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Review Article

Post-polypectomy bleeding after colonoscopy on uninterrupted aspirin/non steroideal antiflammatory drugs: Systematic review and meta-analysis

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ABSTRACT

Background and aim: The aim of this systematic review and meta-analysis was to assess the risk of post-polypectomy bleeding (PPB) in patients that underwent colorectal polypectomy and exposed to ASA/NSAIDs.
Methods: Relevant publications were identified in MEDLINE/EMBASE for the period 1950–2016. Studies with specified ASA/NSAIDs exposure and bleeding rate were included. Study quality was ascertained according to Newcastle-Ottawa Scale. Forest plot was based on fixed or random effect models in relation to the heterogeneity.
Results: 11 studies (4 prospective and 7 retrospective) including 9307 patients were included in the analyses. Overall, 344 patients (OR 1.8; 95% CI 1.2–2.7; p-value 0.001, I^2 52%) experienced rectal bleeding after procedure. While the rate of immediate PPB on aspirin and/or NSAIDs was not increased (OR 1.1; CI 95% 0.6–2.1; d.f. = 1, p = 0.64, I^2 0%), the risk of delayed PPB was augmented (OR 1.7; 95% CI 1.2–2.2; d.f. = 8, p = 0.127, I^2 36%).
Conclusions: ASA/NSAIDs are not a risk factor for immediate PPB but the chance of delayed is increased.
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1. Introduction

Endoscopic resection of adenomas is the best treatment for prevention of colorectal cancer because it is safe and efficacious [1]. Nonetheless, the reported percentage of post-polypectomy bleeding [PPB) ranges from 0.07–1.7% [2–4]. Prior studies recognized some features related to PPB, as polyp size, position in the right colon, sessile morphology, number of polyps, comorbidities, endoscopist's experience and use of antiplatelet/anticoagulant drugs [4–12]. There are no high quality evidences for an association between antiplatelet drugs and PPB. European Society of Gastrointestinal Endoscopy (ESGE) guidelines [13] stated that polyps can be safely removed without interruption (moderate evidence) of aspirin (ASA) therapy (with the exception of large colonic mucosectomy ≥2 cm), but systematic evidence about this matter still lacks and also for post-procedural indications. On the other hand, an increased percentage of patients take these drugs because of a risk of ischaemic events if treatment is interrupted. Most studies indicated that ASA is not a risk factor for PPB [5,9,10,12,14,15]. In a retrospective survey, Pan et al. [16] showed a significant association between PPB and ASA, but not with Non Steroidal Anti-Inflammatory Drugs (NSAIDs). Shiffman et al. [14] concluded that, although the use of ASA/NSAIDs increased the incidence of minor self-limited bleeding, major bleedings were not significant. Metz et al. [17] showed a significant relationship between PPB and ASA, but the study was conducted on large colonic lesions. In particular, the use of a low-dose ASA regimen as an anti-thrombotic measure has become increasingly common. In 2007, the Agency for Healthcare Research and Quality (AHRQ) reported that nearly 20% of adults received a daily ASA regimen in the United States, with this number increasing to nearly 50% in those aged 65 and older [18]. According to data from SPoC prospective multicentre trial [6] based on 5178 polypectomies in 2692 patients, 14.3% of the patients received ASA. ASA, commonly used at low dosage (75–160 mg daily), and NSAIDs decrease platelet aggregation by

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irreversibly inhibiting cyclooxygenase-1. As a result, the synthesis of thromboxane A2, a potent platelet-activating agent, is blocked [19,20].

1.1. Objective

The aim of this systematic review and meta-analysis was to assess the risk of PPB in patients that underwent colorectal polypectomy and exposed to ASA/NSAIDs.

2. Methods

2.1. Eligibility criteria

We included studies with any kind of design, reported as full-text or abstract. We included adults (≥18 years old) who underwent colorectal polypectomy (cold polypectomy, snare polypectomy, endoscopic muscosal resection, endoscopic submucosal dissection). We excluded studies where only endoscopic submucosal dissection was performed. We included studies comparing patients exposed to ASA/NSAIDs with those not exposed to these drugs in the periendoscopic period. Exposure was defined as consuming one of these drugs (any dosage) before (at least three days) or/and after colorectal polypectomy. We excluded studies where exposure status was not clearly defined in the methods. Studies that comprehended aspirin in the generic term "antithrombotics" (where other drugs could be aggregated as thienopyridines) were also excluded.

2.1.1. Outcomes

Immediate PPB was defined as passage of blood from the site of polypectomy during the procedure (or within 6 h). Delayed PPB was defined as hematochezia between 6 h and 14 days after polypectomy. Clinically significant (CS) PPB was defined by [21] the International Society on Thrombosis and Haemostasis (ISTH) definition (death, hemodynamic instability, drop of >2 g/dl of haemoglobin, need to repeat endoscopy with or without intervention, or need for >2 blood transfusion). Primary outcome was to evaluated the risk of PPB in patients exposed and not exposed to ASA/NSAIDs. Secondary outcomes evaluated, in patients exposed and not exposed to ASA/NSAIDs, the risk of immediate, delayed, clinically significant bleeding and for lesions ≥2 cm.

2.2. Information sources

We identified studies in English language in Medline (from 1950 to November 2016) and Embase (from 1974 to November 2016) databases. We searched all records using the search strategy in Appendix A–Supplementary material. We checked reference lists of all primary studies and review articles for additional references.

2.3. Study selection

Two review authors [FP, HB] independently screened the abstracts for inclusion of all the potential studies identified as a result of the search. Then, the same authors independently screened the full-text and identified studies for inclusion and recorded reasons for exclusion. Any disagreement was resolved through discussion or, if required, a third person [RLC] was consulted. Duplicates were identified and excluded. If there was any suspicion of cohort overlap between studies, only the most large study was included. The selection process was recorded in sufficient detail to complete a PRISMA flow diagram [22].

2.4. Data collection process

A database was used to collect the data of studies' characteristics and outcomes. One review author [FP] extracted the following data from included studies: publication year, setting (single/multicentre study), study design (prospective/retrospective), matching of cases with controls and adjusting factors, method to define and to find cases and controls. Characteristics from participants and lesions were: number, age, gender, ASA/NSAIDs dosage, intake of ASA/NSAIDs before and/or after polypectomy, characteristics of lesions (size, location, morphology), techniques of polypectomy, endoscopist's experience, prophylactic tools employed for prevention of bleeding, exclusion criteria, management of incomplete information. For every study it was reported the severity (minor/major) and time (immediate/delayed) of bleeding. It was reported if data about cases/controls were explicated in the text or were deduced from the reviewer. For Example, Choung et al. [9] reported the rates of not bleeding and bleeding events in patients exposed to ASA (262/3746 and 3/42 respectively), aspirin + clopidogrel (67/3746 and 1/67 respectively) and anticoagulant (127/3746 and 4/67 respectively). The rate of events (bleeding) and the rate of no-event (not bleeding) were computed taking into account exposed cases (patients receiving ASA/NSAIDs with a bleeding event), not exposed cases (total of cases – cases exposed to ASA/NSAIDs – cases exposed to ASA and clopidogrel – cases exposed to anticoagulants), exposed controls (patients receiving ASA/NSAIDs without a bleeding event), not exposed controls (total of controls – controls exposed to ASA – controls exposed to aspirin and clopidogrel – controls exposed to anticoagulants). The process was checked from another reviewer [GG].

2.5. Risk of bias in individual studies

Two review authors (FP, HB) independently assessed the risk of bias for each study using the criteria outlined in the Newcastle-Ottawa Scale [23]. We resolved any disagreements by discussion or by involving another author (RLC). We graded each potential source of bias together with a justification for our judgement. We summarised the risk of bias judgements across different studies for each of the domains listed.

2.6. Dealing with missing data

We contacted investigators in order to obtain missing numerical outcome data if possible (e.g. when a study is identified as abstract only). When it was not possible and the missing data were thought to introduce serious bias, we will explore the impact of including such studies in the overall assessment of results by a sensitivity analysis.

2.7. Statistical analysis

For each endpoint, we analysed data obtained from different studies to determine pooled Odds Ratio (OR) and the 95% Confidence Intervals (CIs). We used a fixed-effect Mantel–Haenszel model in order to obtain a preliminary summary estimate. Heterogeneity in meta-analysis refers to the variation in study outcomes between studies. Heterogeneity was explored by Cochran's Q, which is calculated as the weighted sum of squared differences between individual study effects and the pooled effect across studies. The I^2 statistic describes the percentage of variation across studies that is due to heterogeneity rather than chance [24]. If heterogeneity was consistent (by p-value of $Q < 0.1$ and by I^2 degree > 50%), a random effect model was used. Sources of heterogeneity were explored using subgroup analyses and meta regression analyses (only if there were more than 10 trials and data

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