INTRODUCTION

The Nathanson retractor has been developed for liver retraction in both adult and paediatric patients undergoing laparoscopic upper gastrointestinal surgery to retract the liver. Intra-operatively it has been noted that the left lobe of the liver often exhibits visual signs of retraction ischemia [1] (Figure 1) which is thought to resolve spontaneously following the removal of the retractor. It has been recognised, in a number of studies reviewing laparoscopic upper abdominal surgery in adult patients, that the use of the Nathanson Retractor is associated with a transient rise in hepatic enzymes which peaks at 24 - 48 hours and resolves spontaneously within 3 - 7 days of the operation [2-3]. Liver retraction has not previously been associated with post-operative clinical illness or permanent liver damage. We present two patients in whom the Nathanson retractor was used in an uneventful fashion and subsequently were found to have clear signs of atrophy of the left lobe of the liver.

CASE PRESENTATION

The first patient was a young boy with chronic respiratory disease requiring intermittent oxygen and the other a young girl with congenital hypoaldosteronism requiring large volumes of sodium chloride daily. Both patients had an uneventful laparoscopic hiatal repair, Nissens fundoplication and insertion of gastrostomy for persistent reflux, vomiting and failure to thrive at 9 months and 3 months of age respectively. At surgery the left lobe of the liver was retracted using a small sized Nathanson retractor through the abdominal wall at the left side of the midline close to the xiphisternum. The retraction time was 120 minutes and 70 minutes respectively. There were no liver lacerations, blood transfusions or sepsis and their post-operative medical and surgical recovery was uneventful. Both patients started oral fluid on day 1 and gastrostomy feed on day 3. They were discharged home at 8 and 9 days post-operatively due to their complicated medical backgrounds and social circumstances. There was a transient rise in ALT after their operations but bilirubin and full blood counts remained normal throughout. Both did well initially but required robotically assisted re-do hiatal repair and fundoplication for recurrent symptoms at 3.5 years and 17 months of age respectively. At revision surgery the lateral segment of the left lobe of the liver was noted to be clearly atrophied with a line of atrophic demarcation corresponding to the position of the Nathanson retractor (Figure 2).

DISCUSSION

Transient elevation of serum liver enzymes has been reported...
Central using the Nathanson retractor. The mechanism seems to be one of revision surgery, represents the consequences of liver retraction segment of the left lobe of the liver, as witnessed at the time of clinical outcomes. In our patients we believe the atrophied lateral levels in the early post operative period and all had favourable increase in liver enzymes.

In both studies the patients liver enzymes returned to normal levels in the early post operative period and all had favourable clinical outcomes. In our patients we believe the atrophied lateral segment of the left lobe of the liver, as witnessed at the time of revision surgery, represents the consequences of liver retraction using the Nathanson retractor. The mechanism seems to be one of the retraction ischemia. The angle, force and duration of traction are likely to have contributed to the process. However, such factors are difficult to predict or measure in individual paediatric patients who present in various sizes and with different anatomical characteristics. Liver retraction and adequate exposure are essential components of open and laparoscopic hiatal repair and fundoplication procedures. We routinely place the retractor high in the epigastrium using minimal retraction force for duration of 45 to 120 minutes depending on the nature and complexity of the case. Other factors that may have contributed to the insult include age and pre-existing medical conditions.

In the presented patients it would seem that ischemia and atrophy of the lateral segments of the left lobe of the liver has caused no significant clinical squeal. However, the outcome in the long term remains uncertain though unlikely to be different. Further, such a favourable outcome might not be the case if the use of the retractor is confined to the central point of the liver as seen in portoenterostomy, partial liver resection, bile duct surgery and pancreatic-duodenal surgery.

CONCLUSION

Contrary to previously held views the Nathanson retractor can cause permanent damage to the liver. Such an insult, if limited to the left lobe would probably cause no significant ill health. However it seems reasonable to suggest that fixed rigid liver retractors should be released intermittently where forced or prolonged periods of retraction are required, in surgery which necessitates retraction at the central point of the liver and in patients with a large liver volume or relevant pre-existing illness.

REFERENCES


Cite this article