GASTROENTEROLOGY 1997;112:1817–1822

Famotidine for Healing and Maintenance in Nonsteroidal Anti-inflammatory Drug–Associated Gastroduodenal Ulceration

NICHOLAS HUDSON,* ALI S. TAHA,† ROBIN I. RUSSELL,† PENELOPE TRYE,§ JEREMY COTTRELL,§ STEPHEN G. MANN,§ ANTHONY J. SWANELL,‖ ROGER D. STURROCK,† and CHRISTOPHER J. HAWKEY*

* Division of Gastroenterology, University Hospital, Nottingham, England; †Department of Gastroenterology and Rheumatology, Glasgow Royal Infirmary, Glasgow, Scotland; §Merck Research Laboratories UK, Hoddesdon, Herts, England; and ‖Department of Rheumatology, University and City Hospitals, Nottingham, England

See editorial on page 2143.

Background & Aims: Nonsteroidal anti-inflammatory drugs (NSAIDs) are strongly associated with gastroduodenal ulceration. How to manage patients with NSAID-associated ulcers is a common clinical dilemma. High-dose famotidine in the healing and maintenance of NSAID-associated gastroduodenal ulceration was therefore evaluated. *Methods:* One hundred four patients with rheumatoid or osteoarthritis who had gastroduodenal ulceration received famotidine, 40 mg twice daily. Sixteen patients stopped and 88 continued their NSAID treatment. Ulcer healing was assessed endoscopically at 4 and 12 weeks. Seventy-eight NSAID users with healed ulcers were then randomized to receive 40 mg twice daily famotidine or placebo and underwent endoscopy at 4, 12, and 24 weeks. *Results:* Cumulative ulcer healing rates at 12 weeks were 89.0% (95% confidence interval [CI], 82.3%–95.7%) for patients who continued NSAID treatment and 100% (95% CI, 82.9%–100.0%) for those who stopped. The subsequent estimated cumulative gastroduodenal ulcer relapse over 6 months for NSAID users who took placebo was 53.5% (95% CI, 36.6%–70.3%). This was reduced to 26.0% (12.1%–39.9%) in patients taking famotidine ($P = 0.011$). *Conclusions:* High-dose famotidine is effective ulcer healing therapy in patients who stop or continue NSAID treatment and significantly reduced the cumulative incidence of gastroduodenal ulcer recurrence compared with placebo when given as maintenance therapy.

Nonsteroidal anti-inflammatory drugs (NSAIDs) are strongly associated with gastroduodenal ulceration and the complications of ulcer hemorrhage and perforation.[1–4] These risks seem to be increased in patients with a history of gastroduodenal ulceration.[5–8] Use of the prostaglandin analogue misoprostol for prophylaxis against development of NSAID-associated ulceration is well established,[6–11] but there is very little evidence about ulcer healing.[12] Treatments that suppress acid are better tolerated, but high doses are needed to prevent acute mucosal injury.[13–15] Because management of patients presenting with ulceration represents the most common dilemma in the management of such patients, we conducted a study to assess the efficacy of high-dose famotidine, an H_2 antagonist, in both the healing of NSAID-associated gastroduodenal ulceration and the subsequent prevention of ulcer relapse when given as maintenance therapy. This enabled us to compare ulcer development rates with those observed in a study of primary prophylaxis conducted to an identical design, at the same time, in a cohort of patients drawn from the same population.

Patients and Methods

Design

The study consisted of two phases: an open study of famotidine, 40 mg twice daily, in the healing of endoscopically proven gastroduodenal ulceration and, in patients with successful ulcer healing, a prospective randomized double-blind placebo-controlled maintenance study of famotidine, 40 mg twice daily, as secondary prophylaxis against endoscopically detected recurrent gastroduodenal ulceration.

Patients

Adult patients (aged ≥18 years) with rheumatoid arthritis or osteoarthritis were recruited from the rheumatology and orthopedic clinics, provided they had been receiving an NSAID within the range of standard recommended dosage for at least 1 month before endoscopy. Patients were not considered for the study if they had been taking antiulcer drugs other than antacids <7 days before study entry or were taking steroids at a dosage equivalent ≥7.5 mg prednisolone daily, methotrexate, or antineoplastic drugs. The other main exclu-

Abbreviations used in this paper: CI, confidence interval; HAQ, Health Assessment Questionnaire.

0016-5085/97/$3.00

sion criteria were lactation, child-bearing potential, renal failure, diabetes, or clinically significant prestudy laboratory abnormalities.

Recruitment Procedures

Recruitment was performed by two gastroenterologists who attended the Rheumatology and Orthopedic Clinics and approached all potentially suitable arthritic patients regardless of dyspeptic symptoms. Patients who accepted the invitation to participate underwent screening endoscopy. At endoscopy, ulcers, erosions, and intramucosal hemorrhages were recorded separately for the esophagus, gastric body, gastric antrum, duodenal bulb, and second part of the duodenum.

Definitions and Assessments

An ulcer was defined as an excavated mucosal break >3 mm in diameter, measured using biopsy forceps or a custom-made measuring device. Erosions (defined as superficial mucosal breaks) and intramucosal hemorrhages (defined as hemorrhagic lesions without an overlying mucosal break), together with any ulcer in each of the target areas, were used to derive Lanza scores.[16] *Helicobacter pylori* status was determined by CLO test and gastric antral histology. Before commencement of the study, the two endoscopists (A.S.T. and N.H.) attended each other's endoscopic sessions and reviewed still and video images as a process of standardization of reporting criteria for ulcers and other lesions. Both studies were approved by the Nottingham and Glasgow Hospital ethics committees.

Healing Phase

Patients with ulcers were entered into the healing study and invited to discontinue their NSAID therapy. Patients without ulceration entered a prophylaxis study, reported elsewhere.[17] Patients received two 20-mg famotidine tablets twice daily. In addition, antacid tablets (Maalox; Rhone-Poulenc Rorer, Eastbourne, East Sussex, England) were provided for relief of dyspepsia as required. Patients underwent endoscopy after 4 weeks of treatment and, if the ulcer remained unhealed, underwent a repeat endoscopy at 12 weeks. Patients who were unhealed at 12 weeks were designated as treatment failures.

Maintenance Phase

Patients who wished to continue NSAID therapy and whose ulcers had healed successfully during the study or during the following 4 weeks were invited to enter the maintenance study. These patients were randomized to receive either famotidine, 40 mg twice daily, or placebo in a double-blind fashion and underwent further endoscopy at 4, 12, and 24 weeks or until ulcer relapse during the study.

Nonendoscopic Assessments

Patients were assessed routinely at baseline and at the time of each subsequent endoscopy. In addition to endoscopy, the following assessments were performed: NSAID and other drug usage, arthritis-related physical disability measured by the Health Assessment Questionnaire (HAQ), vital signs, complete physical examination (baseline and end of study), urinalysis, hematology, and biochemistry. Antacid use and abdominal symptoms were recorded daily on specific diary cards. Compliance with study drugs was assessed by tablet count and adverse events by open questioning at each visit.

End Points

The primary end point of the healing study was healing of gastroduodenal ulceration at the 4- or 12-week endoscopy. The primary end point of the maintenance study was the cumulative incidence of gastroduodenal ulcer relapse as assessed at follow-up endoscopies (at 4, 12, and 24 weeks). The main secondary end points were the corresponding findings related to gastric and duodenal ulcers separately, Lanza scores for lesser degrees of gastroduodenal injury, abdominal pain, and antacid consumption. The main safety analyses included assessment of adverse events, arthritis, HAQ, physical examination, and laboratory results.

Statistical Methods

The study was designed to be pragmatic rather than explanatory. Efficacy data in both the healing and maintenance study were therefore subjected to a primary "all patients treated" analysis. Product limit (Kaplan–Meier) estimates of the cumulative incidence of ulceration, the primary end point, were made for each endoscopy visit. Comparisons between treatment groups were made using the log rank test to allow for those withdrawn for reasons other than ulceration. The cumulative incidence of gastric ulceration and duodenal ulceration were also similarly analyzed separately. Similar per-protocol analyses of efficacy were also performed on assessable patients. Assessable patients were defined as those who satisfied the inclusion and exclusion criteria, consumed >80% of both the prescribed NSAID and study drugs, did not consume additional full-dose salicylates, and had their end-of-study endoscopy performed no more than 5 days after the end of study treatment. Secondary efficacy end points were also analyzed using "all patients treated" as the primary analysis and a per-protocol approach as the secondary analysis. The Mantel–Haenszel test or Kruskal–Wallis test were used where appropriate. Comparisons were considered significant at P values of <0.05.

Prognostic factors. A Cox proportional regression model was constructed to assess the effects of 26 potential prognostic factors on cumulative ulcer incidence. The potential prognostic factors were identified before the study. These factors were added to or removed from the model in a stepwise regressive procedure, based on a threshold P value (<0.1 to be added or >0.1 to be removed), with the effect of treatment group being included at each stage. The prognostic variables included the following; recruiting center, age, sex, smoking habit, alcohol use, type of NSAID, duration of prior NSAID use, rheumatological diagnosis, duration of arthritis, history of peptic ulceration, time to ulcer healing, presence of erosions or hemorrhagic lesions at screening endoscopy, ulcer size at

Table 1. Patient Characteristics in Healing and Maintenance Study ("All Patients Treated" Population)

		Maintenance	
	Ulcer healing (n = 104)	Famotidine 40 mg (n = 39)	Placebo (n = 39)
Median age (*yr*, range)	58 (31–89)	58 (32–79)	55 (35–89)
M/F	32/72	14/25	12/27
Smokers (%)	32 (31)	13 (33)	12 (31)
Alcohol consumption (%)	45 (43)	17 (44)	18 (46)
Rheumatoid/osteoarthritis	82/22	34/5	33/6
Previous PUD (%)	26 (25)	12 (31)	11 (28)
Second-line therapy (%)	25 (24)	12 (31)	10 (18)
Mean HAQ score (SD)	1.4 (0.8)	1.5 (0.8)	1.5 (0.8)
NSAID type			
Naproxen (%)	31 (30)	14 (36)	11 (28)
Indomethacin (%)	19 (18)	3 (8)	9 (23)
Diclofenac (%)	14 (13)	6 (15)	4 (10)
Other (%)	40 (38)	16 (41)	15 (39)

PUD, peptic ulcer disease.

endoscopy, abdominal pain at baseline, HAQ score, second-line antirheumatoid agent prednisolone, total leukocyte count, hemoglobin, and platelet count.

Results

Five hundred seventy patients (Glasgow, n = 299; Nottingham, n = 271) were invited to enter the study, of whom 389 accepted (Glasgow, n = 235; Nottingham, n = 154). Of these, 104 had gastroduodenal ulcers at screening endoscopy (gastric ulcers, n = 76; duodenal ulcers, n = 42; and both, n = 14) and were entered into the healing study; 69 (66%) were recruited from Glasgow and 35 (34%) from Nottingham. *H. pylori* status was established (histology and urease test) in 93 patients. The patients with no ulcer at screening participated in a primary prophylaxis study run concurrently. As shown in Table 1, patients were well matched for age, sex, smoking status, alcohol usage, underlying arthritis, ulcer history, frequency of joint pain, HAQ score, and use of individual NSAIDs or disease-modifying drugs. Eighty-two patients had rheumatoid arthritis and 22 had osteoarthritis. Sixteen patients agreed to stop their NSAID therapy during the course of the healing study, and 88 continued NSAID therapy.

Ulcer Healing Study

At 4 weeks, 65.9% (95% confidence interval [CI], 56.0%–75.8%) of patients who continued and 81.3% (95% CI, 54.4%–96.0%) who discontinued NSAID therapy had successfully healed ulcers on "all patients treated" analysis. Two patients unhealed at 4 weeks were not assessed further (see below). At 12 weeks, the cumulative healing rate was 89.0% (95% CI, 82.3%–95.7%) in patients who remained on NSAID therapy and 100% (95% CI, 82.9%–100.0%) in those who discontinued

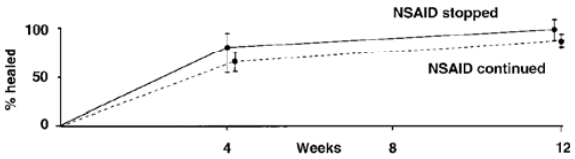

Figure 1. Proportion of patients with ulcers healed with famotidine, 40 mg twice daily, at 4 and 12 weeks in those who continued or discontinued NSAID therapy.

NSAID therapy. Differences in healing rates were not significant at either 4 or 12 weeks (Figure 1). Ulcers failed to heal in 9 patients after 12 weeks of therapy, all of whom continued NSAID therapy. For the corresponding results per protocol, the cumulative healing rates were 67.6% (95% CI, 56.7%–78.5%) vs. 76.9% (95% CI, 54.0%–99.8%) at 4 weeks and 91.2% (95% CI, 84.4%–97.9%) vs. 100% (95% CI, 79.4%–100.0%) at 12 weeks in NSAID users and nonusers, respectively.

Of the prognostic variables, only ulcer size was significantly and inversely correlated with ulcer healing. Nonetheless, the 12-week cumulative healing rate for 49 ulcers that were >5 mm in diameter was 81.6% (95% CI, 70.7–92.5). Healing occurred in 85.7% (95% CI, 76.6%–94.8%) of patients who were *H. pylori* negative and 93.0% (95% CI, 86.4%–99.6%) of those who were *H. pylori* positive. When healing rates for gastric and duodenal ulcer were analyzed separately, the cumulative healing rates for gastric ulcers in 63 patients who continued NSAID therapy were 63.5% (95% CI, 51.6%–75.4%) at 4 weeks and 86.7% (95% CI, 78.2%–95.2%) at 12 weeks. For duodenal ulcers, healing rates were 75.7% (95% CI, 61.9%–89.5%) and 97.0% (95% CI, 91.1%–100.0%), respectively. Similar results were obtained from the per-protocol analysis.

Maintenance Study

Seventy-eight patients who continued to use NSAIDs agreed to enter the maintenance study, although 1 patient did not undergo repeat endoscopy. Patient characteristics are shown in Table 1. On "all patients treated"

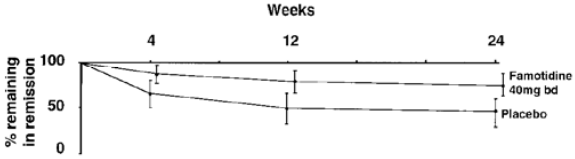

Figure 2. Life table of cumulative incidence of ulcer recurrence rates at 4, 12, and 24 weeks in patients taking maintenance famotidine, 40 mg twice daily, compared with placebo.

Table 2. Prognostic Variables for Ulcer Relapse During Maintenance Phase

	Cumulative relative risk (95% CI)	*P* value
Famotidine 40 mg twice daily	0.093 (0.025–0.34)	0.0003
Duodenal ulcer in healing study	4.919 (1.33–18.16)	0.017
Length of time to healing	4.944 (1.25–19.53)	0.023
HAQ score	3.361 (1.41–8.01)	0.006
Use of ketoprofen	8.067 (0.91–71.39)	0.061
Shorter duration of arthritis	0.935 (0.87–1.01)	0.071
Model with *H. pylori* included		
Famotidine 40 mg twice daily	0.290 (0.108–0.774)	0.013
Shorter duration of arthritis	0.953 (0.893–1.017)	0.149
Baseline *H. pylori* status	2.08 (0.696–6.218)	0.190

analysis, the estimated cumulative incidence of gastroduodenal ulceration over the following 24 weeks for the 39 patients taking placebo was 53.5% (95% CI, 36.6%–70.3%) compared with 26.0% (95% CI, 12.1%–39.9%) ($P = 0.011$) in patients taking famotidine, 40 mg twice daily (Figure 2). The crude ulcer incidence at 4 weeks was 34.2% (95% CI, 19.1%–49.3%) in the placebo group vs. 12.8% (95% CI, 2.3%–23.3%) in the famotidine group, and at 12 weeks was 49.9% (95% CI, 33.3%–66.5%) for placebo vs. 20.5% (95% CI, 7.8%–33.2%) for famotidine. Similar results were obtained following the per-protocol analysis.

Famotidine also significantly reduced the incidence of gastric ulceration at 24 weeks when analyzed separately, from 41.4% (95% CI, 24.0%–58.7%) in the placebo group to 19.1% (95% CI, 6.3%–31.9%) in the famotidine group ($P = 0.026$). For duodenal ulcers, the estimated rate was 16.7% (95% CI, 2.8%–30.6%) compared with 7.9% (95% CI, 0.0%–16.5%) with famotidine ($P = 0.31$). Similar results were obtained following the per-protocol analysis.

Risk Factors

The Cox proportional regression analysis (Table 2) confirmed the efficacy of famotidine. Famotidine, 40 mg twice daily, was associated with an estimated conditional risk ratio of 0.093 (95% CI, 0.025–0.337) compared with placebo ($P < 0.0003$). Other significant adverse prognostic factors were as follows: not having a duodenal ulcer in the healing study, long time to ulcer healing, longer duration of arthritis, low HAQ score, and use of ketoprofen.

A similar analysis was performed that included *H. pylori* status as a prognostic variable. In this analysis, baseline *H. pylori* infection was associated with a 2.08 (range, 0.70–6.22) cumulative relative risk of relapse, although this did not reach statistical significance ($P = 0.19$; Table 2). Estimated cumulative relapse rates in patients taking placebo were 63.6% (range, 43.5%–83.7%) in patients who were *H. pylori* positive (n = 25) and 23.8% (range, 0%–52.8%) in those who were *H. pylori* negative (n = 10). For famotidine, the figures were 23.1% (range, 3.1%–43.0%) for *H. pylori*–positive patients (n = 18) and 21.4% (range, 4.7%–50.8%) for *H. pylori*–negative patients (n = 14).

Secondary Efficacy Analyses

Gastric mucosal injury expressed as a Lanza score was significantly lower in the famotidine group compared with placebo at the 4-week endoscopy in both the healing and maintenance study ($P = 0.042$). Data for later endoscopies could not be analyzed directly because they were confounded by selective dropout of ulcer patients. Differences between the treatment groups for abdominal pain scores, mean daily antacid consumption, and arthritic pain scores were not significant, and there was no correlation between abdominal pain and the presence or absence of ulceration.

Safety and Withdrawals

In the ulcer healing study, 1 patient died of bronchopneumonia and multisystem disorder and 1 of pancreatic cancer after completing the study. One patient withdrew because of nausea and vomiting. In the maintenance study, 4 patients in the famotidine group and 7 in the placebo group were withdrawn for reasons other than ulcer relapse. One patient in each group was lost to follow-up evaluation, and 3 patients in the placebo group were unwilling to continue. There were three withdrawals in both the famotidine and placebo groups because of adverse events; none of them were drug related.

Famotidine was well tolerated as both healing and maintenance therapy. In the healing study, 10.6% of patients appeared to have drug-related adverse events, and 1 patient withdrew. In the maintenance study, 28.2% of patients taking famotidine had adverse events, of which 12.8% were believed to be drug related, and 35.9% of those in the placebo group had adverse events, of which 12.8% were also believed to be drug related.

Discussion

This study shows that famotidine, 40 mg twice daily, heals ulcers in arthritic patients who continue NSAID therapy and reduces subseqent relapse when continued after successful healing. There was no significant retardation of healing in patients who continued NSAID therapy compared with those who stopped. Although relatively few patients stopped NSAID therapy and we cannot completely exclude the possibility that retardation in those who continued was missed, this is unlikely

to be a major effect because the healing rate in this group was high and similar to that previously reported for omeprazole, 40 mg daily.[18] Famotidine was effective in healing both gastric and duodenal ulcers and in those who were either *H. pylori* positive or negative and seemed to be effective for larger as well as smaller ulcers. Subgroup analysis of the relapse data must be more guarded because of the possibility of confounding due to the differential relapse rate and because numbers were small. It seems clear that famotidine was able to prevent gastric ulcer relapse. In our study, there were too few duodenal ulcers to know whether the relapse rate was truly reduced. However, there was a trend in this direction, and previous studies have shown that duodenal ulcers are easier to prevent than gastric ulcers using standard doses of H_2 antagonists under primary prophylaxis conditions.[5,19] Relapse rates in patients taking famotidine were similar whether patients were *H. pylori* positive or negative, but small numbers limit the confidence of this conclusion. Whether *H. pylori* eradication would affect the clinical course of NSAID users is unknown and is currently the subject of investigation. However, epidemiological data suggest that the risk of NSAID-associated ulcer complications may not be substantially influenced by *H. pylori* status.[20]

We used a high dose of famotidine because previous work has suggested that healing rates with standard doses of H_2 antagonists are reduced if NSAID therapy is continued[21,22] but that this effect is abolished if a more profound effect with proton pump inhibitors is used.[18] We have also found that only the higher dose of famotidine, when used as primary prophylaxis, was capable of reducing significantly the incidence of gastric ulcer.[17] Our data are compatible with earlier evidence that more profound acid suppression can overcome the retardation of healing by NSAID therapy.[18] These data are also supported by short-term studies in volunteers that show that proton pump inhibitors or high doses of famotidine are more effective than standard doses of H_2 receptor antagonists in preventing acute gastric erosions[13–15] and more recent evidence, reported in abstract form, concerning large trials of the proton pump inhibitor omeprazole.[23]

Previous studies have examined the use of both ranitidine and misoprostol in the prevention of NSAID-associated gastroduodenal ulceration.[5–7,17] Misoprostol appears to confer protection against both gastric and duodenal ulcers, whereas standard doses of ranitidine prevented duodenal ulcers but were relatively ineffective in preventing gastric ulcers. However, in most of these studies, patients with ulceration at baseline endoscopy were either excluded from entry or a mixed group was studied and *H. pylori* status was not established. Epidemiological evidence suggests that patients with a history of peptic ulcer disease are at greater risk of subsequent ulceration and ulcer complications.[7,8] This phenomenon was also observed in previous studies in that the rate of ulceration observed endoscopically was greater in patients with an ulcer history[5,6] or in those in whom ulcers were healed before entry.[24] Our study directly establishes the importance of ulceration detected during NSAID use as a risk factor for further ulceration, because the placebo relapse rate was 52.3%, significantly higher than the 25.8% we found in patients who did not have ulcers at baseline endoscopy and who were studied under primary prophylaxis conditions.[17] We are confident that this difference is likely to be genuine for a variety of reasons. Patients in the two studies were drawn from the same populations and studied concurrently to an identical protocol. All examinations were conducted by two endoscopists, each of whom agreed on criteria for ulcer diagnosis, thereby avoiding the large interobserver variation in the assessment of NSAID-associated gastroduodenal lesions that is likely to characterize other large studies conducted in multiple centers.[25] The high subsequent relapse rate associated with detection of an ulcer in NSAID users has important implications for management and reinforces the notion that this is a group in whom maintenance treatment should be considered if the NSAID therapy cannot be stopped. Our data suggest that treatment with famotidine, 40 mg twice daily, would be appropriate to consider for these patients.

References

1. Griffin MR, Piper JM, Daugherty JR, Snowden M, Ray WA. Nonsteroidal anti-inflammatory drug use and increased risk for peptic ulcer disease in elderly persons. Ann Intern Med 1991;114: 257–263.
2. Guess HA, West R, Strand LM, Helston D, Lydick EG, Bergman U, Wolski K. Fatal upper gastrointestinal haemorrhage or perforation among users and nonusers of anti-inflammatory drugs in Saskatchewan, Canada, 1983. J Clin Epidemiol 1988;41:35–45.
3. Hawkey CJ. Non-steroidal anti-inflammatory drugs and peptic ulcers. Facts and figures multiply, but do they add up? Br Med J 1990;300:278–284.
4. Langman MJS, Weil J, Wainwright P, Lawson DH, Rawlins MD, Logan RFA, Murphy M, Vessey MP, Colin-Jones DG. Risk of bleeding peptic ulcer associated with individual non steroidal anti-inflammatory drugs. Lancet 1994;343:1075–1078.
5. Ehsanullah RSB, Page MC, Tildesley G, Wood JR. Prevention of gastroduodenal damage induced by non-steroidal anti-inflammatory drugs: controlled trial of ranitidine. Br Med J 1988;297: 1017–1020.
6. Graham DY, White RH, Moreland LW, Schubert T, Katz R, Jaszewski R. Duodenal and gastric ulcer prevention with misoprostol in arthritic patients taking NSAIDs: Misoprostol Study Group. Ann Intern Med 1993;119:257–262.
7. Fries JF, Williams CA, Bloch DA, Michel BA. Nonsteroidal anti-inflammatory drug–associated gastropathy: incidence and risk factor models. Am J Med 1991;91:213–222.

DOCKET ALARM

Explore Litigation Insights

Docket Alarm provides insights to develop a more informed litigation strategy and the peace of mind of knowing you're on top of things.

Real-Time Litigation Alerts

Keep your litigation team up-to-date with **real-time alerts** and advanced team management tools built for the enterprise, all while greatly reducing PACER spend.

Our comprehensive service means we can handle Federal, State, and Administrative courts across the country.

Advanced Docket Research

With over 230 million records, Docket Alarm's cloud-native docket research platform finds what other services can't. Coverage includes Federal, State, plus PTAB, TTAB, ITC and NLRB decisions, all in one place.

Identify arguments that have been successful in the past with full text, pinpoint searching. Link to case law cited within any court document via Fastcase.

Analytics At Your Fingertips

Learn what happened the last time a particular judge, opposing counsel or company faced cases similar to yours.

Advanced out-of-the-box PTAB and TTAB analytics are always at your fingertips.

API

Docket Alarm offers a powerful API (application programming interface) to developers that want to integrate case filings into their apps.

LAW FIRMS
Build custom dashboards for your attorneys and clients with live data direct from the court.

Automate many repetitive legal tasks like conflict checks, document management, and marketing.

FINANCIAL INSTITUTIONS
Litigation and bankruptcy checks for companies and debtors.

E-DISCOVERY AND LEGAL VENDORS
Sync your system to PACER to automate legal marketing.