Human Pathology (2017) 67, 45–53

ELSEVIER

Human PATHOLOGY

www.elsevier.com/locate/humpath

Original contribution

The impact of knowledge transfer on the detection of venous invasion in colorectal cancer☆

Richard Kirsch MBChB, PhD[a,*], Naziheh Assarzadegan MD[b], David E. Messenger MBChB[c], Ari Juda MD[d], Robert H. Riddell MD[a], Aaron Pollett MD[a], Catherine J. Streutker MD[e], Dimitrios X. Divaris MBChB[f], Ken J. Newell MD, PhD[g], Russell G. Price MD[h], Sharyn Smith MD[i], Sahar Al-Haddad MBChB[j], Jeremy R. Parfitt MD[k], David K. Driman MBChB[k]

[a]*Mount Sinai Hospital and University of Toronto, Toronto, Ontario, Canada M5G 1X5*
[b]*University of Florida, Gainesville, FL 32610*
[c]*University Hospitals Bristol NHS Foundation Trust, Bristol, United Kingdom Bs28hW*
[d]*University of Toronto, Toronto, Ontario, Canada M5S 1A5*
[e]*St. Michael's Hospital, Toronto, Ontario, Canada M5B1W8*
[f]*Grand River Hospital, Kitchener, Ontario, Canada N2G 1G3*
[g]*Grey Bruce Health Services, Owen Sound, Ontario, Canada NK4 6M9*
[h]*Royal Victoria Hospital, Barrie, Ontario, Canada L4N 6M2*
[i]*Stratford General Hospital, Stratford, Ontario, Canada N5A 2Y6*
[j]*Hamilton Health Sciences Centre, Hamilton, Ontario, Canada L8S 4K1*
[k]*London Health Sciences Centre, London, Ontario, Canada, N6A 5A5*

Received 20 April 2017; revised 21 June 2017; accepted 5 July 2017

Keywords:
Colorectal cancer;
Venous invasion;
Elastin;
Knowledge transfer;
Pathology

Venous invasion (VI) is an independent predictor of hematogenous metastasis and mortality in colorectal cancer (CRC) yet remains widely underreported. Its detection may require recognition of subtle morphologic clues, which at times are only unmasked with an elastin stain. This study evaluates the impact of a knowledge transfer initiative (KTI) on VI detection in a "real-world" pathology practice setting. Following participation in an interobserver variability study of VI detection (Kirsch et al, 2013), 12 participants received educational materials highlighting key issues in VI detection. Eighteen months later, participants were invited to submit pathology reports from all CRC resections signed out 18 months prior to and 18 months following the KTI (n = 266 and n = 244, respectively). Nine pathologists participated. Reports were reviewed for VI and other established prognostic factors. Numbers of elastin stains and tumor-containing blocks were also recorded. Comparative analyses were adjusted for baseline differences in tumor, lymph node, and metastasis stage; tumor location; use of neoadjuvant therapy; and number of tumor-containing blocks. VI detection increased significantly post-KTI versus pre-KTI (39.3% versus 18.4%, adjusted odds ratio [OR] 2.86 [1.91-4.28], $P < .001$). Increased VI detection post-KTI was observed in both stage II (31.8% versus 12.5%, adjusted OR 3.27 [1.45-7.42], $P = .004$) and stage III CRC (62.4% versus 28.2%, adjusted

☆ Disclosures: none to disclose.

* Corresponding author at: Department of Pathology and Laboratory Medicine, Mt Sinai Hospital, 600 University Ave, Toronto, Ontario, M5G 1X5.
E-mail address: rkirsch@mtsinai.on.ca (R. Kirsch).

http://dx.doi.org/10.1016/j.humpath.2017.07.004

OR 4.23 [2.37-7.55], $P < .001$). All pathologists demonstrated increased VI detection post-KTI. Use of elastin stains was significantly higher post-KTI versus pre-KTI (61.5% versus 5.3% of cases respectively, $P < .001$). This study demonstrates the effectiveness of knowledge transfer in increasing VI detection in routine pathology practice.

1. Introduction

Venous invasion (VI) is a well-established independent predictor of hematogenous metastasis and mortality in colorectal cancer (CRC) [1-14]. Its detection is of particular importance in stage II CRC because it may prompt oncologists to consider adjuvant chemotherapy. The most recent data set from the Royal College of Pathologists, United Kingdom (RCPath UK), recommends that VI should be detected in at least 30% of CRC resections [15]; no audit standard is suggested in the College of American Pathologists (CAP) CRC protocol.

Population-based studies from both the UK and Canada suggest that VI is underreported [16,17]. For example, an audit from 2007 in the Northern and Yorkshire Region in the UK, which included 5947 CRC pathology reports, found a rate of extramural VI of 18%, below the 25% minimum threshold set by the RCPath at that time [16]. In 2010, audit data revealed a VI detection rate of 14% in Ontario, Canada [17]. A concurrent survey of Ontario pathologists found that 70% of pathologists estimated their VI detection rates to be less than 10%. Practice in a university-affiliated hospital, a subspecialty interest in gastrointestinal (GI) pathology, and application of the so-called orphaned artery sign (Fig. 1) were shown to be independently associated with self-reported VI detection rates of greater than 10% [17].

The underreporting of VI in Ontario prompted us to evaluate the utility of elastin staining in increasing VI detection. Using a predefined study set and 12 observers, we demonstrated that the use of an elastin stain was associated with a 3-fold increase in VI detection compared with hematoxylin and eosin (H&E) alone [18]. The findings from this study provided the impetus for a knowledge transfer initiative (KTI), whereby each of the participants (6 general and 6 GI pathologists) received the following interventions: (1) detailed feedback on their individual and the overall VI detection rates, (2) illustrated handouts highlighting key morphologic clues to VI detection and the utility of elastin stains, and (3) benchmark data for VI detection rates (based on RCPath UK CRC data set). The aim of the current study was to determine whether this KTI (ie, participation in the interobserver variability study and educational initiatives that followed) increased the detection of VI in routine clinical practice.

2. Materials and methods

Research Ethics Board approval was obtained (Mount Sinai Hospital, Toronto).

2.1. Case materials and study periods

Immediately following completion of the Ontario VI study [18], all participating pathologists (6 general [non-GI]and 6 subspecialist GI) received the following interventions: (1) detailed feedback on their individual and the overall VI detection rates, (2) handouts illustrating morphologic clues to VI detection and utility of elastin stains (Supplementary Figs. 1 and 2), and (3) benchmark data for VI detection rates based on the RCPath UK CRC data set (second edition) [19]. Eighteen months later, participants were invited to submit pathology reports from all CRC resections signed out 18 months prior to and 18 months following completion of the KTI (ie, the Ontario VI study and the interventions that followed). The pre-KTI period was from January 1, 2010, to June 30, 2011, whereas the post KTI period was from January 1, 2012, to June 30, 2013.

Pathologists were unaware that their pathology reports would be audited until completion of both pre- and post-KTI periods. Of the 12 pathologists who participated in the Ontario VI study, 9 were available for participation in the follow-up study. Of the 3 pathologists who did not participate, one had moved to a different province during the study period, one had an extended leave of absence during the study period, and one (D. K. D.) was involved in the design of the current study and therefore excluded. Given that the 2010 audit data from Ontario showed that VI was reported in 14% of cases [17], it was estimated that an 85% increase in the VI detection rate could be achieved following the KTI. At 90% power and a 5% significance level, 235 pathology reports from the participating pathologists would be required for each of the pre- and post-KTI time periods. For those pathologists who had reported fewer than 30 CRC resections over the study period (n = 4), pre- and post-KTI periods were extended by an additional 6 months to ensure that each pathologist provided an adequate number of reports. A total of 510 pathology reports were examined (266 prestudy, 244 poststudy).