° to 40° in the first case and from −18° to 16° in our second patient and the anterior spinal line was elongated by 13.7 and 13.3 mm respectively. The alteration of the anatomy as demonstrated from these measurements and the subsequent elongation of the aorta may be the causing factor of MAL syndrome. Theoretically a less intervertebral distraction or a less increase of the lumbar lordosis could limit this elongation, potentially limiting the possibilities of a MALS. However, such an undercorrection would potentially result in a sagittal imbalance or/and inadequate decompression of the spinal canal.

Moreover, there is evidence to show that the MALS may not be the only cause of celiac truck occlusion postoperatively. This is remarkable that all the reported cases (five, including the two presented in this manuscript) follow a lateral surgical approach. All the cases involved the lateral approach to Th12 or L1, which according to anatomical studies is the level where the celiac artery is usually exiting the aorta and is presumably vulnerable to kinking against the median arcuate ligament [40, 41]. Additionally, in both our cases the revasculization of the liver and stomach recovered spontaneously without the need of any surgical intervention, either division of MAL or altering of the spine correction to the preoperative situation. This may add to the hypothesis that an intraoperative direct or indirect traction injury of the celiac trunk and pressure against the MAL may also play a pathogenetic role. A hypothesis would be that the placement of the patient in a lateral decubitus with further flexion of the table in order to open the space between chest and iliac crest may add to the pathogenesis by stressing the celiac trunk. To our knowledge though, it is not demonstrated that a similar positioning in other type of surgeries (e.g. kidney) may lead to an occlusion of celiac trunk. However, this positioning may potentially add to the stress on the celiac trunk which is anyway evident due to the spinal correction. Although this is not yet proved, the existence of a (probably hypoclinical) MALS may be the pre-existing factor that in both theories (spinal correction and indirect intraoperative traction) plays

a crucial role to the occlusion of the celiac truck and to the subsequent ischemia of liver and stomach. In both our cases no lever near the celiac trunk was used and a Syn-Frame Access and Retractor system (Synthes©) was utilised to reduce the risk of direct contact and subsequent injury of the anteriorly located spine or abdominal vessels.

Our first case shows a stenosis of the celiac trunk probably caused by its course under the median arcuate ligament together with a high grade stenosis at the origin of the superior mesenteric artery. In case of MALS the superior mesenteric artery together with the inferior mesenteric artery serves as a collateral blood supply [42, 43]. Blood supply to the liver is also maintained through the portal vein. Probably the preexisting anatomical situation together with the stenosis of the SMA and the intra-operative positioning of the patient and perhaps also hypotonia and hypovolemia lead to a critical hypoperfusion of liver and stomach resulting in temporary ischemia.

Nevertheless, the arterial wall insufficiency and predisposition to thrombosis seems also to be an additional factor for the celiac trunk occlusion after spine surgery. Almost all of the reported cases seem to suffer from either an Ehler-Dahnlos syndrome, diabetes, high blood pressure or/and prolonged age that predispose to artery thrombosis per se [44]. So, at least in our cases, a potential arteriosclerosis due to the patients' prolonged age may have predisposed and along with the other potential factors (spine correction, intra-operative traction and potentially pre-existing MALS) may have led to the celiac trunk occlusion.

A remaining open question based on the so few reported cases represents the importance of the individual possible predisposing factors for a celiac truck occlusion and subsequent liver and stomach necrosis. It seems that more than one of the above discussed factors (spine correction, lateral approach with presumably celiac trunk traction, preexistance hypoclinical MALS, arterial wall insufficiency-thrombosis predisposition) are required for the development of such a complication.

This remains unclear whether patients with pre-existing symptoms compatible with MAL syndrome (unexplained abdominal pain, nausea, vomiting or weight loss) that will undergo a correction of a spine deformity should be examined also for the possibility of anatomic variations of celiac aorta and its branches. The fact that most of the patients have already preoperatively a CT and/or MRI scan, could simplify the conduction of a CT or MRI angiography preoperatively in a more regular basis, at least for high risk patients. There is no evidence though to detect the specificity and the accuracy of such a measure.

In any case, an appearance of abdominal pain, nausea or vomiting after a spine correction surgery may be indicative, although not diagnostic for an acute MALS. Clinical signs of peritonitis may be indicative of stomach perforation. The concomitant acute and progressive elevation of the liver enzymes in laboratory examinations increase the suspicion for MALS, but the diagnosis is confirmed by CT or MR angiography. In chronic severe cases of MALS a surgical release of the celiac trunk is indicated [45]. However, the necessity of an emergency surgical release in acute cases is not well studied. In any case a stomach perforation and subsequent peritonitis should be prevented and when apparent should be immediately surgically treated. In both our cases though, no surgical treatment was needed and the situation was eventually self-limited.

Conclusions

Celiac trunk occlusion is a rare complication that may follow a correction of a spinal deformity with lateral approach of the spine. Although the patients had experienced severe clinical symptoms the natural history at the end was self-limited and benign in both of our two cases. Only five cases (including ours) have been so far reported in the literature and the pathogenetic mechanism remains still obscure. Although there seem to be a few predisposing factors, it is still unknown which of them plays the most important role and under which circumstances the syndrome is provoked.

In any case, the surgeon should be aware of the possibility of postoperative ischemia of the liver and stomach by occlusion of the celiac artery or its supplying branches. Specifically when a large correction of a kyphotic/kyphoscoliotic spine is planed, the surgeon should be alert for an appearance of a MALS.

Compliance with ethical standards

Conflict of interest None of the authors declare a conflict of interest. The study was not sponsored.
The authors declare that they have full control of all primary data and agree to allow the journal to review their data if requested.

References

1. Tay BBK, Berven S (2002) Indications, techniques, and complications of lumbar interbody fusion. Semin Neurol 22:221–230. doi:10.1055/s-2002-36545
2. Cloward RB (1985) Posterior lumbar interbody fusion updated. Clin Orthop 193:16–19. doi:10.1097/00003086-198503000-00003
3. Ozgur BM, Aryan HE, Pimenta L, Taylor WR (2006) Extreme Lateral Interbody Fusion (XLIF): a novel surgical technique for anterior lumbar interbody fusion. Spine J Off J N Am Spine Soc 6:435–443. doi:10.1016/j.spinee.2005.08.012
4. Pimenta L (2001) Lateral endoscopic transpsoas retroperitoneal approach for lumbar spine surgery. In: Proceedings of the 8th Brazilian Spine Society Meeting, Belo Horizonte, Minas Gerais, Brazil, May 2001

5. Daubs MD, Lenke LG, Cheh G et al (2007) Adult spinal deformity surgery: complications and outcomes in patients over age 60. Spine 32:2238–2244. doi:10.1097/BRS.0b013e31814cf24a
6. Berjano P, Lamartina C (2013) Far lateral approaches (XLIF) in adult scoliosis. Eur Spine J Off Publ Eur Spine Soc Eur Spinal Deform Soc Eur Sect Cerv Spine Res Soc 22(Suppl 2):S242–S253. doi:10.1007/s00586-012-2426-5
7. Phan K, Rao PJ, Scherman DB et al (2015) Lateral lumbar interbody fusion for sagittal balance correction and spinal deformity. J Clin Neurosci Off J Neurosurg Soc Australas 22:1714–1721. doi:10.1016/j.jocn.2015.03.050
8. Grimm BD, Leas DP, Poletti SC, Johnson DR (2014) Postoperative complications within the first year after extreme lateral interbody fusion: experience of the first 108 patients. J Spinal Disord Tech. doi:10.1097/BSD.0000000000000121 **(Epub ahead of print)**
9. Hrabalek L, Adamus M, Gryga A et al (2014) A comparison of complication rate between anterior and lateral approaches to the lumbar spine. Biomed Pap Med Fac Univ Palacký Olomouc Czechoslov 158:127–132. doi:10.5507/bp.2012.079
10. Peterson MD (2015) Complications avoidance in extreme lateral interbody fusion (XLIF): video lecture. Eur Spine J Off Publ Eur Spine Soc Eur Spinal Deform Soc Eur Sect Cerv Spine Res Soc 24(Suppl 3):439–440. doi:10.1007/s00586-015-3947-5
11. Andrews SN, Sanders G, Cooper MJ (2005) The acute surgical abdomen following Kyphoscoliosis corrective surgery. Ann R Coll Surg Engl 87:W3–W5. doi:10.1308/147870805x60752
12. Braun SV, Hedden DM, Howard AW (2006) Superior mesenteric artery syndrome following spinal deformity correction. J Bone Joint Surg Am 88:2252–2257. doi:10.2106/JBJS.E.00348
13. Tsirikos AI, Jeans LA (2005) Superior mesenteric artery syndrome in children and adolescents with spine deformities undergoing corrective surgery. J Spinal Disord Tech 18:263–271
14. Daniels AH, Jurgensmeier D, McKee J et al (2009) Acute celiac artery compression syndrome after surgical correction of Scheuermann kyphosis. Spine 34:E149–E152. doi:10.1097/BRS.0b013e3181918f05
15. Debnath UK, Sharma H, Roberts D et al (2007) Coeliac axis thrombosis after surgical correction of spinal deformity in type VI Ehlers-Danlos syndrome: a case report and review of the literature. Spine 32:E528–E531. doi:10.1097/BRS.0b013e31813162b3
16. von Glinski KS, Krettek C, Blauth M, Oldhafer KJ (2000) Hepatic ischemia as a complication after correction of posttraumatic gibbus at the thoracolumbar junction. Spine 25:1040–1044. doi:10.1097/00007632-200004150-00021
17. Arnold PM, Anderson KK, McGuire RA (2012) The lateral transpsoas approach to the lumbar and thoracic spine: a review. Surg Neurol Int 3:S198–S215. doi:10.4103/2152-7806.98583
18. Rodgers WB, Gerber EJ, Patterson JR (2010) Fusion after minimally disruptive anterior lumbar interbody fusion: analysis of extreme lateral interbody fusion by computed tomography. SAS J 4:63–66. doi:10.1016/j.esas.2010.03.001
19. Heth JA, Hitchon PW, Goel VK et al (2001) A biomechanical comparison between anterior and transverse interbody fusion cages. Spine 26:E261–E267. doi:10.1097/00007632-200106150-00012
20. Laws CJ, Coughlin DG, Lotz JC et al (2012) Direct lateral approach to lumbar fusion is a biomechanically equivalent alternative to the anterior approach: an in vitro study. Spine 37:819–825. doi:10.1097/BRS.0b013e31823551aa
21. O'Brien J, Haines C, Dooley ZA et al (2014) Femoral nerve strain at L4–L5 is minimized by hip flexion and increased by table break when performing lateral interbody fusion. Spine 39:33–38. doi:10.1097/BRS.0000000000000039
22. Dakwar E, Le TV, Baaj AA et al (2011) Abdominal wall paresis as a complication of minimally invasive lateral transpsoas interbody fusion. Neurosurg Focus 31:E18. doi:10.3171/2011.7.FOCUS11164
23. Dakwar E, Vale FL, Uribe JS (2011) Trajectory of the main sensory and motor branches of the lumbar plexus outside the psoas muscle related to the lateral retroperitoneal transpsoas approach. J Neurosurg Spine 14:290–295. doi:10.3171/2010.10.SPINE10395
24. Benglis DM, Vanni S, Levi AD (2009) An anatomical study of the lumbosacral plexus as related to the minimally invasive transpsoas approach to the lumbar spine. J Neurosurg Spine 10:139–144. doi:10.3171/2008.10.SPI08479
25. Ebraheim NA, Xu R, Huntoon M, Yeasting RA (1997) Location of the extraforaminal lumbar nerve roots. An anatomic study. Clin Orthop 230–235. doi:10.1097/00003086-199707000-00030
26. Gu Y, Ebraheim NA, Xu R et al (2001) Anatomic considerations of the posterolateral lumbar disk region. Orthopedics 24:56–58
27. Kepler CK, Bogner EA, Herzog RJ, Huang RC (2011) Anatomy of the psoas muscle and lumbar plexus with respect to the surgical approach for lateral transpsoas interbody fusion. Eur Spine J Off Publ Eur Spine Soc Eur Spinal Deform Soc Eur Sect Cerv Spine Res Soc 20:550–556. doi:10.1007/s00586-010-1593-5
28. Lehmen JA, Gerber EJ (2015) MIS lateral spine surgery: a systematic literature review of complications, outcomes, and economics. Eur Spine J Off Publ Eur Spine Soc Eur Spinal Deform Soc Eur Sect Cerv Spine Res Soc 24(Suppl 3):287–313. doi:10.1007/s00586-015-3886-1
29. Moro T, Kikuchi S, Konno S, Yaginuma H (2003) An anatomic study of the lumbar plexus with respect to retroperitoneal endoscopic surgery. Spine 28:423–428. doi:10.1097/01.BRS.0000049226.87064.3B **(discussion 427–428)**
30. Patel VC, Park DK, Herkowitz HN (2012) Lateral transpsoas fusion: indications and outcomes. ScientificWorldJournal 2012:893608. doi:10.1100/2012/893608
31. Uribe JS, Arredondo N, Dakwar E, Vale FL (2010) Defining the safe working zones using the minimally invasive lateral retroperitoneal transpsoas approach: an anatomical study. J Neurosurg Spine 13:260–266. doi:10.3171/2010.3.SPINE09766
32. Dakwar E, Cardona RF, Smith DA, Uribe JS (2010) Early outcomes and safety of the minimally invasive, lateral retroperitoneal transpsoas approach for adult degenerative scoliosis. Neurosurg Focus 28:E8. doi:10.3171/2010.1.FOCUS09282
33. Oliveira L, Marchi L, Coutinho E, Pimenta L (2010) A radiographic assessment of the ability of the extreme lateral interbody fusion procedure to indirectly decompress the neural elements. Spine 35:S331–S337. doi:10.1097/BRS.0b013e3182022db0
34. Sharma AK, Kepler CK, Girardi FP et al (2011) Lateral lumbar interbody fusion: clinical and radiographic outcomes at 1 year: a preliminary report. J Spinal Disord Tech 24:242–250. doi:10.1097/BSD.0b013e3181ecf995
35. Balsano M, Carlucci S, Ose M, Boriani L (2015) A case report of a rare complication of bowel perforation in extreme lateral interbody fusion. Eur Spine J Off Publ Eur Spine Soc Eur Spinal Deform Soc Eur Sect Cerv Spine Res Soc 24(Suppl 3):405–408. doi:10.1007/s00586-015-3881-6
36. Curl JH, Thopson NW, Stanley JC (1971) Median arcuate ligament compression of the celiac and superior mesenteric arteries. Ann Surg 173:314–320. doi:10.1097/00000658-197102000-00022
37. Kazan V, Qu W, Al-Natour M et al (2013) Celiac artery compression syndrome: a radiological finding without clinical symptoms? Vascular. doi:10.1177/1708538113478750
38. Park CM, Chung JW, Kim HB et al (2001) Celiac axis stenosis: incidence and etiologies in asymptomatic individuals. Korean J Radiol 2:8–13. doi:10.3348/kjr.2001.2.1.8
39. Soman S, Sudhakar SV, Keshava SN (2010) Celiac axis compression by median arcuate ligament on computed tomography

among asymptomatic persons. Indian J Gastroenterol Off J Indian Soc Gastroenterol 29:121–123. doi:10.1007/s12664-010-0028-x
40. Yan H, Kaneko M, Kato T et al (1994) Relationship of the celiac and superior mesenteric arteries to the vertebral bodies and its clinical relevance. Radiat Med 12:105–109
41. Yang IY, Oraee S, Viejo C, Stern H (2011) Computed tomography celiac trunk topography relating to celiac plexus block. Reg Anesth Pain Med 36:21–25. doi:10.1097/AAP.0b013e318203067f
42. Johnston KW, Lindsay TF, Walker PM, Kalman PG (1995) Mesenteric arterial bypass grafts: early and late results and suggested surgical approach for chronic and acute mesenteric ischemia. Surgery 118:1–7. doi:10.1016/s0039-6060(05)80002-9
43. Mensink PBF, van Petersen AS, Geelkerken RH et al (2006) Clinical significance of splanchnic artery stenosis. Br J Surg 93:1377–1382. doi:10.1002/bjs.5481
44. Vogel LC, Lubicky JP (1996) Neurologic and vascular complications of scoliosis surgery in patients with Ehlers-Danlos syndrome. A case report. Spine 21:2508–2514. doi:10.1097/00007632-199611010-00021
45. Rubinkiewicz M, Ramakrishnan PK, Henry BM et al (2015) Laparoscopic decompression as treatment for median arcuate ligament syndrome. Ann R Coll Surg Engl 97:e96–e99. doi:10.1308/rcsann.2015.0025